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Perspective

A generative adversarial model of intrusive imagery in the human brain

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Abstract

The mechanisms underlying the subjective experiences of mental disorders remain poorly understood. This is partly due to long-standing over-emphasis on behavioral and physiological symptoms and a de-emphasis of the patient's subjective experiences when searching for treatments. Here, we provide a new perspective on the subjective experience of mental disorders based on findings in neuroscience and artificial intelligence (AI). Specifically, we propose the subjective experience that occurs in visual imagination depends on mechanisms similar to generative adversarial networks that have recently been developed in AI. The basic idea is that a generator network fabricates a prediction of the world, and a discriminator network determines whether it is likely real or not. Given that similar adversarial interactions occur in the two major visual pathways of perception in people, we explored whether we could leverage this AI-inspired approach to better understand the intrusive imagery experiences of patients suffering from mental illnesses such as post-traumatic stress disorder (PTSD) and acute stress disorder. In our model, a nonconscious visual pathway generates predictions of the environment that influence the parallel but interacting conscious pathway. We propose that in some patients, an imbalance in these adversarial interactions leads to an overrepresentation of disturbing content relative to current reality, and results in debilitating flashbacks. By situating the subjective experience of intrusive visual imagery in the adversarial interaction of these visual pathways, we propose testable hypotheses on novel mechanisms and clinical applications for controlling and possibly preventing symptoms resulting from intrusive imagery.

Introduction

Despite decades of research, the treatment of mental disorders still represents a considerable challenge. Trauma and stressor-related disorders are devastating and common. For example, post-traumatic stress disorder (PTSD) shows an estimated lifetime prevalence of 6.8% in the United States with substantially higher prevalence amongst war veterans (1–4). It results in billions of dollars of cost annually (5). Better understanding of this disorder could both relieve suffering in patients and reduce the burden on the healthcare system caused by inadequate treatments.

Over the years, multiple therapeutic approaches for mental disorders have been designed using physiological or behavioral models to explain the symptoms of diseases. Relatively few conceptualizations have actually been specifically centered on patients' subjective experience (6, 7). While we still have a limited understanding of the brain mechanisms generating such experiences, recent scientific advances have begun to suggest how the troubling experiences of patients might come about. Here, we present one such perspective to explain how intrusive imagery may come to be experienced in PTSD.

Mental imagery is a common experience in the daily life of many people. We imagine what an anticipated dinner, or a predicted tornado, might look like. Even when imagining threatening or disturbing content, one feels generally safe since they know it is just a product of their own imagination, with no real harm likely at the moment. But, how do we distinguish between what is imaginary and real?

It would seem that some process is necessary to monitor ongoing brain activity to make the distinction. Such a process has begun to be modeled in artificial intelligence (AI) using generative adversarial networks (GANs). In a simple case, a generator network simulates a prediction of the world while a discriminator

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decides whether the input is real or not. We suggest that the human brain possesses a similar mechanism to distinguish whether our conscious experiences are perceptions or mental images. When this complex interplay goes awry, overconfidence in the prediction of threat or hasty prediction of threat where there is none could lead to intrusive images being perceived as genuine sources of harm in patients with PTSD.

In this paper, we review the literature from vision science and human threat perception to understand how perception and encoding of traumatic events can lead to intrusive imagery in disorders such as PTSD. We propose that a GAN-like model in the brain might improve understanding of intrusive imagery in PTSD. In the model, a generator influenced by the "nonconscious" visual pathway changes bottom-up processing in the "conscious" pathway. A discriminator then needs to decide, based on the sensory information available from both pathways, whether a given percept stems from reality or imagination. During intrusive imagery, imagery is generated, which over-represents traumatic or disturbing content, and this imagery in turn influences bottom-up processing in the conscious pathway. The result is overly realistic imagery presented to a potentially faulty PFC discriminator that can be misclassified as taking place in reality.

Monitoring What is Real and What is Imagined

Perceived reality does not always match sensory input. This is made evident by phenomena such as visual illusions, hallucinations, dreams, and mental imagery. These phenomena each reflect visual experiences in the absence of external stimulation (8, 9). Such experiences highlight the fact that visual experience can exist totally independent of the outside world.

Such observations have led to the idea of perception being a prediction rather than a representation of objective reality (10). If visual experience is generated by the brain, it should possess mechanisms for distinguishing between coexisting representations, as is the case when imagination competes with reality. Under typical circumstances, we have very little trouble discerning which visual images come from the world in front of us and those which we are imagining in our mind.

Machine learning reality from imagination

Computer science has found a way to create efficient learning for neural networks by giving them an "imagination" in GANs (11). In this framework, a "generative" model generates its own simulated data in an attempt to model the objective reality of the world. Meanwhile, a "discriminator" model is paired adversarially to the generative model to discriminate between real observed data and generated simulation data. The adversarial process results in highly efficient learning for both systems. For example, a generator can be trained to simulate images while a discriminator learns to discriminate whether a photograph is real or not. After sufficient learning, the generator can produce strikingly realistic photographic stimuli, while the discriminator can outperform the human eye in telling the fake photographs from the real ones (12).

Perceptual Reality Monitoring in the Brain

Building from GANs, neuroscientists have proposed that a similar mechanism controls conscious awareness (13–15). Under this framework, mental imagery mechanisms act as the generator while reality monitoring acts as the discriminator parsing between sensory activations coming from real or imagined sources.

Just like a sufficiently trained generator, the brain can generate very convincing visual experiences without external input. Anyone who has woken up relieved that a dream did not really occur can attest to this. Waking visual imagery can also generate convincing percepts with high fidelity, but rarely are these percepts mistaken for reality. What, then, makes our sleeping dreams so convincingly realistic while our waking imagination safely stays in the backseat, given that internally generated percepts are subjectively experienced by the observer in both cases? A neural mechanism, which monitors the origins of perceptual representations, otherwise known as perceptual reality monitoring, must be in play.

Reality monitoring is an established process in the memory literature wherein memory traces are "source" monitored to determine their origins (16). This is how we are able to tell whether that fight with our friend actually happened yesterday or if it was just a dream. A similar mechanism must also exist in perception to parse between internal imagery and external perception.

Empirical evidence indicates such a mechanism is indeed present in the human brain. Subjective visual experiences seem to rely on common perceptual mechanisms irrespective of whether the subjective experience comes from exteroceptive perception, imagery, or memory (17, 18). The neural overlap between imagery and perception throughout the visual processing hierarchy is now well-established. Visual imagery, like visual perception, engages early visual cortical regions (such as V1) in a spatially and retinotopically specific way (18–20), and this neural overlap between imagery and perception extends to higher category-specific regions of visual cortex such as the fusiform face area (FFA) and the parahippocampal place area (20). For example, neural activity that selectively occurs in FFA when we consciously see a face also activates when we simply imagine a face (21), yet we do not commonly tend to mistake imagined faces for physical faces in front of us. Moreover, these similar neural activities also occur when we are trying to retrieve a certain face from memory (22) or when we are dreaming about faces (23). Empirical evidence also indicates that the networks supporting visual imagery generation overlap considerably with those supporting episodic autobiographical memory (24, 25). For instance, individuals who lack visual imagery (a condition termed "aphantasia") show a diminished ability to re-experience the past (26) likewise, individuals with a condition known as "Severely Deficient Autobiographical Memory" (SDAM) also disproportionately report visual imagery deficits (27). Memory also influences conscious perception through implicit recollection of how similar a current experience is to previous experiences (28). Collectively, these findings indicate that perception, imagery, and memory all utilize a similar visual representation format based on the fundamentally depictive properties of perception.

Accordingly, some higher-order function in the brain must be responsible for deciding between a percept present in objective reality and one only held in the mind's eye (29). Exactly how complex this higher-order function needs to be is an open question. In its simplest form, something like a "neuronal switch" could account for whether a visual representation comes from perception, imagery, or memory. However, disruption to such a simple switch would have far-reaching consequences beyond the specific intrusions observed in trauma-related disorders. It would most likely generate all kinds of partial and irrelevant subjective experiences. Additionally, such a simple neuronal switch would not be able to

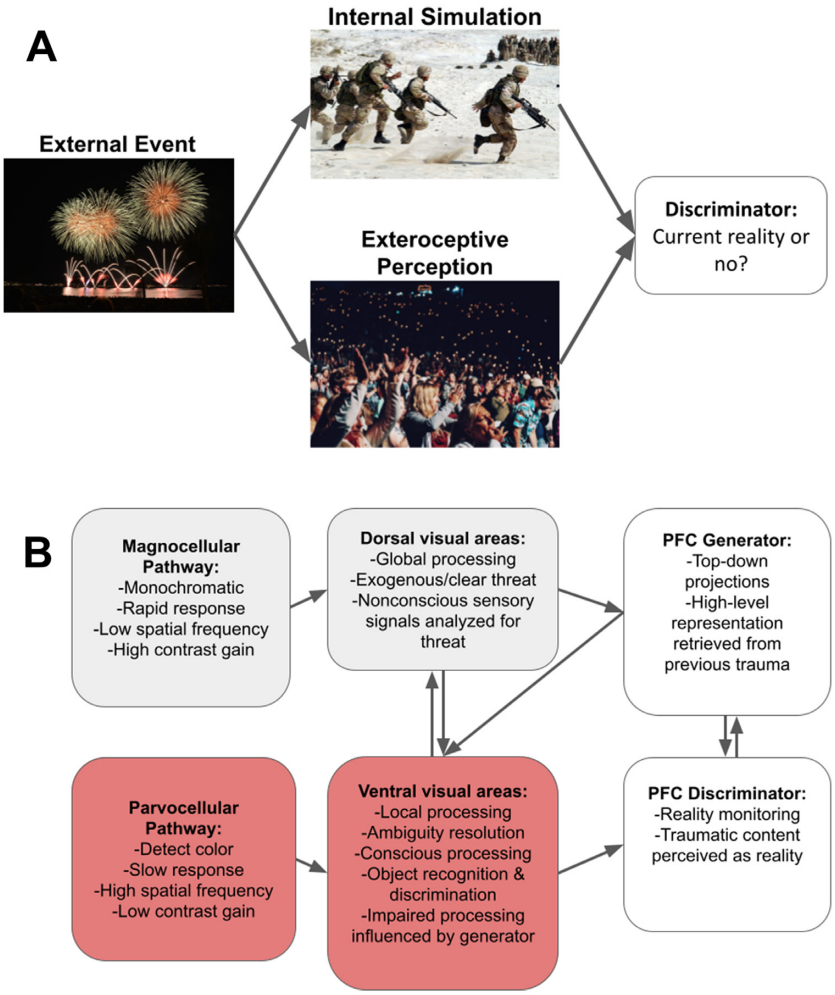

Fig. 1. Perceptual Reality Monitoring Model of PTSD Flashbacks. (A) The reality monitoring process. An external stimulus triggers competing representations during perceptual processing. Current representations of reality are compared to ongoing prediction influenced by previous memory. The discriminator performs reality monitoring to decide whether a representation is current reality or internally generated (e.g., imagery). (B) Neurodynamic model of PTSD intrusive imagery. Magnocellular neurons primarily feed the dorsal visual pathway. The parvocellular neurons feed the ventral visual pathway. A generator sends top-down predictions influenced by the M pathway that influence bottom-up processing in the P pathway. A discriminator in the prefrontal cortex (PFC) decides whether a given percept stems from reality or imagination based on the sensory information available from both pathways. During intrusive imagery, generations related to a traumatic experience coming from the M pathway influence bottom-up processing in the P pathway. Overly realistic imagery is then presented to the PFC discriminator. A faulty discriminator indicates that this imagery represents reality causing the intrusive experience to occur.

account for why certain visual representations are subjectively experienced while others remain unconscious. Nonconscious imagery is processed by the visual system just like conscious imagery and can impact perception and be decoded in visual and executive areas (30, 31). As such, a simple neuronal switch would not distinguish unconscious and conscious processing in the visual system, providing little explanation as to why one becomes consciously experienced and not the other. Consequently, whether or not a visual representation is subjectively experienced seems to depend on a more complicated higher-order function than a simple neuronal switch.

A New Model of PTSD Intrusions

Deficits in either or both the imagery-generating (i.e., generator network) and reality-monitoring processes (i.e., discriminator network) could lead to delusions and hallucinations. Simulations created by the generator that normally have a low probability of being real could be accepted as real by a potentially defective discriminator. Similarly, a defective generator may be capable of fooling the discriminator by generating highly realistic content that is unrelated to one's current surroundings and experience.

In the context of PTSD, it could be that the extreme stress induced by the traumatic event damages the discriminator. Alternatively or additionally, the generator may replay elements of the traumatic event in a way that makes them more likely to be accepted as real by the reality discriminator. For example, the generator is likely to replay these elements frequently in an effort to learn about the traumatic event. Perhaps this replay becomes pathological following trauma due to increased frequency, overly liberal retrieval, and/or overly realistic mental simulation. In this case, the generated activations may come to be treated as real by the discriminator, resulting in an intrusive flashback (Fig. 1A). Multiple cycles of this process could lead to the repetitive intrusive experiences commonly reported in PTSD.

The kind of reality monitoring that would fit the role of discriminator in the human brain is predominantly thought to be

carried out in the prefrontal cortex (13, 16). Thus, this new model predicts PFC dysfunction in PTSD pathophysiology, of which there is considerable evidence. We will discuss these dysfunctions in PTSD below. The generator would lie in the perceptual system and also critically rely on PFC. In the case of vision, a GAN-like adversarial process is already thought to characterize the process of perception with rapidly generated top-down predictions influencing bottom-up processing (32, 33).

Adversarial Perception

Dual systems have long been theorized to underlie visual perception via the dorsal and ventral visual pathways (34, 35). With parallel processing beginning from the retina, the magnocellular (M) and parvocellular (P) pathways engage in distinct but interacting roles resembling an adversarial process (Fig. 1B). These pathways also primarily feed the dorsal and ventral visual pathways, respectively (Fig. 1B). The M pathway processes visual information rapidly along the dorsal pathway reaching the PFC to in turn provide top-down modulation of slower processing in the P pathway along the ventral pathway (33, 36, 37). In this way, the M pathway feeds a generator whose predictions influence the competing bottom-up reality formed by the P pathway. These top-down predictions are influenced both by prior experience (38) and contextual information (39).

Divergent M and P processing begins as soon as light reaches the retina (40, 41). M retinal ganglion cells have high contrast gain and carry monochromatic, low spatial frequency information at a fast conduction velocity. Consequently, it is thought that the M pathway carries coarse "gist-like" information to rapidly inform top-down predictions (36, 42). Evidence indicates visual information is rapidly carried by the M pathway to the orbitofrontal cortex to provide top-down facilitation of bottom-up processing occurring in the P-fed ventral pathway (33, 36). This finding has been recently confirmed using more time sensitive magnetoencephalography (MEG). Increased time resolution compared to fMRI showed activity traveling along the dorsal pathway rapidly fed back to early visual areas (43). Visual stimuli can be accurately decoded as soon as 60 ms in the PFC, indicating rapid projection of visual information to prefrontal areas (44)

Conversely, neurons in the P pathway exhibit high sensitivity to color contrast, but substantially less luminance contrast gain (45), picking up information at a higher spatial frequency (45, 46). This color- and feature-rich information is transmitted at a slower rate while being modulated by top-down predictions from the M pathway (37, 43).

Additionally, the M and P pathways are responsible for global and local processing, respectively. The fast processing of the M pathway leads to a "global precedence" effect—seeing the forest before the trees (47). Removing low spatial frequencies from stimuli stops the global precedence effect, indicating the effect's reliance on the M pathway (48). Moreover, patients with damage to the dorsal pathway can present with a condition called simultanagnosia, where they are unable to perceive more than one object at a time. They have impaired global processing but intact local processing; a damaged M pathway but intact P pathway. By biasing stimuli to the P pathway, patients instantaneously recover global processing, interestingly at the expense of local processing (49). This curious result highlights the interactive nature of these pathways as functional roles of the networks can be swapped under the right conditions. Importantly for our model, the dynamics between the M and P pathways are also differentially tuned to threat.

Threat Perception: Fast and Slow

Experiencing a traumatic event involves processing threatening or otherwise negative stimuli. Threat perception mechanisms need to differentiate an impending threat from a "merely negative," yet still traumatic, situation such as discovering a dead body (50). The rapid M pathway reflexively analyzes incoming sensory data to identify clear direct threats. The slower P pathway builds a detailed representation capable of resolving threat ambiguity.

Showcasing fast response to threat, people identify direct threats significantly faster than indirect threats or merely negative situations (51). Threat images activate the amygdala more strongly and quickly than other merely negative images, while merely negative images activate contextual association areas associated with the P pathway (51).

Rapid threat evaluation in the amygdala is thought to be driven by M inputs (52, 53). The M pathway drives transient visual attention (54) and has shown a bias for exogenous attention captured by biologically salient threats (e.g., spiders, (55)). Rapid response to threat cues by the M pathway is sensitive to social stimuli like faces over scenes (53). Amongst social threat cues (e.g., fearful faces), the M pathway is tuned to clear, unambiguous signals of threat (52, 56). M response to these clear threat cues is heightened by observer trait anxiety (57). The dorsal pathway has also been shown to be activated as quickly as 68 ms when perceiving crowds of emotional faces enabling rapid categorization of crowd emotion (58).

Concurrently, the P pathway processes incoming cues at a slower, more reflective pace, generating a more finely detailed representation. The P pathway has been shown to be sensitive to ambiguous threat cues (52, 56). P response to these ambiguous cues is impaired in people with high anxiety (57). The amygdala response to fearful faces presented to the P pathway occurs approximately 150 ms later than when presented to the M pathway (53). The ventral pathway also responds slower when perceiving emotional face stimuli and responds more to individual faces rather than crowds (58). These latency and processing differences highlight the independent functions carried out by the two pathways when people are confronted with threatening or negative situations.

A direct nonconscious pathway for trauma

As the phylogenetically older neurons, M neurons also feed a subcortical pathway, which carries threatening information to the amygdala via the pulvinar nuclei of the thalamus, bypassing the cortex—colloquially referred to as the "low road" (59). This thalamo–amygdala pathway allows for rapid (both in latency and learning trials) acquisition of conditioned threat responses compared to cortical learning (60).

Although the existence of thalamo–amygdala pathways in humans has been questioned (61), numerous imaging and electrophysiolgical recording studies support this idea. Emotional stimuli have been shown to elicit an early (under 150 ms) response in the amygdala to emotional stimuli that is unaffected by attentional load or conscious awareness (62–65). This early processing is most sensitive to clear signals of threat (66), and interacts with higher-order regions like PFC to bring emotional stimuli to the forefront of conscious awareness (67). It is thought to serve as a type of "emotional attention" that provides top-down modulation of sensory areas to focus on salient threats in the environment (68, 69).

This initial rapid response is thought to be nonconscious due to its rapid and automatic nature (70). In fact, it has been shown to appear even before visual responses in V1 and to be specific to low spatial frequencies, which indicate its reliance on M neurons

(53). This nonconscious processing of emotion is automatic and persists even in the face of incongruent conscious information (71).

Unseen threatening stimuli have also been found to increase connectivity between amygdala, pulvinar, and superior colliculus in humans (72). Recent neuroimaging studies in humans have supported the existence of such a subcortical route in humans (73–75). Similarly, superior colliculus and pulvinar can be engaged with emotional presentations in patients with striate cortex damage that present no subjective awareness of the stimulus (76). Importantly, these patients can recognize emotional stimuli successfully despite their lack of conscious awareness. This capability, referred to as "affective blindsight" (77), has been shown to be driven primarily by low spatial frequencies processed by the M pathway (78). Even in healthy participants with normal vision, amygdala responses to fearful faces are greatest for low spatial frequency stimuli which also selectively activate pulvinar and superior colliculus (79). Similarly, damage to the pulvinar has also been shown to reduce rapid response of threat in the amygdala (80).

Conscious and Nonconscious Processing of Trauma

Rather than functioning in isolation, nonconscious and conscious processing of trauma works in concert to provide an adaptive response. The fast feedforward sweep of processing carried by the M pathway does not occur exclusively in the subcortical route mentioned above. Processing occurs along the dorsal pathway in an initial sweep to inform top-down modulation from OFC of slower processing in the ventral pathway (33, 37, 43). This influence over the detailed processing of the ventral pathway is intended to modulate the fine features of conscious visual experience. (Fig. 1B). The idea that dorsal pathway representations are nonconscious while ventral pathway representations support the perceptual contents of conscious experience has been suggested since the conception of the two visual pathways. For example, in patient DF, it was observed that ventral pathway damage resulted in visual form agnosia. Nonetheless, DF was able to appropriately adjust her grasp to an object indicating the form was understood in some nonconscious capacity. This nonconscious ability relied on the intact dorsal pathway (34, 35, 81).

So, when a traumatic event is experienced there are conscious and nonconscious processes working in confluence. Nonconscious processes in the subcortical and dorsal pathways guide defensive action and triggering of defensive systems. Conscious processes then come online receiving top-down guidance from the nonconscious processing. The particulars of the traumatic experience are generated from this processing—the individual objects, faces, the scene context, and so on. The memory of these processes will then influence future perception, opening the possibility for intrusive memories and imagery later depending on the characteristics of the observer (82).

Generating Intrusive Imagery

These perceptual processes inform our generative adversarial model of intrusive imagery in disorders like PTSD. The top-down influence of the M pathway represents a generator-like process modulating the bottom-up exteroceptive perception of the P pathway. The resulting representations are monitored by the discriminator along with sensory information. Ultimately the discriminator, residing in PFC, decides whether the perceptual input it is receiving represents reality. In the case of intrusions, one or multiple processes might malfunction. The generator process could become faulty and present such convincing traumatic representations (decoupled from any external input) that the discriminator is fooled into accepting them as reality. Conversely, a faulty discriminator could accept low-probability content from the generator as reality in place of genuine exteroceptive percepts. Perhaps most likely, there could be a deficit in both processes, leading to convincing but false percepts getting past the discriminator and causing an intrusive imagery experience to become accepted as current reality. This would suggest the M pathway is primarily to blame in intrusive experiences, but studies from imagery also point to an important role for the P pathway.

PTSD Intrusions as Visual Imagery Dysfunction?

Empirical evidence indicates PTSD may partially rely on the neural mechanisms of visual imagery, and thus the mechanisms of visual working memory and perception itself.

Though intrusive imagery is involuntary, it is related to the capacity to produce voluntary imagery, e.g., the ability to "see" a red ball in your mind should you try and imagine one (25). For instance, the self-reported vividness of voluntary imagery predicts the experience of intrusive memories following the viewing of traumatic content in healthy individuals (83). These voluntary mental images utilize similar neural representations to visual working memory (84) and perception (85). In fact, the mechanisms are shared so intimately that perceptual learning and fear conditioning can occur from purely imagined content (86, 87). This imagery is also known to directly influence conscious perception. For example, imagining an image can subsequently influence which of two competing stimuli are consciously perceived in a binocular rivalry task (18, 88). These effects are retinotopically specific to spatial location and orientation, and are also associated with individual differences in V1 surface size and excitability, indicating the involvement of low-level sensory areas (89–91).

Imagery's ability to influence conscious perception goes beyond what is voluntarily controlled. Perception continues to be influenced by imagery even after people think they have successfully suppressed a mental image (31). Moreover, suppressed representations are still decodable in visual and executive areas when people are not consciously aware of the imagery (30) This indicates nonconscious imagery involuntarily influences conscious experience. These implications should motivate future studies aimed at understanding the nonconscious influences on conscious experience of PTSD.

Aphantasia as PTSD Protection

Aphantasia is a condition in which individuals are unable to voluntarily produce mental imagery—they are "blind in the mind's eye" (92). If voluntary mental imagery ability is associated with the experience of intrusions (83), then inversely, a lack of mental imagery ability should be associated with lower intrusions. Thus, a total lack of mental imagery perhaps offers protection against intrusions and PTSD.

It has been suggested that people identifying as aphantasics do so as a difference of criterion rather than a genuine deficit in imagery. That is, aphantasics in reality could have a typical inner experience when trying to voluntarily produce imagery, but metacognitively it may not match their criterion for having a "visual experience." Thus, they report having none. On the contrary, a study of self-reported aphantasics found they had no imagery-

based influence on binocular rivalry perception (93). Additionally, aphantasics do not demonstrate an oblique orientation effect during visual working memory tasks, commonly thought to result from recruitment of sensory regions during visual working memory tasks (94). Furthermore, aphantasics confirmed by a lack of binocular rivalry priming also demonstrate no physiological threat response when reading and imagining frightening stories compared to controls while responses to visually presented fearful images remain intact (95) As no sensory component of imagery can be detected in aphantasics, it would seem their claims of no visual experience are accurate.

Despite this lack of object-specific imagery, aphantasics do not seem to lack spatial imagery or mental rotation ability (92, 93, 96). This suggests that aphantasia may be characterized by a deficient P pathway imagery while the M pathway imagery remains intact.

So, while P pathway dysfunction may restrict the tools of imagination, it may serendipitously protect aphantasics from intrusions and the development of PTSD. This highlights the P pathway as potentially critical in PTSD neuropathophysiology—a hypothesis that should be tested in future experiments. However, self-report findings have suggested overall similar responses to traumatic life events between aphantasics and controls, but with a decrease in recurrent and involuntary memory intrusions (97). As the DSM places specific emphasis on intrusive symptoms for diagnosis, aphantasics may be less likely to be diagnosed with PTSD while still experiencing other trauma-related symptoms. Future studies should explore how trauma may affect aphantasics differently.

Consequently, despite top-down prediction from the M pathway being critical for generating traumatic content for intrusive imagery in our model, it alone is not sufficient for the intrusive experience. If insufficient P processing results in a lack of imagery and protection from PTSD, clearly the P pathway must be involved in an intrusive imagery experience. Perhaps a strong predictive prior from the M pathway paired with modest processing from the P pathway creates particularly challenging imagery for the discriminator. Perhaps a malfunction in some specific P pathway processes makes it more reliant on top-down information from the M pathway. If the discriminator is also operating suboptimally, then intrusive experiences may occur, leading to the neurobiology of PTSD.

Emotional Response to Intrusions

While our perspective focuses primarily on the visual aspect of the subjective experience of intrusive imagery, the emotional consequences of these intrusions are equally important. It is not simply the intrusive experience that is so debilitating in trauma-related disorders, such as PTSD and acute stress disorder, but the following evoked emotional response. Experiencing intrusive imagery may generate intense emotional experiences associated with trauma such as anger, guilt, and powerlessness. Furthermore, the experience can generate significant distress as individuals may be worried they are losing touch with reality. Such negative emotional response is a normal reaction to traumatic intrusive imagery, especially when those intrusions feel like the trauma is being experienced all over again. Framing these emotional responses within a higher-order framework similar to our GAN model also has the potential for clinical benefit as we have argued elsewhere recently (6, 82, 98–100). These emotional responses may even directly impact our model presented here. The emotional cascade experienced following intrusive imagery may serve to further heighten the brain's sensitivity to threat by increasing top-down activity of the generator, which may in turn cause more trauma-related imagery to be generated. Additionally or alternatively, this heightened emotional state may lower the threshold for the discriminator to accept threatening imagery and memory leading to further subjective experience of intrusive imagery. These distressing emotional experiences must, therefore, be taken into consideration when discussing the complex interplay of symptoms in trauma-related disorders.

Dual Pathways Fill in the Gaps of Current PTSD Theories

Multiple modern theories of PTSD neurobiology have focused on contextual processing deficits underlying PTSD (101, 102). PTSD patients demonstrate contextual processing deficits both within and outside threat processing situations (102, 103). Other theories have suggested this contextual processing deficit is specifically a problem with associative learning in the hippocampus (104).

However, an fMRI study found widespread dorsal pathway activation during flashbacks from PTSD (105). This result points to general M pathway involvement in the experience. The same study also found lessened activation in the parahippocampal area of the ventral pathway. These data can be accounted for by viewing these representations as generated by an upregulation of M processing with a deficiency in P processing. Similarities should be noted between our model presented here and dual representation theory (101). Both rely on a dual process stemming from the dorsal and ventral visual pathways. However, our model emphasizes perceptual over memory mechanisms while being physiologically grounded in the M and P pathways to explain the contents and form of visually experienced representations. Additionally, contrary to the unlinking of sensory and contextual representations proposed by Brewin et al.'s (101) dual representation theory, our model proposes a direct interaction of M and P processing along with critical reality monitoring processes in PFC. Top-down influences of the dorsal pathway on processing in the ventral pathway result in intrusive images that fool reality monitoring processes in the PFC. Contextual processing deficits may be a byproduct of general P pathway dysfunction in our model.

Our perspective builds on previous theories of intrusive memory as memory or contextual processing alterations by instead suggesting a specific and neurobiologically based mechanism for intrusive memory grounded in vision and consciousness science, which explains how an intrusive memory may come to be experienced subjectively through visual imagery. Indeed, current clinical models of treatment options for PTSD such as imagery rescripting and eye movement desensitization and reprocessing operate on the premise that visual representations play a core role in intrusive memory (106–108). By proposing a mechanistic basis for these treatment interventions, we hope that our framework can be used to guide further research and improve the efficacy of clinical treatment options for PTSD sufferers.

Previous theories have similarly sought to explain the debilitating subjective experience that is at the center of trauma-related disorders. Memory and context accounts have both critically pointed to contextual processing deficits to explain the character of the subjective experience of intrusions (e.g., missing contextual representation in Dual Representation Theory, deficient contextual processing in Contextual Processing Theory). However, our account seeks to add to these ideas by taking clues from consciousness science to explain why memory and imagery intrude

on subjective experience in the first place rather than being dismissed at the nonconscious level. By explicitly considering conscious and nonconscious processes and individual differences in imagery capability, we hope to provide a direct mechanism for the disturbing subjective experience that is at the heart of trauma-related disorders, and which so disproportionately contributes to the effects of this disorder on sufferers' emotional and psychological well-being.

While contextual processing deficits are evident in PTSD phenomenology, contextual information actually increased the occurrence of intrusions in a trauma film paradigm (109, 110). Additionally, a key component in proposed contextual processing deficits is hippocampus structural abnormality in PTSD patients (101, 102). However, hippocampal structural alteration as a consequence of trauma exposure has not been substantiated by empirical evidence. Conversely, it is thought that hippocampal volume may be a risk predictor for PTSD rather than a consequence (111).

Moreover, the only fMRI study examining flashbacks in PTSD patients did not indicate hippocampal involvement in the experience of a traumatic flashback (105). It did, however, find activity in the parahippocampal gyrus whose activity is found to correlate with mental imagery vividness (112), in addition to PFC and dorsal pathway activation as our model predicts. Future studies should look outside the typical hippocampal circuitry as context-based memory distortions have been tied to PFC, lateral parietal areas, and retrosplenial cortex rather than PHC or hippocampal areas (113).

Additionally, PTSD patients demonstrate impaired local processing and increased global bias (114). These altered biases reflect a bias for M pathway processes over the P pathway, corroborating general fMRI results of altered ventral processing in PTSD (115). Conversely, local processing bias actually predicts experience of intrusive imagery following trauma film viewing in healthy participants as well as stronger contextual cueing (116, 117).

These changes in ventral pathway processing could be the mechanism that traumatic experience alters in a pathological way as ventral pathway processing seems to be a predictor of intrusions in healthy participants but is dysfunctional in PTSD. The M pathway has also been hypothesized to extract gist-like information of scenes to enable rapid contextual identification with its top-down predictions (33, 36, 42). So, in the presence of contextual processing deficits in the P pathway, contextual processing could become more reliant on the gist-like low spatial frequency representations in the M pathway. Less detailed contextual representations could lead to easier confusion between traumatic imagery and current reality.

As we discussed in the section regarding imagery and aphantasia, the P pathway must still be performing some important functional role in this process. If aphantasia is characterized by a deficiency in the P pathway, a pre-existing lack of P processing should not be to blame for PTSD development. Perhaps following trauma, an upregulation of M processing leads to strong probability being placed on traumatic predictions compared to bottom-up information from the P pathway. Over time perhaps the post-trauma brain forms a bias for M pathway information over P pathway information. The resultant representations then present a distinct challenge for the discriminator. If the discriminator is faulty as well, then intrusive experiences and PTSD develop. The empirical evidence reviewed thus far supports this view. For the final piece of the puzzle, we must look to evidence of a faulty discriminator in the PFC.

PFC Dysfunction in PTSD

According to our hypothesis PFC dysfunction should be integral in PTSD neurobiology as the site of both top-down predictions and reality monitoring. Stress does indeed lead to dampening of PFC function and PTSD patients tend to present with symptoms that indicate PFC dysfunction (118).

Imaging studies have also provided a wealth of evidence implicating PFC dysfunction in PTSD (119), (120,121), (102),(122), (123, 124). The intensity of flashbacks has also been directly correlated with decreased PFC activation (125).

PFC dysfunction is also a central point in contextual processing theory (102). As most spontaneous activity testing the discriminator remains nonconscious, it could be that memories associated with the nonconscious dorsal pathway are more prone to being involuntarily retrieved. When presented to a faulty discriminator, they are experienced as reality, generating a flashback.

PFC activation in a specific area (Brodmann Area 10) associated with reality monitoring was implicated in experiencing a flashback compared to an episodic memory in PTSD patients (105).

Empirical evidence to date is supportive of reality monitoring in PFC being affected in PTSD pathophysiology. In the context of our model, this suggests a faulty discriminator is likely involved in the experience of intrusions in PTSD. A faulty discriminator in and of itself may not be sufficient for the intrusive experience. Aberrations in the dynamics of the M and P pathways likely also play a key role as we have argued, thus far. Future experiments designed to test this model will be needed to identify which components are most important in determining the subjective experience of intrusions. These experiments will also be useful in identifying individual differences that may be predictive of PTSD development risk.

Generating Hypotheses

The model of intrusive imagery we have presented here generates a number of new testable hypotheses that should be explored in future research. M and P processing contributions can be examined through the use of stimuli using luminance and chromatic or spatial frequency manipulations that exploit M and P response properties. Our model predicts that information biased to the P pathway may have processing deficits while information biased to the M pathway may have a relative processing boost in patients with intrusive imagery. With specific regard to contextual stimuli, our model predicts that contextual information biased to the M pathway may rescue context processing deficits observed in PTSD patients. Compared to controls who have been exposed to trauma without developing PTSD, contextual processing for P-biased stimuli (isoluminant or high spatial frequency) may be specifically impoverished while contextual processing of M-biased stimuli (low-luminance contrast or low spatial frequency) may be unaffected or relatively enhanced.

Our model also makes predictions about individual differences in imagery being pre-existing risk factors for PTSD development. Specifically, people presenting with aphantasia should be at lower risk for developing intrusive imagery-related symptoms in PTSD or other disorders with intrusions. Additionally, our model makes predictions about reality monitoring processes in PTSD patients as well as M and P pathway contributions to reality monitoring and conscious perception. Direct tests of reality monitoring should show impoverished reality monitoring capabilities in PTSD

patients compared to controls. In PTSD patients, top-down feedback from the M pathway should make traumatic predictions from ambiguous sensory stimulation (e.g., fireworks, cars backfiring). In turn, these predictions should cause P pathway representations to be prone to errors in object recognition. We predict these errors would manifest as consciously experienced traumatic content (e.g., sound of a car backfiring is consciously experienced as a gunshot). This could be tested experimentally by multivoxel pattern analysis (MVPA) of opposing contextual scenes (e.g., neighborhood park vs. war battlefield) to see what representation is activated in PFC following ambiguous stimuli (car backfire audio clip). The activation level of this contextual representation should predict how the ambiguous stimulus is consciously experienced and categorized (car sound or gun sound).

Extending the GAN Framework Beyond Intrusive Imagery

Though our current perspective focuses on intrusive imagery in disorders like PTSD and acute stress disorder, we believe this GAN model holds explanatory power for a number of other disorders such as schizophrenia and depression. Although the link between GANs and schizophrenia has been discussed previously (13), our current model extends upon this discussion in several ways. Empirical evidence has demonstrated reduced PFC activation during reality monitoring in individuals with schizophrenia and in individuals that express schizotypal or psychotic traits (13, 126, 127). Additionally, schizophrenia has a history of being associated with deficient M pathway processing (128, 129), with concurrently intact P pathway processing (130). (130). Furthermore, heightened imagery is strongly associated with increased susceptibility to hallucinations (131–135). Together this points to a simultaneous deficit in both the generator and discriminator mechanisms in schizophrenia. Due to the M pathway's specific role in top-down prediction, it is likely that M pathway deficiencies in schizophrenia lead to the prediction of improbable percepts that are increasingly vivid and quasi-perceptual due to intact P pathway processing. These vivid and improbable percepts may be easily accepted by a faulty discriminator, leading to hallucinations. Additionally, compared to trauma-related disorders where M pathway processing is relatively intact, it is possible that the nature of this M pathway deficiency in schizophrenia explains the difference in character between hallucinations found in schizophrenia compared to the intrusive imagery experienced in trauma-related disorders.

In depression, neither reality monitoring capabilities or M and P pathway contributions have been extensively studied. However, the limited evidence available suggests these ideas would be worth investigating in future studies. Depression seems to be associated with a reduction in global processing bias, which is driven by the M pathway (136–138). This could potentially be driven by an uptick in P processing in depression as greater contrast sensitivity in the P pathway has also been observed in depression (139).

Within this framework, the specific symptoms associated with intrusions in PTSD, hallucinations in schizophrenia, or processing deficits in depression would depend on which specific components of the GAN framework are operating suboptimally or out of balance. Based on the relative interactions of these moving parts, debilitating subjective experiences could manifest in a number of forms. Future experiments should explore these possibilities and the potential to treat symptoms by rebalancing these interactions or identify those at risk based on existing processing biases.

Concluding Remarks

Moving forward, a neurobiological understanding of any mental health disorder, including trauma and stressor-related disorders need to be one that satisfactorily explains the subjective experience endured by patients. The model of PTSD intrusions we have proposed here seeks to do just that. Inspired by the generative adversarial process from GANs in machine learning, we proposed a novel perceptual reality monitoring framework based on these perceptual mechanisms to explain intrusive experiences in PTSD. We believe that our GAN framework provides a number of advantages. Perhaps most importantly, this model provides an answer as to why intrusive experiences in trauma-related disorders are subjectively experienced by the observer as well as why memory can be mistaken for current reality. Subjective experience has not been given as central of a focus in previous accounts. Second, we also gain new research targets for potential treatments as well as pathways to improve existing treatments. Explicit consideration of both the generator (including M/P dynamics underlying the generation process) and discriminator components leads to new research targets and potential treatment avenues beyond what has been suggested by previous accounts. Additionally, investigation of these components may help identify pre-existing risk factors for PTSD and acute stress disorder development such as impoverished reality monitoring, hyperactive imagery, and M/P processing biases.

Future studies with this model in mind will need to disentangle the two processes during intrusive experiences. It could be that observed deficits in reality monitoring are a result of learning from a faulty generator post trauma. Conversely, perhaps the generator learns bad practice from a rogue discriminator.

Expanding the conceptualization of PTSD neuropathology to include the mechanisms of perception and consciousness will produce many fruitful experiments to progress understanding of intrusive experiences.

Authors' Contributions

C.A.C., A.J.D., S.G.H., H.L., J.E.L., and V.T-D. wrote and edited the manuscript.

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