

Neural basis of intrapsychic and unconscious conflict and repetition compulsion

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Recently, psychologists, psychiatrists, and neuroscientists have shown interest in scientific data relevant to analytic theory (Bilder & LeFever, 1998; Westen, 1999; Solms & Turnbull, 2002) and in the reformulation of its concepts using advances in cognitive science (Erdelyi, 1985; Kihlstrom, 1987; Horowitz, 1988; D. Stein, 1992, 1997; D. Stein et al., 2006; Turnbull & Solms, 2007; Berlin, 2011). Psychodynamic theories emphasize unconscious dynamic processes and contents that are defensively removed from consciousness as a result of conflicting attitudes. Empirical studies in normative and clinical populations are beginning to elucidate the neural basis of some psychodynamic concepts like repression, suppression, dissociation, and repetition compulsion.

The neural underpinnings of conflict

Repression

S. Freud (1892a) wrote that human behavior is influenced by unconscious processes, which work defensively to manage socially unacceptable ideas, motives, desires, and memories which might otherwise cause distress. He argued (1915d) that repression works defensively to conceal these “unacceptable” mental contents and their accompanying distress, but that the concealed thoughts, emotions, or memories may still influence conscious thoughts and feelings as well as behavior. Mental illness arises when these unconscious contents are in conflict with each other.

Research suggests a link between physical illness and people with repressive personality style (usually measured by questionnaires and/or psychological tests), who tend to avoid feeling emotions and defensively renounce their affects, particularly anger (Jensen, 1987; Schwartz, 1990;

Weinberger, 1992, 1995). The inhibition of conscious access to emotions puts the body, especially the heart and immune system, under significant stress (Westen, 1998). These memories and emotions continue to influence behavior, for example, when a person with repressed memories of childhood abuse later has difficulty forming relationships. Repressed contents may leak into consciousness via a Freudian slip (accidentally revealing a hidden motive), free association, or dreams, or they may be expressed through symptoms (e.g., a repressed sexual desire may resurface as a nervous cough [Breuer & Freud, 1895]).

Studies show that while people who repress report healthy coping and adaptation, objective physiological or cognitive measures indicate that they are hypersensitive to anxiety-provoking information, especially when it is personally relevant (Furnham et al., 2003). For example, in one study (Adams et al., 1996), homophobia was associated with homosexual arousal. Heterosexual men in the study exhibited increases in penile circumference when exposed to both heterosexual and female homosexual videos. However, among those men who identified as heterosexual, only those who also endorsed homophobic ideas showed an increase when exposed to male homosexual stimuli. Homophobia may thus be a response to a threat to an individual's own homosexual impulses causing repression, denial, or reaction formation to such impulses (West, 1977).

The neural mechanisms underlying repression are unknown; however, some studies have revealed neural activity associated with it. People with a repressive personality style have been found to have smaller evoked potentials to subliminal stimuli and to give significantly fewer verbal associations to the stimuli (Shevrin et al., 1969, 1970; Shevrin, 1973). Repressiveness was also related to the presence of unconscious conflict reflected in differential brain responses to subliminal and supraliminal conflict-related words (Shevrin et al., 1996). There is also evidence that subliminal conflicts are resolved without a significant contribution from the anterior cingulate cortex, which is normally active during conscious conflict monitoring tasks (Dehaene et al., 2003).

In a study of memory repression Kikuchi et al. (2010) investigated the neural activity associated with memory retrieval in two dissociative amnesia patients using fMRI. Their findings suggest that the dorsolateral PFC plays an important role in inhibiting the activity of the hippocampus during repression of unwanted memories (Figure 16.1).

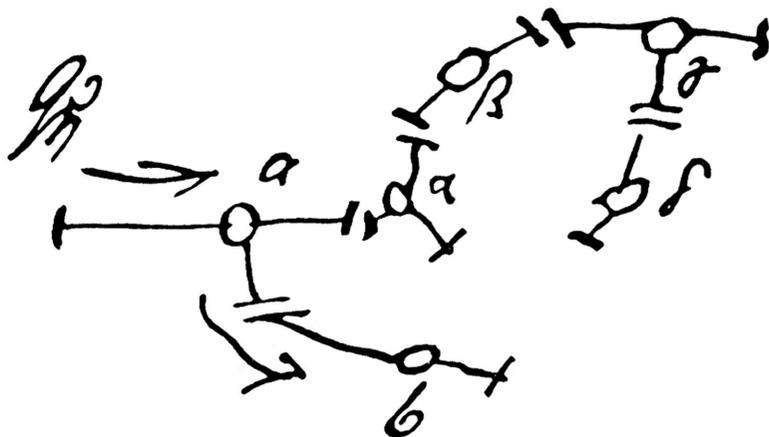


Figure 16.1 Freud's (1895) own sketch of neurons and the flow of neural energy, illustrating his concept of diversion of neural energy via a "side-cathexis." The normal flow of energy (arrow on left labeled $Q'\eta$) is from neuron "a" to "b." Freud proposed that a side-cathexis of neuron "a" would attract the $Q'\eta$ and divert the flow from neuron "b." He believed this postsynaptic attraction of energy or side-cathexis was the neuronal mechanism underlying repression of forbidden wishes in both waking and dreaming (from McCarley, 1998).

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Although some have technical objections to his account (e.g., Koch, 2004), Libet (1966, 1973, 1978, et al., 1964) found that a critical time period for neural activation is needed for a stimulus to become conscious. During neurosurgical treatment for dyskinesias, the patient's primary somatosensory cortex was stimulated with an electrode, eliciting a sensation in a portion of the contralateral hand, wrist or forearm. A train of repetitive 0.5-ms pulses of liminal intensity had to persist for about 500 ms to elicit a sensation. This was known as the minimum "utilization train duration" (UTD). UTD values varied little over time within subjects but varied between subjects from 200–750 ms. Those with a longer UTD exhibited a greater tendency to repression, as measured by a battery of psychological tests (Shevrin et al., 2002). It may be that, just as people with high intelligence may be prone to develop intellectualization as a defense against unacceptable unconscious wishes, people who need a longer time period of neural activation in order to develop a conscious experience of a stimulus may be prone to utilize repression.

Using a clever paradigm and technique called “continuous flash suppression” (Tsuchiya & Koch, 2005; Tsuchiya et al., 2006), Jiang and He (2006) demonstrated that interocularly suppressed (“invisible”) images of naked men and women, that do not enter the subjects’ consciousness, can attract or repel subjects’ spatial attention based on their gender and sexual orientation. Despite being unaware of the suppressed images, heterosexual males’ attention was attracted to invisible female nudes, heterosexual females’ and homosexual males’ attention was attracted to invisible male nudes, and homosexual/bisexual females performed in-between heterosexual males and females. What was particularly interesting was that *heterosexual* males were actually *repelled* by pictures of naked men in that their attention was diverted away from areas of their visual field where invisible naked men were presented. None of the other groups showed this repulsion effect. This appears to be an example of the Freudian concept of repression, that is, the unconscious prevention of anxiety-provoking thoughts or desires (in this case, perhaps latent homosexual desires in heterosexual men) from entering consciousness. Another controversial implication of this experiment is that it suggests that an individual’s sexual orientation can be statistically inferred from their unconscious attentional biases (Koch, 2008). Although these results are only behavioral and do not uncover the neural pathways that enable such unconscious attentional modulation, the authors suggest that because the stimuli were arousing erotic images, the amygdala is likely to play a critical role.

Despite the evidence described earlier, the existence of repression remains contentious, due in part to its association with trauma, and the practical and ethical problems of studying it in controlled animal and human experiments. Therefore, creative paradigms with which to study the mechanism underlying repression in the laboratory are needed.

Suppression

Suppression, the voluntary form of repression proposed by S. Freud (1892a), is the *conscious* process of pushing unwanted information (thoughts, emotions) out of awareness and is thus more amenable to controlled experiments than repression. While some claim that memory repression or suppression is a clinical myth with no scientific support (Kihlstrom, 2002), others have provided evidence for memory suppression (Anderson & Green, 2001; Anderson et al., 2004; Anderson & Hanslmayr, 2014). Memory suppression requires people to override or stop

the retrieval process of an unwanted memory, and this impairs its later retention (Anderson & Green, 2001). Executive control processes can be recruited to prevent unwanted declarative memories (provoked by cues) from entering awareness, and this cognitive operation makes later recall of the rejected memory harder (Anderson & Green, 2001). If suppression by executive control processes becomes habitual over time, inhibition may be maintained without any intention of avoiding the unwanted memory, evolving from an intentional to an unintentional process (i.e., repression).

Anderson et al. (2004) used a “think/no-think paradigm” where participants first learned word pairs (e.g., ordeal-roach), and then, during fMRI, were shown one member of a pair (e.g., ordeal) and told to recall and think about the associated response (e.g., roach) (respond condition) or to prevent the associated word from entering consciousness for the entire 4-second stimulus presentation (suppression condition). Suppression impaired memory. After scanning, cued recall for Suppression items, when given the originally trained cue, was inferior to recall of Baseline items that did not appear during scanning. So suppression during scanning made subjects unable to recollect memories that had been formed pre-scanning, and this memory deficit was beyond what was measured for simple forgetting over time. Further, controlling unwanted memories (suppression) was associated with increased dorsolateral PFC activation and reduced hippocampal activation. The magnitude of forgetting was predicted by both PFC and right hippocampal activations. These results establish a neurobiological model for guiding research on motivated forgetting (suppression) and integrate it with widely accepted mechanisms of behavior control.

Depue et al. (2007) employed Anderson’s (Anderson & Green, 2001; Anderson et al., 2004) think/no-think paradigm but instead used neutral faces as cues and negative pictures as targets. The behavioral evidence showed that subjects effectively suppressed memory. Using fMRI, they found that emotional memories are suppressed by two neural mechanisms: (1) initial suppression by the right inferior frontal gyrus over areas that support sensory elements of the memory representation (e.g., thalamus, visual cortex), proceeded by (2) right medial frontal gyrus control over areas that support emotional and multimodal elements of the memory representation (e.g., amygdala, hippocampus), both of which are influenced by fronto-polar areas. This implies that memory suppression does, in fact, occur and is under the control of prefrontal regions, at least in healthy populations.

In a comprehensive review, Anderson and Hanslmayr (2014) summarize neuroimaging and behavioral evidence that suggests that inhibitory control processes mediated by the lateral PFC are responsible for suppressing awareness of unpleasant memories at the level of encoding or retrieval. These lateral PFC mechanisms interact with brain structures that encode memories, like the hippocampus, and disrupt memory traces and retention.

Dissociation

The concept of “dissociation” was originally put forth by the French psychiatrist Pierre Janet (1859–1947) to describe the “dual consciousness” characteristic of hysteria (Ellenberger, 1970). Dissociation is as a psychological state in which certain thoughts, emotions, sensations, or memories are separated from the rest of the psyche (APA, 2013). Dissociation is not inherently pathological but it is more prevalent in people with mental illness. The *Diagnostic and Statistical Manual of Mental Disorders-V* (APA, 2013) describes dissociative disorders as “a disruption of and/or discontinuity in the normal integration of consciousness, memory, identity, emotion, perception, body representation, motor control, and behavior” and specifies five dissociative disorders: dissociative identity disorder (DID), dissociative amnesia, depersonalization/derealization disorder (DPD; Simeon & Abugel, 2006), other specified dissociative disorder, and unspecified dissociative disorder (Kihlstrom, 2005). Dissociation may also present as a symptom in other psychiatric disorders (Sar & Ross, 2006). We will discuss studies in patients with DPD and DID in particular that give us some insight into the neural mechanisms involved in dissociation.

Depersonalization disorder

DPD is a dissociative disorder characterized by a persistent or recurrent feeling of being detached from one’s mental processes or body, accompanied by a sense of unfamiliarity/unreality and hypoemotionality, but with intact reality-testing (APA, 2013). People with DPD have difficulties with information processing in relation to the dissociative detachment feature of depersonalization, especially in early perceptual and attentional processes, and with effortful control of the focus of attention (Guralnik et al., 2000, 2007; D. Stein & Simeon, 2009). They have also been shown to have attenuated emotional perception, disrupted emotional memory, and

a difficulty in identifying feelings (Medford et al., 2006; Montagne et al., 2007; Simeon et al., 2009).

Sierra and Berrios (1998) put forward a “corticolimbic disconnection hypothesis,” which is supported by functional neuroimaging and psychophysiological studies. This hypothesis suggests that depersonalization occurs via a fronto-limbic suppressive mechanism which is mediated by attention and generates a state of subjective emotional numbing and disables the process by which perception (including that of one’s own body) and cognition become emotionally colored. This emotional “decoloring” results in a qualitative change of conscious awareness and feelings of “unreality” or detachment, which becomes persistent and dysfunctional in people with DPD (Sierra & Berrios, 1998; Sierra, 2009). More specifically, they suggest that hyperactivity of the right PFC (in particular the right dorsolateral PFC) increases alertness, while left PFC activation inhibits the amygdala and other limbic structures (in particular the anterior insula), causing chronic hypoemotionality in DPD (Sierra & Berrios, 1998; Phillips & Sierra, 2003; Sierra, 2009). Understanding the neural basis of consciousness requires an account of the neurocognitive and neurobiological mechanisms that underlie distortions of self-perception such as those seen in the context DPD.

Dissociative identity disorder

DID is a complex, chronic, and severe of dissociative disorders, and it also presents as a symptom in the other dissociative disorders. Challenging the notion of a unitary self-consciousness, DID is characterized by identity fragmentation, rather than proliferation, and is usually associated with a history of severe childhood trauma (Putnam, 1997). DID involves the presence of two or more distinct dissociative identity states, characterized by different emotional responses, cognitions, moods, and perceived self-images that recurrently and alternately take control of one’s behavior and consciousness. Clinical data suggest the “traumatic identity state” (TIS) has access to traumatic autobiographical memories and intense emotional responses to them. But when in the “neutral identity states” (NIS), patients claim amnesia for traumatic memories (coinciding with the notion of suppression) too extensive to be explained by normal forgetfulness. In the NIS they appear to inhibit access and responses to traumatic memories, processing and responding to trauma-related information as if it pertains to neutral and/or non-autobiographical information, thus enabling daily life function.

Neurobiological studies support the validity of the diagnosis of DID and provide clues to the neural basis of dissociation. In the first controlled structural MRI study of DID, Vermetten et al. (2006) found that compared to healthy controls, DID patients had 19.2% smaller hippocampal and 31.6% smaller amygdalar volumes. Ehling et al. (2008) also found that DID patients had smaller hippocampal (25%–26%) and amygdala (10%–12%) volumes than healthy controls, and those who recovered from DID had more hippocampal volume than those who did not. Stress acting via *N*-methyl-D-aspartic acid (NMDA) receptors in the hippocampus may mediate symptoms of dissociation (Chambers et al., 1999). Early life exposure to elevated glucocorticoid levels, released during stress, may result in progressive hippocampal (a target for glucocorticoids) atrophy (M. B. Stein et al., 1997; Bremner et al., 2003). However, stress may not cause hippocampus damage; rather, those born with a small hippocampus and/or amygdala, perhaps owing to genetics, may be at greater risk for DID. In fact, abused subjects without DID had larger hippocampal and amygdalar volumes than non-abused subjects without DID (Vermetten et al., 2006), perhaps helping protect against sequelae of early trauma. Psycho- and/or pharmacotherapy for dissociative disorders may increase hippocampal volume (Vermetten et al., 2003), but longitudinal studies are needed.

Interestingly, electrical stimulation of the hippocampus in epilepsy patients resulted in dissociative-like symptoms, including feelings of déjà vu, depersonalization, derealization, and memory alterations (Penfield & Perot, 1963; Halgren et al., 1978). And ketamine, an NMDA receptor (concentrated in the hippocampus) antagonist, resulted in dissociative symptoms in healthy subjects, including feelings of being out of body, of time standing still, perceptions of body distortions, and amnesia (Krystal et al., 1994).

In relation to an orbitofrontal hypothesis of DID (Forrest, 2001), using SPECT, Sar et al. (2001, 2007) found that compared to healthy controls, DID patients had decreased perfusion (regional cerebral blood flow [rCBF] ratio) in the orbitofrontal cortex bilaterally, and increased perfusion in median and superior frontal and occipital regions bilaterally, and in the left lateral temporal region. Dysfunctional interaction between anterior and posterior brain areas may contribute to the neurophysiology of dissociation. Reinders et al. (2003) found specific changes in localized brain activity (via positron emission tomography [PET]) consistent with DID patients' ability to generate at least two distinct mental states of self-awareness, each with its own access to trauma-related memories.

The rCBF patterns showed involvement of medial PFC and posterior associative cortices (including parietal areas) in the representation of the different states of consciousness. Based on findings with other “disorders” of consciousness (e.g., Laureys, Goldman et al., 1999; Laureys, Lemaire et al., 1999; Laureys et al., 2000, 2004; Laureys, 2005), these highly connected areas have been suggested to be part of the neural network for consciousness.

Data suggest that one brain can generate at least two distinct states of self-awareness, each with its own pattern of perception, reaction, and cognition (Dorahy, 2001; Nijenhuis et al., 2002) and displaying different psychobiological traits that are generally not reproducible in DID-simulating controls (e.g., S. Miller & Triggiano, 1992; Putnam, 1997). Differential responses in DID patients have been reported in electrodermal activity (Ludwig et al., 1972; Larmore et al., 1977), autonomic nervous system variables, arousal (Putnam et al., 1990), EEG (Mesulam, 1981; Coons et al., 1982; Hughes et al., 1990; Putnam, 1993), visual evoked potentials (Putnam, 1992), and rCBF (Mathew et al., 1985; Saxe et al., 1992; Tsai et al., 1999). Brain areas directly or indirectly involved in emotional and memory processing are most consistently reported as being affected in DID (Dorahy, 2001; Nijenhuis et al., 2002).

Physiologic differences across identity states in DID also include differences in dominant handedness (which may indicate opposing hemispheric control of different identity states), response to the same medication, allergic sensitivities, endocrine function, and optical variables like variability in visual acuity, refraction, oculomotor status, visual field, color vision, corneal curvature, pupil size, and intraocular pressure in the various DID identity states, compared to healthy controls (Birnbaum & Thomann, 1996). One patient (BT) with DID in response to trauma gradually regained sight during psychotherapy after 15 years of diagnosed cortical blindness by neuro-ophthalmic examination (Waldvogel et al., 2007). Initially only a few personality states regained vision, while others remained blind. Amazingly, visual evoked potentials were absent in the blind personality states but normal and stable in the sighted ones. This case shows that, in response to personality changes, the brain has the ability to prevent early visual processing and consequently obstruct conscious visual processing at the cortical level. The neural basis of this ability is being explored (Strasburger et al., 2010). Top-down modulation/suppression of activity in the early stages of visual processing, perhaps at the level of the thalamus or primary visual cortex, may be the neural basis of psychogenic blindness (Berlin & Koch, 2009).

Reinders et al. (2006) were the first to compare the response to trauma-related stimuli in the same DID patients in different dissociative identity states. Differences were found between the NIS and TIS, in response to a trauma-related vs. neutral memory, in subjective reactions (emotional and sensorimotor ratings), cardiovascular responses (heart rate, blood pressure, heart rate variability) and cerebral activation patterns (rCBF via PET). When exposed to identical trauma-related stimuli, the two dissociative identity states exhibited different autonomic and subjective reactions and rCBF patterns implicating different neural networks. This extends findings in healthy subjects (Anderson et al., 2004) that memory suppression can be transferred to unrelated memories, which Reinders et al. (2006) suggest may result in psychopathology.

Neural basis of dissociation

Evidence suggests there is “splitting” of consciousness in DID patients. But how does this relate to the neural correlates of consciousness? By what mechanism can multiple selves co-exist or alternate in the same brain? There is remarkable similarity between psychiatric and neurological dissociation syndromes, but the main difference is that the former are conceived as a disconnection between psychic functions like seeing and acting, while the latter are defined in terms of physical disconnection between specialized brain regions like vision and motor areas. But both types of disorders can be considered disorders of integration, the former because of a “functional” or dynamic impairment of connectivity and the latter because of a neuroanatomical lesion.

Thus, what appears to be altered in both neurological disconnection syndromes and dissociative disorders is not so much the degree of *activity* of a brain area or psychic function, but the degree of *interactivity* between such areas or functions. Integration of various cortical and subcortical areas appears to be necessary for cohesive conscious experience (Laureys, Goldman et al., 1999; Laureys, Lemaire et al., 1999; Laureys et al., 2000; Tononi, 2004, 2005). Dissociation may involve disruption of cortico-, thalamo-, amygdalo- or hippocampo-cortical connectivity (Krystal et al., 1998). Many of these connections are excitatory NMDA receptor-mediated and are blocked by the NMDA antagonist ketamine, which results in dissociative symptoms in healthy subjects. Psychopathologies, like dissociative disorders, that defy the apparent unity of the self, may be failures of coordination or integration of the distributed neural circuitry that represents subjective self-awareness (Kinsbourne, 1998).

Hysteria and hypnosis

The French neurologist Jean-Martin Charcot (1825–1893) believed that the transient effects of hypnosis and the inexplicable neurological symptoms of “hysteria,” currently known as “dissociative (conversion) disorder,” involved similar brain mechanisms. In line with this, recent studies in cognitive neuroscience reveal that the brain processes involved in symptoms of “hysteria” are, in fact, similar to those seen in hypnotic phenomena (Bell et al., 2011). Studies also indicate that hypnotizability is associated with a tendency to develop dissociative symptoms, particularly in the area of sensorimotor function, and that suggestions in highly hypnotizable people can replicate dissociative symptoms (Bell et al., 2011). Interestingly, converging evidence indicates that dissociative symptoms, whether simulated through hypnosis or diagnosed clinically, are linked to increased PFC activation. This implies that interference by the prefrontal/executive system in voluntary and automatic cognitive processes is a shared neural feature of both dissociation and hypnosis. However, systematic, well-controlled, and well-designed experiments investigating the neurocognitive basis of dissociation and hypnosis are needed.

The repetition compulsion

The repetition compulsion – the behavioral compulsion expressed by many people to repeat or re-experience certain painful, traumatic experiences from the past – is one of Freud’s most seminal and important ideas and is a primary tenet and guiding force of current psychodynamic counseling approaches (Corradi, 2006). Freud’s idea of the repetition compulsion was most fully elaborated in *Beyond the Pleasure Principle*, where he described the paradoxical phenomenon of people being “fixated” to specific past traumas that “include no possibility of pleasure” (S. Freud, 1920, p. 21). Freud concluded that “there really does exist in the mind a compulsion to repeat which overrides the pleasure principle” (S. Freud, 1920, p. 24) and noted that the “manifestations of a compulsion to repeat . . . give the appearance of some ‘daemonic’ force at work” (S. Freud, 1920, p. 41).

Post-traumatic stress disorder and the repetition compulsion

Perhaps the clearest examples of a repetition compulsion are cases arising from traumas during warfare or from other severe traumas such as sexual abuse. Freud began to more deeply explore the concept of the repetition

compulsion after the conclusion of World War I, when he met and treated many soldiers who were experiencing what Freud called a “war neurosis,” with symptoms that included repetitive dreams during which the soldiers relived and re-experienced devastating traumatic experiences, such as witnessing close friends being blown apart by grenades or artillery. Such patients – who would now be diagnosed as having post-traumatic stress disorder, or PTSD – were “obliged,” Freud said, to compulsively re-experience their trauma rather than “remembering it as something belonging to the past” (S. Freud, 1920, p. 19).

In 1987, Van der Kolk and M. Greenberg made the provocative suggestion that PTSD may be driven by an “addiction to trauma” that is very similar to an addiction to alcohol or addictive drugs. Studies have shown that when old traumas are re-experienced in PTSD, the stress response is activated at very high levels (Newport & Nemeroff, 2000; Liberzon et al., 2007), and several lines of evidence suggest that the stress hormones released by the stress response may become reinforcing in the brain under certain conditions (Piazza & Le Maol, 1997; Montgomery & Ritchey, 2008). The stress response is highly conserved evolutionarily and is extremely similar in all mammals (Nicolaidis et al., 2015). Studies have shown that rats in the laboratory will do work, such as pressing a lever, to receive intravenous injections of stress hormones, much as rats will work to receive other reinforcers such as food or addictive drugs (Piazza & Le Maol, 1997). There is also extensive evidence in both humans and experimental animals that stress hormones and addictive drugs such as cocaine or methamphetamine “cross-sensitize” – that is, a chronically hyperactive stress response, which typically produces chronically high levels of stress hormones, makes a person or non-human animal far more likely to develop an addiction to drugs such as methamphetamine, and developing a drug addiction makes it far more likely that a person or animal will develop a chronically hyperactivated stress response (Yavich & Tiihonen, 2000; Wand et al., 2007; Kippin et al., 2008). In further support of the idea that stress hormones and addictive drugs often have very similar effects in the brain, it has been found (Saal et al., 2003; Ungless et al., 2003) that stress and various addictive drugs, including cocaine, morphine, and nicotine, produce nearly identical modifications in excitatory synapses in the ventral tegmental area (VTA), a brain reward area known to be critical for the development and maintenance of drug addiction.

Part of the normal stress response is the release of β -endorphin – an endogenous opioid closely related to morphine, heroin, and other opiates – which, when released in the brain, has been shown to be associated with pleasure, euphoria, and the reduction of physical or emotional pain (Kelley & Berridge, 2002; Nicolaides et al., 2015). Dopamine, which along with β -endorphin has been shown to be a critical neurotransmitter involved in drug addiction and alcoholism, is also released at very high levels as a consequence of the stress response in reward areas of the brain, such as the nucleus accumbens or ventral tegmental area, that are known to be critically involved in drug addiction and alcoholism (Lekners & Tracey, 2008). Thus, as originally suggested by Van der Kolk and M. Greenberg (1987), it is possible that when an old trauma is reactivated, the release of high levels of dopamine and β -endorphin, and perhaps other stress hormones such as cortisol, acts as the equivalent of a drug exposure, thus reinforcing the pattern of repeatedly re-experiencing the old trauma much as a drug addiction is reinforced by repeatedly taking addictive drugs. Furthermore, much as substance abusers repeatedly and compulsively continue to self-administer drugs despite negative consequences, people with PTSD repeatedly re-experience and re-engage in distressing and painful traumas.

Physical and emotional self-harm

Drug addiction and the repetition compulsion are widely considered to be destructive and maladaptive behavior patterns (Nielsen & Germain, 2000; Paulus, 2007), and thus may be closely related to what are perhaps the clearest examples of maladaptive behavior – overt patterns of self-harm, such as with “cutters” who compulsively and repeatedly make painful cuts to themselves on various parts of their bodies (Strong, 1998; Klonsky, 2007). Other common types of self-harm include head banging, skin picking, interfering with wound healing (dermatillomania), and hair pulling (trichotillomania) (Laye-Gindhu & Schonert-Reichl, 2005). Self-harming behavior involves self-administered pain, and various types of physical pain, including sustained pain in a jaw muscle (Zubieta et al., 2001) and burns and electrical shocks (Becerra et al., 2001; Schmidt et al., 2002), have been shown to strongly activate the stress response and to release high levels of dopamine and β -endorphin into reward areas of the brain. The cuts made by a cutter have also been shown to release large amounts of β -endorphin (Sandman et al., 1997; Sher & Stanley, 2008; Bresin & Gordon, 2013). Sandman et al.

(1999) found that administering μ -opioid receptor blockers that interfere with the activity of endogenous opioids such as β -endorphin, significantly reduces the amount of self-harm in patients, suggesting that at least part of the underlying behavioral reinforcement for self-harming acts may result from β -endorphin release within the brain during those acts. In further support of a possible relationship between self-harming behaviors, addiction, and PTSD, Pitman et al. (1990) found that Vietnam veterans who were triggered into old war traumas by watching videos of dramatized combat released large amounts of β -endorphin while at the same time experiencing pain reduction. Pain reduction was found to be absent with the administration of naloxone, a blocker of the μ -opioid receptor, suggesting that the pain-reduction effect was a direct consequence of β -endorphin release in the brain (Nicolaidis et al., 2015).

Emotional pain appears to activate many of the same pathways in the brain as physical pain (Eisenberger & Lieberman, 2004), and studies have shown that emotional pain, which is typically triggered by and re-experienced in any repetition compulsion, can also release significant amounts of endorphin in the brain. Brain imaging studies using PET found that when people with major depressive disorder (Kennedy et al., 2006) or borderline personality disorder (Prossin et al., 2010) were told to think of sad or painful thoughts from their past – such as a painful romantic breakup – significant amounts of β -endorphin were released in their brains as a direct and immediate consequence of the painful thought.

Depression is typically a prominent feature of PTSD, and clinical depression, similar to PTSD, often involves repeatedly revisiting or ruminating about painful events from the past (Beck, 2006) – a compulsive thought pattern that appears to qualify as a repetition compulsion. An apparent neural correlate of anhedonia, another major feature of depression, is hypoactivation in reward areas such as the ventral striatum, which, in clinically depressed patients, has been shown to be hypoactivated in response to positive words (Epstein et al., 2006) and happy autobiographical or facial-expression stimuli (Keedwell et al., 2005). These studies, combined with PET studies showing that β -endorphin is released in response to sad or painful thoughts in people with clinical depression (Kennedy et al., 2006), suggest that people with clinical depression not only have a reward deficit in receiving healthy rewards but also, perhaps as a compensation, may compulsively recall and ruminate about painful memories, which then release β -endorphin in their brains. Thus similar

to cutters who self-administer physically-painful cuts, people with clinical depression may unconsciously, in effect, “self-administer” emotional pain that, by releasing β -endorphin and perhaps other stress hormones in the brain, reinforces the ruminative thought pattern (Montgomery & Ritchey, 2008).

Evolutionary mismatch, homeostasis, and addictive patterns

Montgomery and Ritchey (2008, 2010) have proposed that nearly all psychological disorders, including clinical depression and anxiety disorders, are driven by literal biochemical addictions to exaggerated and inappropriate negative emotional states, such as fear, anxiety, or emotional pain, that are compulsively re-experienced in a repetition compulsion. The model further proposes that the dysfunctional thought and behavior patterns that are associated with maladaptive emotional responses are set up by “evolutionary mismatch” effects that are generated whenever human beings or animals live in environments, such as modern cities or cages, for which they are not biologically or evolutionarily adapted (Glantz & Pearce, 1989; A. Stevens & Price, 1996; Montgomery & Ritchey, 2008).

Overt behavioral or psychological dysfunctions have only rarely been observed, for example, in wild chimpanzees. However, when chimpanzees are placed in environments, such as cages, that differ significantly from their environment of evolutionary adaptedness, psychopathologies that are very similar to human psychopathologies tend to arise at high frequencies (Brüne et al., 2006). Hence cage-living chimpanzees, particularly when individually housed, frequently demonstrate abnormal, often overtly self-harming, behavior patterns such as head banging, compulsive rocking, self-mutilation, and urine drinking. Although dysfunctional behavior patterns such as these are very common in captive chimpanzees, most of these patterns have never been observed in wild populations of chimpanzees.

Humans appear to exhibit psychological evolutionary mismatch effects that are very similar to the evolutionary mismatch effects observed in non-human primates. While rates of clinical depression, for example, in modern, industrialized cultures such as the United States are roughly 10%–20% (Blazer et al., 1994; Ilardi, 2009), clinical depression may be relatively rare in contemporary hunter-gatherer cultures, whose lifestyles appear to be broadly similar to those of our evolutionary ancestors (Lieberman, 2013). An extensive study of the Kaluli hunter-gatherers of the New

Guinea highlands, for example, found that of over 2,000 of these hunter-gatherers who were interviewed in detail, only one person was found to exhibit symptoms indicative of possible clinical depression – a rate of less than 0.05% (Schieffelin, 1985). Other cross-cultural studies strongly support evolutionary mismatch effects in the etiology of depression, generally showing that the more industrialized, or “modern,” a culture or society is, the higher its rate of depression will typically be (Ilardi, 2009). Rates of anxiety and depression, for example, have been shown to be far higher in fully industrialized cultures than in pre-industrial cultures or than in, for example, Amish cultures, which maintain many elements of a traditional, 18th-century lifestyle (K. Miller et al., 2007). One could argue that some of these studies may be biased because Western diagnostic criteria have been applied to non-Western cultures, where the social and cultural expression of psychological disorders may differ from Western models. However, this potential bias does not appear to apply to the Amish, who, although only semi-industrialized, are completely Western, and whose rates of anxiety and depression are about half of those found in the surrounding, fully industrialized Western culture (K. Miller et al., 2007). This possible bias also does not account for studies showing that rates of anxiety and depression, as well as rates of psychopathologies such as schizophrenia, are significantly higher in urban areas compared to rural areas within the same Western culture (Lederbogen et al., 2011). All of these data taken together appear to support the existence of significant evolutionary mismatch effects in the etiology of various psychological disorders.

The dysfunctional thought and behavior patterns that are part of any specific repetition compulsion, and that may, in turn, be strongly associated with states of evolutionary mismatch, consistently and maladaptively drive a person out of equilibrium, or homeostasis. Paulus (2007) proposed that psychiatric disorders, such as drug addiction, are associated with “altered homeostatic processing” that may lead to non-homeostatic decisions, such as compulsively taking addictive drugs. Similarly, Montgomery and Ritchey (2008) proposed that nearly all psychological disorders – including the repetition compulsion and associated states of chronic anxiety or depression – are generated by a “non-homeostatic drive” that consistently acts to throw a person, both physically and emotionally, out of homeostasis. This non-homeostatic drive may be generated because the stress response, which is intimately associated with any non-homeostatic state, releases neurochemicals such as β -endorphin that may be rewarding

to the brain and thus may reinforce the thought and behavior patterns that generate the maladaptive states of non-homeostasis. These rewards, however, may often be unconscious and not associated with overt states of “pleasure” (Montgomery & Ritchey, 2010). Studies have indicated, for example, that low concentrations of ingested methamphetamine, though not consciously perceived as being rewarding, may influence behavioral decisions (Hart et al., 2001).

This proposed non-homeostatic, or “addictive,” drive that leads to maladaptive states of stress and disequilibrium, frequently conflicts with and directly opposes the functional, adaptive “homeostatic drive,” which is biologically and evolutionarily designed to keep humans and other animals in states of homeostasis whenever possible (Montgomery & Ritchey, 2008; Craig, 2009). The importance of the general maintenance of homeostasis to any organism, and particularly to mammals, is a fundamental principle of modern biology (Cooper, 2008). Craig (2003) has suggested that pain is a “homeostatic” emotion that is at once a subjective feeling but that is also biologically or evolutionarily designed to create a behavioral drive state intended to re-establish a state of homeostasis. The maintenance of synaptic homeostasis in the brain is also seen as being critical for adaptive neural functioning (O’Leary et al., 2014; Wang et al., 2014). The non-homeostatic drive, however, is proposed (Montgomery & Ritchey, 2010) to consistently generate inappropriate states of non-homeostasis, particularly emotional pain and distress, that serve no functional purpose, but that are instead dysfunctionally and unconsciously reinforced by the release of neurochemicals such as β -endorphin during the stress response. In this view, the dysfunctional and maladaptive non-homeostatic drive – enabled by evolutionary mismatch effects and reinforced by rewarding neurochemicals released by the stress response – consistently opposes the healthy, functional homeostatic drive, and may represent the unconscious “daemon force” that Freud long ago observed as being an essential part of the repetition compulsion.

Conclusion

Freud had the foresight to look to the brain for answers (Figure 16.1), but his efforts were limited by the mechanistic understanding and technologies available at the time. New advances in neuroscience and technology are now enabling the neurobiology of the dynamic unconscious that

Freud envisioned to come to fruition (e.g., Solms, 1995; Ramachandran, 1996; Vuilleumier et al., 2001, 2003; Vuilleumier, 2004, 2005; Berti et al., 2005; de Gelder et al., 2005). In the process, a good deal of what Freud originally put forth based solely on clinical observations has been revised, refined, and enhanced (Guterl, 2002). But this is to be expected as the initial insights of every discipline in its early stages requires modification over time (Turnbull & Solms, 2007). Only by studying precisely how the human brain processes information will we fully comprehend the true nature of the dynamic unconscious (Tallis, 2002). Devising novel ways, using modern technology, to empirically test dynamic unconscious processes will help unveil their neural basis and ultimately lead to more effective treatment options for psychiatric patients, completing the task that Freud began over a century ago.

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