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FRONTOLIMBIC AFFECTIVE BIAS AND FALSE NARRATIVES FROM BRAIN DISEASE

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Abstract

Since the nineteenth century, clinicians and investigators have systematically evaluated the origin of delusions and psychotic thinking. One major clue to understanding the neurobiological underpinnings of delusions is the emergence of false narratives from brain disease. In addition to delusions themselves, there are a range of other false narratives not due to deliberate lying and resulting from neurological disorders, including provoked confabulations, fantastic confabulations, false memories, magical thinking, dream delusions, and “fantastic thinking”. A comparison of their characteristics, similarities, and differences suggest a hypothesis: despite different sources for their false narrative experiences, such as unusual thoughts or perceptions, all false narratives from brain disease involve erroneous or mismatched “affective biases” applied to the experiences. Affective labels usually signal the sense of rightness, sense of familiarity, and the external vs. internal origin of an experience, and they can be altered by limbic neuropathology. The location and involvement of neuropathology that facilitates false narratives involves frontolimbic regions and their connections, particularly on the right. Future investigations can focus on frontolimbic mechanisms involved in the provision of the intrinsically-linked affective biases, which indicate the nature and external/internal origin of experiences.

INTRODUCTION

Delusions are a primary symptom of psychosis. There are many classifications of delusions, but they all share the concept of false narratives that do not correspond to real events of personal history and are not deliberate deceptions. Despite 130 years or more of discussion as to the nature of delusions (1), clinicians and investigators do not understand their mechanism and origin in the brain. Current knowledge of the different types of false

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narratives from neurological disorders may clarify the origin of delusions, and, thereby, shed light on the nature of psychosis. A broad characterization of false narratives reported from brain disorders include delusions, provoked or fantastic confabulations, false memories, magical thinking, dream delusions, magical thinking, and even the phenomena of "fantastic thinking" (2). A comparison of the similarities and differences of these different false narratives, and the neuropathological localization leading to these reports, suggests a hypothetical common mechanism underlying delusions and, perhaps, psychosis in general.

HYPOTHESIS/THEORY

False narratives result from a mismatch, or absence, of the appropriate, frontally-mediated affective bias as applied to different sources of experience. Although not entirely new (3), compared to other novel formulations (4), this hypothesis gives priority to the altered feeling states that motivate erroneous interpretations, rather than to alterations in the content of the experiences themselves. Brain disorders alter the affective labels for experiences such as cognitive/analytic (thoughts), perceptual, imaginative events, or abnormally reconstructed memories. "Affective bias" is used here as broadly representing values, attitudes, or judgments prompted by automatic feelings (5, 6), which are often unconscious and unrecognized, that underlie experiences, such as the feelings of familiarity or strangeness that accompany perceptions, memories, or internal vs. external events. Linked intrinsic feelings are fundamentally frontolimbic, originating in mesial frontal regions, including ventromedial prefrontal cortex (VMPFC), anterior cingulate cortex (ACC), frontal-insular cortex (FIC), and their frontal-subcortical connections. These neuroanatomical areas participate in an unconscious evaluation that labels thoughts, perceptions, or other experiences (7). Ultimately, this hypothesis, of an erroneous affective bias for experiences, could lead to a new conceptualization on the management of delusions and greater emphasis on targeting "emotional" dysfunction.

EVIDENCE FROM BRAIN DISEASE

This hypothesis is informed from recurrent reports of the type of neurological patients and lesions that result in delusions and other false narratives (2, 8). A delusion is a fixed belief that is false but firmly held with absolute conviction and despite all evidence to the contrary (9). Delusional beliefs are unshakable and often implausible to others; a perverted view of reality that departs from consensual reality (10). The content of delusions vary from the persecutory to the grandiose, but all seem to involve the misinterpretation of an experience, either internally generated or externally perceived, as having an altered emotional meaning. For example, content specific delusion of Capgras, De Clérambault, or Cotard syndromes indicate disturbed feelings of person familiarity and trust, false beliefs of requited affection, or personal feelings of degeneration or dying, respectively. Two neurological conditions are illustrative. After schizophrenia, Alzheimer's disease (AD) may be the second most common causes of delusions. Content specific delusions based on impaired memory, such as the delusion of theft or of the "phantom boarder syndrome" are common in AD as the disorder spreads to the right frontal lobe and couples feelings of apprehension and distrust with the perception of lost or moved items (11). In addition, an interictal psychosis may occur among epileptics who have frequent and long-standing temporal lobe epilepsy with

frequent epileptic activity in limbic structures (12). Temporal limbic areas are intimately connecting with mesiofrontal areas in a broader limbic region. A further clue is the epileptic emergence of déjà vu, jamais vu, déjà vecu, and other abnormal feelings of familiarity associated with experiences (13).

In contrast to delusions, confabulation are false recollections that create a “coherent self-narrative” of oneself in the presence of amnesia or in the context of an imagined experience (14). Although confabulations have similarities to delusions, they are usually quite distinct (15, 16) (See Table 1). Simple provoked or “momentary” confabulations are memory-related misstatements resulting from attempts to fill-in memory gaps often in response to questions (8, 15, 17). They occur in amnestic disorders such as Wernicke-Korsakoff syndrome, herpes or limbic encephalitis, ruptured communication artery aneurysms especially anterior, strategic diencephalic strokes, traumatic brain injury, nicotinic acid deficiency, multiple sclerosis, hypoxic-ischemic injury such as from attempted hanging, normal pressure hydrocephalus, frontotemporal dementia (FTD), and focal frontal lesions (8, 18). These confabulations differ from delusions in the usual involvement of memory impairment, a fleeting or variable nature, and in the absence of systematized beliefs held with certainty and against evidence to the contrary (19, 20). They are motivated by a momentary familiarity for past overtly similar events misapplied to explain current non-remembered experiences.

In addition to provoked confabulations, there are spontaneous confabulations, which are often “fantastic” and can be difficult to distinguish from delusions, suggesting overlap between these two types of false narratives (21). Spontaneous, “fantastic” confabulations are rarer grandiose or impossible statements that are internally-generated but persist as externally true despite a wavering of certainty when challenged (22–25). They tend to be pleasant and self-enhancing and can be associated with a wish fulfillment, vivid imagination or daydreams, and proneness to fantasy and stories or embellishment (2, 22, 23). These patients cannot emotionally label their internal daydreams with the appropriate feelings of doubt as to its internal source and have an attenuation of the intuitive “feeling of rightness” that something is true (15, 26, 27). Fantastic confabulations are usually associated with significant VMPFC and other prefrontal pathology (23, 26–28). Spontaneous confabulations have some important similarities with delusions (15, 16, 29). In FTD, the occurrence of both delusions and fantastic confabulations emphasizes the spectrum of false narratives from frontal lobe disturbances in this disorder, specifically erroneous affective bias indicating doubt of external truth for thoughts or internal daydreams (7).

Both forms of confabulations are internally-generated external-world attributions and associated frontal-executive deficits (21,23, 30). Among patients with either form of confabulations, there is a bias towards identifying imagined and often wish fulfilling events as occurring externally (23, 26, 31), resulting in inability to tell real memories or events from internally-generated thoughts (15, 23, 27). These observations seem to indicate a disturbed affective bias that provides an intuitive, automatic, and unconscious “feeling of rightness” for experiences (7). Some studies show a positive bias for false recognition among those with confabulations (32). Affective bias regarding an experience or the absence of uncertainty regarding an experience, may be incorrectly linked to it, resulting in a false narrative (7, 21).

False memories are another form of false narratives that can occur from brain disease (33). In 1886, Emil Kraepelin described “pseudoreminiscences”, or hallucinations of memory, that arise spontaneously in the mind and appear as false narratives (1, 34). False memories overlap with confabulations and may occur in the same patients. Memory is modified with each reconsolidation so that it can lead to the retrieval of distorted information (35, 36). Memory is a process or active modification and reconstruction with each retrieval, and parts can be erroneously retrieved or be illusory if there is an affective bias in the reconstruction or reconsolidation(35), including from free-floating or irrelevant memory fragments and fuzzy-traces with dilapidation of details (36, 37), or an abnormal attribution of internal vs. external source or memory trace (27). Memories may be biased because of emotionally-facilitated or “free-floating” memory fragments or an attention to a “verbatim” level of detail while retaining the basic “gist” of memory (37). Studies show a systematic affective bias for more pleasant places when there are false beliefs about place (38). Other studies implicate the prefrontal grey matter, particularly VMPFC, with false recollections and recognition (39–41), as well as the occurrence of frontal-executive dysfunction with disturbed organization for retrieval and ordering of retrieved memories (15, 42).

Four other rarer false narrative types occur in brain disease. First, magical thinking refers to erroneous beliefs of a relationship between one’s thoughts or actions and resultant but unconnected actions or events in the real world. One example would be the belief that wishing or thinking about someone’s demise could precipitate their death. Magical thinking can occur not only in patients with schizophrenia or other mental illness, but also in those with prefrontal lesions, especially if they are predisposed because of religious or spiritual beliefs to altered feelings of causality (43). Second, dream delusions are false narratives that emerge from persistent dream memories, most commonly in narcolepsy, and possibly in dementia with Lewy bodies (44). Difficulty distinguishing dreams from reality may be related to frequent sleep-wake transitions, the intrusion of aspects of rapid eye movement sleep into waking, or to the abnormal encoding of dream content into long-term memory (9, 44). Dream delusions are associated with heightened feeling of reality of fantastic intrusions from sleep (9). Third, the phenomena of fantastic thinking occurs in some patients with frontal disease. It is distinguished from spontaneous confabulations in that these patients know and acknowledge that their narratives are in their head and originate internally, sometimes from daydreams and often with wish fulfillment (24). In fact, fantastic thinking usually emerges as the product of vivid daydreaming or dreams, and may be facilitated by a predisposition to imagination. FTD is a cause of fantastic thinking where “thinking may be as good as doing it” (2), and many false narratives among patients with FTD are actually fantastic thinking (2, 45). Finally, a last consideration is a particularly emotionally overvalued idea that is pursued by the patient beyond bounds of reason and dominating his/her life. Overvalued ideas may apply to some of the compulsive-like behaviors of FTD and Parkinson’s disease.

NEUROPATHOLOGY

Disease in the mesial frontolimbic regions is implicated in patients with false narratives. Patients with delusions and confabulations and brain disorders usually have frontal dysfunction with impairment in accurately evaluating the truth of retrieved or generated

information (21,27, 30). When patients accept false narratives as real, this frontal dysfunction may involve impairment in self-monitoring mediated by frontolimbic medial and orbital frontal regions. Studies show decreased prefrontal cortex gray matter density correlates with false memories or recognition (40). Some patients with frontal hypometabolism have unfettered imaginations characterized by wish-fulfillment, which may be likened to persistent dreaming. Delusions have been associated with frontal lobe abnormalities, particularly on the right (11), and, patients with Wernicke-Korsakoff syndrome, a common source of provoked confabulations, have frontal-executive dysfunction (18).

Confabulations and other false narratives appear to originate from prefrontal disease particularly involving VMPFC and the adjacent orbitofrontal cortex (OFC) (31, 46, 47). Neuroimaging studies indicate that VMPFC is involved in confabulations and false memory (39), source monitoring and external origin of experience, and with identifying whether imagined words were previously seen (28). The VMPFC is associated with correct retrieval of a narrative with its feeling of rightness (42), decoupling in false belief attribution (48), and source monitoring with temporal context (47), and all these attributes can be disturbed with pathology (7, 21, 42, 48, 49). The adjacent OFC (posterior, medial) may also have a role in producing false narratives as this region affects suppression of interference from thoughts, prior anticipations, and irrelevant memories traces (15, 17, 21,50–52). Disease in the posterior medial OFC, or “orbitofrontal reality filter”, is associated with behaviorally spontaneous confabulation (53, 54). The combination of VMPFC and OFC damage may be necessary for fantastic or spontaneous confabulations (15). Finally, the right hemisphere may be involved more than the left. The right PFC is mainly activated with false recollections (39), and the right VMPFC/OFC may be responsible for adding the emotional label to experiences (51).

Other neuropathological localizations have resulted in false narratives. Delusions have been associated with lesions in the heads of the caudate nuclei (CN) (55), and in schizophrenia, studies associate delusions of reference and negative symptoms with decreased gray matter density in the CN (56, 57). Right CN strokes can present with content-specific delusions and reduced metabolism in the inferior prefrontal cortex (55); however, left CN head strokes can also present with recurrent delusions, disturbed sleep, and decreased cerebral blood flow in the frontal lobes (58). Investigators speculate that frontal hypoperfusion or hypometabolism associated with CN infarctions are associated with delusions and other false narratives, presumably from disruption of fronto-subcortical circuits traversing the heads of the CN (55, 58–61). In a review of neuroimaging studies in schizophrenia, the greatest gray matter reductions over time occurred in medial frontal gyrus, ACC, and insula (62), and psychosis may be linked to dysfunction of a dorsal fronto-striato-thalamic circuit (63). Moreover, a review of longitudinal functional magnetic resonance imaging studies suggests that treatment of schizophrenia induces changes in prefrontal cortex and ACC (64).

CONSEQUENCES AND DISCUSSION

Patients with medial frontal (“frontolimbic”) disease may develop false narratives from disturbed or absent affective biases or labeling of specific experiences. False narratives differ

in the targeted experience, which can be thoughts and beliefs, perceptions, memories, imaginations, or breakthrough dreams. These experiences become delusions, confabulations, false memories, and other false narratives when there is an erroneous mental feeling towards the source, or an incorrect affective bias. This erroneous intrinsic label alters the interpretation of experiences, thought, or action, independent of his or her conscious awareness of the presence or absence of affective bias (5). This hypothesis is in accord with the emotional and motivational aspects of the frontolimbic role in motivating complex human behavior. Future research may explore this model of delusions and false narratives derived from disturbances observed among patients with brain disorders. Specifically, investigators can test this hypothesis with studies of unconscious affective bias among patients with false narratives.

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TABLE 1:

Characteristics of False Narratives

	DELUSIONS	PROVOKED CONFABULATION	FANTASTIC CONFABULATION	FALSE MEMORY	MAGICAL THINKING	DREAM DELUSION	FANTASTIC THINKING
Certainty or absolute conviction of an external world origin	+	−	+	+	+	+	−
Incorrigibility against all evidence	+	−	±	+	+	+	±
Implausibility or highly improbable	±	−	+	−	+	±	+
Factor-Dependent. eg provocations, REM breakthrough	−	+	−	−	−	+	−
Persistent Ongoing vs past memory deficit	+	−	+	−	+	±	±
Short-term Consistency	+	−	−	+	+	+	−
Multiple False Narratives possible	±	+	+	−	±	±	+
Frontal Dysfunction often present	+	+	+	+	+	+	+