24.

FALSE TAGGING THEORY

TOWARD A UNITARY ACCOUNT OF PREFRONTAL CORTEX FUNCTION

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> I started out by trying to find a unitary concept, but as I moved along, it became rather clear that no single-factor hypothesis could carry one far enough to cover all the manifestations of frontal lesions. And yet the thing that is so tempting to me ... is to think that there may be a family resemblance among symptoms, even among those which seem in part dissociable.
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> H. L. TEUBER (1964, P. 442)

Perhaps the most bewildering mystery in contemporary neuroscience is the functional mechanics of the prefrontal cortex. No neural region has so stubbornly refused to divulge its operational secrets as the most anterior association cortex, to which some early investigators ascribed the highest intellectual and moral functions (Ackerly, 1935; Brickner, 1936; Goldstein, 1936; Goldstein & Katz, 1937; Halstead, 1947; Rylander, 1939, 1943). At the same time, researchers have repeatedly noted the striking lack of behavioral deficits following removal of large areas of the prefrontal cortex and even went so far as to deny any special importance of human prefrontal structures (Hebb, 1939, 1945; Mettler, 1949; Pollack, 1955; Teuber, 1959, 1964; Teuber, Battersby, & Bender, 1951). In the past few decades (see the first and current editions of this book), research has made great advances in our understanding of the prefrontal cortex and its functions. Innovative methodologies such as single-unit neuronal recording, lesion-symptom mapping techniques, transcranial magnetic stimulation (TMS), event-related potential (ERP) recordings, and functional neuroimaging have provided a wealth of data regarding prefrontal cortex function. Yet, over 150 years after Harlow (1848) published his seminal observations of the prefrontal patient Phineas Gage, and despite forecasts for present-day clarity (Knight & Stuss, 2002), much remains obscure.

A central challenge to the prefrontal cortex theorist is the confusing array of cognitive deficits and behavioral abnormalities that can follow damage to the human frontal lobe, in terms of both their disparate nature and their wide variation in presenting symptoms. Often patients with frontal lobe lesions have no primary neurological deficits, and demarcation of their cognitive and behavioral problems into subtypes is quite difficult. The rubric "frontal lobe syndrome" has been used to describe the pattern of cognitive and behavioral deficits following the onset of frontal lobe lesions; while vague and imprecise, this term has never completely disappeared from the literature. Clinical tests and observations tend to reveal perseveration, disinhibition, confabulation, and distractibility as frequent symptoms (Damasio, Anderson, & Tranel, 2012; Moscovitch & Wincour, 2002; Stuss & Benson, 1984). Patients with prefrontal lesions also tend to evince impairments in "executive functions," the unsettled concept used to describe such processes as planning, decision making, judgment, and self-perception (Tranel, Anderson, & Benton, 1994). Beneath these problems tends to be a consistent flat or blunted affect that usually presents as an apathetic state (Damasio & Van Hoesen, 1983). Indeed, given the motley assortment of symptoms following damage, limitations in methodologies, discrepancies in the literature, the disparate and vague functions attributed to the region, and its structural complexity, one wonders whether it is even possible for the prefrontal cortex to recursively solve the riddle of itself. Perhaps, however, there is an undiscovered unifying principle that cuts across the heterogeneous, amorphous functional concepts, which can reconcile the strange array of symptoms following damage. This chapter indulges Teuber's (1964) temptation (see the introductory quote) and seeks to unite the functional concepts often attributed to the prefrontal cortex with a single process by examining the peculiar deficits following the onset of prefrontal cortex lesions with a new theoretical model.

We begin the chapter with a look at the debate between homogeneity of prefrontal cortex functioning versus heterogeneity of functioning with regional fractionation. Anatomical evidence for each view is highlighted, followed by our take on the homogeneity/heterogeneity debate. This sets the groundwork for a new perspective on the core function of the prefrontal cortex. Next, we offer our perspective on how the mental processes of belief and doubt map onto neuroanatomical regions, followed by a description of the False Tagging Theory (FTT), our model of the basic prefrontal cortex computation. Preliminary evidence directly supporting our model is presented, followed by an elaboration on the theory and its relevance to an influential social psychological theory. The remainder of the chapter aims to reconcile the array of symptoms following damage to the prefrontal cortex in light of the FTT. First, perseveration is examined in both reversal learning and behavioral extinction paradigms. This discussion segues into an analysis of prediction errors and learning from negative feedback. Second, memory retrieval errors are examined in the context of the FTT, including pathological confabulation as well as temporal memory deficits, contextual memory deficits, and planning deficits. Finally, distractibility is examined through this prism and a parsimonious answer to the mnemonic, inhibitory, or attentional debate of prefrontal cortex functioning is offered. This is followed by a discussion regarding delusion and the implications of the FTT for schizophrenia. We argue that the ramifications of the FTT have the potential to unify prefrontal cortex functions and suggest a clear path toward understanding many psychiatric disorders such as schizophrenia.

HOMOGENEITY VERSUS HETEROGENEITY OF FUNCTIONING

One dilemma in the field is whether the prefrontal cortex is a functionally heterogeneous area, regionally fractionated to perform isolated computations (Stuss et al., 2002), or a functionally homogeneous area where various regions can adaptively code any information that is relevant to current behavior (Duncan & Miller, 2002). Much of the field has allied with heterogeneity of frontal lobe functioning (Stuss & Knight, 2002), attempting to localize various cognitive and emotional functions, such as executive functions, working memory, and emotional regulation, to discrete focal regions. However, several problems have been identified that are not easily overcome in the regional fractionation paradigm:

1. There is a common pattern of frontal recruitment for many diverse tasks involving widely differing psychological domains, such as perception, response selection, task switching, problem solving, language, and episodic memory (Duncan & Owen, 2000). In addition, single-unit recordings have revealed that the rule relevant to current behavior appears to drive neuronal activity, not the cue or sensory conditions (Asaad, Rainer, & Miller, 2000). Thus, the same prefrontal neuron codes a relevant rule but does not code for individual stimuli or individual actions (Duncan & Miller, 2002). The prefrontal neuron's relative lack of preference for differing stimuli may explain the difficulty in finding the theorized double dissociation between spatial memory in the dorsolateral prefrontal cortex and object memory in the ventrolateral prefrontal cortex (see Duncan & Miller, 2002).
2. Prefrontal activity decreases as familiarity with stimuli or a task increases (Diamond, 2002; Mesulam, 2002). In neuroimaging studies, task-related prefrontal activation decreases as the task becomes more familiar (Race, Shanker, & Wagner, 2009; Raichle et al., 1994), and familiar stimuli or task conditions produce weaker activity in prefrontal neurons than novel stimuli or newly learned task conditions during single-unit neuronal recording studies (Asaad, Rainer, & Miller, 1998; Rainer & Miller, 2000).
3. One of the most consistent findings in neuroimaging studies is that increasing the demand or difficulty of a task (attentional or computational) increases prefrontal activity (e.g., D'Esposito, Ballard, Aguirre, Zarahn, 1998; Nolde, Johnson, & Raye, 1998). A minority of prefrontal neurons are engaged by simple tasks, such as the delayed matching-to-sample or spatial delayed response, whereas many more neurons are engaged by tasks that are more difficult (Duncan & Miller, 2002). Stuss and colleagues (2002) have shown that increasing the task difficulty increases impairment in patients with various frontal lesions on several different tasks (see also Stuss, 2006; Stuss & Alexander, 2007; Stuss et al., 1999), suggesting that increased demand or difficulty in a task requires recruitment of additional regions in the prefrontal cortex.
4. Similarly, older adults tend to have more prefrontal activation compared to younger adults when their task performance is equivalent, suggesting that recruitment of additional prefrontal areas is compensatory (Cabeza et al., 1997; Grady et al., 1994; Madden et al., 1999; Vallesi, McIntosh, & Stuss, 2011).
5. Following unilateral damage to the prefrontal cortex, reconfigurations of functionality have been observed in contralesional homologous regions as soon as 3 days after damage (Rosen et al., 2000; Thulborn, Carpenter, & Just, 1999). Stuss and colleagues (1987) have suggested that the intact processes in the undamaged prefrontal region may compensate for the functional deficit.

6. Finally, the fundamental challenge for any concept of cognition is how it could map to neuronal functioning. Breaking the theorized central executive into discrete subcomponents has highlighted some processes served by distinct brain regions (Stuss & Alexander, 2007; Stuss, Shallice, Alexander, & Picton, 1995); however, it remains unclear how concepts such as monitoring, task setting, and working memory could be explained on the neuronal level. Unfortunately, attempts to regionally fractionate the prefrontal cortex have led to a confusing bevy of unstructured concepts mapped to uncertain neural regions.

Alternative theories suggesting that prefrontal cortex neurons adaptively code information, depending on task demands, have also been presented (Duncan, 2001; Duncan & Miller, 2002). While these models effectively explain functional recruitment in the prefrontal cortex, they do not adequately account for human lesion findings. It is difficult to see how damage to an area that is theorized to be the neuroanatomical substrate for Spearman's *g* (Spearman, 1927) and is vital for all currently relevant rule behavior (Duncan & Miller, 2002) could allow any normal functioning. Yet, patients with damage to large portions of their prefrontal cortex often appear quite normal, with exemplary performance on most neuropsychological tests (Damasio, 1994; Damasio et al., 2012); this includes clinical tests aimed at measuring fluid intelligence (Tranel, Manzel, & Anderson, 2008), a proximate measure of Spearman's *g* (Carroll, 1993). Moreover, Duncan and Miller's (2002) theory does not provide an adequate explanation for the diverse deficits that are acquired after prefrontal cortex damage. The adaptive coding model is also unclear as to how the prefrontal cortex would actually select or discard information at the neural level (Stuss, 2006).

It is apparent that from a functional perspective, neither hypothesis is free of significant problems. Using the general principle that function follows form, the next section reviews evidence for this debate from an anatomical perspective.

ANATOMICAL CONSIDERATIONS

Support for the homogeneity hypothesis can be found in the anatomy of the prefrontal cortex. As in much of the neocortex, most prefrontal connections are local; the majority of connectivity is between regions within the prefrontal cortex (Duncan & Miller, 2002). The intrinsic anatomical organization of the prefrontal cortex may offer clues in the functional debate.

Some researchers have suggested that the prefrontal architecture should be considered a progression of cortical laminar differentiation (Barbas & Pandya, 1989; Sanides, 1972). The intrinsic organization of the prefrontal cortex can be seen as a series of gradual changes in laminar characteristics, which can be traced from two limbic parts: the hippocampal archicortex (on the mediodorsal line) and the olfactory paleocortex (on the basoventral line; for an extrapolation of this principle on a human template brain, see Figure 24–1). Each line consists of a progression from a less differentiated area (cells are not clearly organized into cortical layers) to a more differentiated area (cells are clearly organized into cortical layers). In a cytoarchitectonic investigation of rhesus monkeys, Barbas and Pandya (1989) observed that the mediodorsal line begins at the periallocortex around the rostral portion of the corpus callosum, gradually transitioning to adjacent proisocortical Brodmann areas 24, 25, and 32 and then to medially situated isocortical areas 9, 10, and 14. The next stage includes lateral areas 10 and 9 and the rostral portion of dorsal

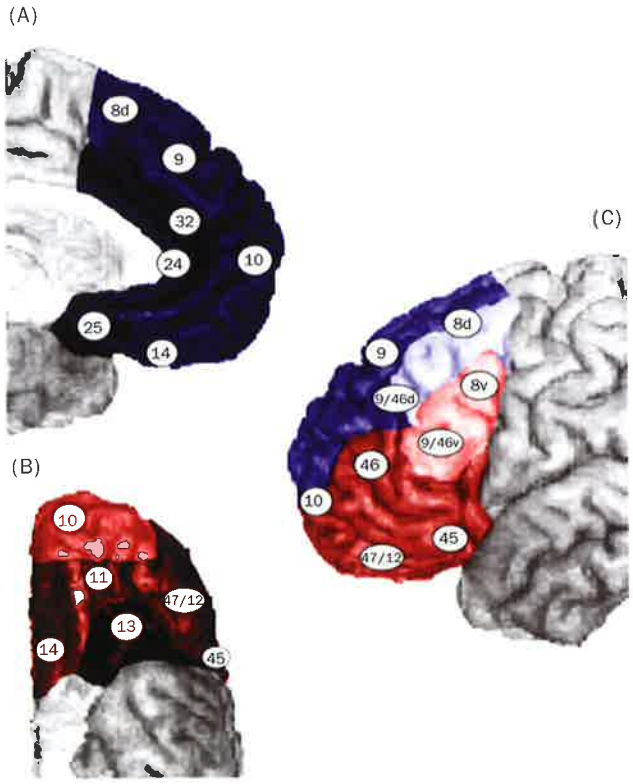

Figure 24–1 Architectonic stages of the human prefrontal cortex. Human architectonic stages were extrapolated from rhesus monkey laminar organization (Barbas & Pandya, 1989) and recent maps that reconcile monkey and human labeling schemes (Petrides & Pandya, 1994). Area 9/46 (dorsal) in the human corresponds to the dorsal portion of area 46 in the monkey; area 9/46 (ventral) in the human corresponds to the ventral portion of area 46 in the monkey; and areas 47/12 and 45 in the human correspond to area 12 in the monkey. The mediodorsal line (represented in blue) and the basoventral line (represented in red) were transposed on a human template brain for the (A) medial, (B) ventral, and (C) lateral perspectives. Within each line, darker colors represent stages with less architectonic differentiation and lighter colors represent stages with more architectonic differentiation. Prefrontal neurons within a line project most strongly to a region that is more differentiated and to a region that is less differentiated.

area 46. The most differentiated regions include the caudal part of dorsal area 46 and dorsal area 8 (Figure 24–1A, C). The basoventral line begins at the periallocortex in the caudal orbitofrontal region to the adjacent proisocortex and then to area 13. Orbital areas 11, 12, and 14 are more differentiated, followed by area 10, lateral area 12, and the rostral part of ventral area 46. Finally, the most differentiated layers reside in the caudal part of ventral area 46 and ventral area 8 (Figure 24–1B, C).

Barbas and Pandya (1989) found that within a cortical line, each area projects most strongly to an area that is more architectonically differentiated and to one that is less differentiated. For example, rostral area 46 projects to the less differentiated area 10 and to the more differentiated area 8. The intrinsic connections are well organized to act in a hierarchical fashion (Badre & D'Esposito, 2009), where demand for a particular process may recruit additional prefrontal cortex regions.

Further support for the homogeneity hypothesis can be seen in the circuitry between the frontal cortex and the basal ganglia. The frontal-subcortical circuitry can be conceived as five subcircuits including the motor circuit, oculomotor circuit, dorsolateral prefrontal circuit, orbitofrontal circuit, and the anterior cingulate circuit (Alexander, DeLong, & Strick, 1986). Each of the subcircuits, however, has the same member structures including the cerebral cortex, striatum, globus pallidus, substantia nigra, and thalamus (Cummings, 1995). Within each subcircuit there is a direct pathway and an indirect pathway (which includes the subthalamic nucleus; Alexander, Crutcher, & DeLong, 1990). Postrolandic regions that are reciprocally innervated with various regions of the frontal lobe project to the striatum in approximately the same area as each particular frontal region (Damasio et al., 2012; Parent & Hazrati, 1995). Although there are wide-ranging functional differences between the frontal-subcortical circuits, common processing may govern each domain, where increasing demand for a particular process may usurp parallel processing in adjacent circuits, as is evident from the general anatomical similarities and the continuing debate concerning functional segregation (DeLong & Wichmann, 2009; Parent & Cicchetti, 1998; Parent & Hazrati, 1995; Percheron & Filion, 1991).

Finally, the critical influence of the monoamine systems, which have a marked influence on much prefrontal functioning (Arnsten & Robbins, 2002), is spread diffusely throughout the prefrontal cortex. Both the dorsolateral and orbitomedial prefrontal cortex receive critical connections from monoamine nuclei in the brainstem (Porrino & Goldman-Rakic, 1982). Connections from the ventral tegmental area, dorsal raphe, and locus coeruleus supply prefrontal cortex with dopamine, serotonin, and norepinephrine, respectively.

However, it is also clear that the prefrontal cortex is not an anatomically monolithic structure. The regional specialization hypothesis is supported by the fact that differing regions in the prefrontal cortex are reciprocally connected with a wide array of distinct brain structures. The dorsolateral prefrontal cortex (dlPFC) is connected via the superior longitudinal fasciculus to the superior parietal region and the adjacent medial parietal cortex. The superior longitudinal fasciculus also connects dlPFC areas with the rostral and caudal inferior parietal lobule. The arcuate fasciculus connects the caudal part of the superior temporal gyrus to dlPFC, while the extreme capsule joins the midportion of the superior temporal gyrus to dlPFC as well. In contrast, several orbitomedial prefrontal cortex (omPFC) areas are connected via the uncinate fasciculus to the rostral temporal lobe. The rostral parahippocampal region (including the amygdala, the subiculum, and the entorhinal and perirhinal cortex) connects to the ventral areas of the omPFC via the uncinate fasciculus, while the caudal region connects to dlPFC via the extreme capsule (see Petrides & Pandya, 2002). Further, the omPFC and, more specifically, the medial network projects to the hypothalamus and periaqueductal gray (An, Bandler, Öngür, & Price, 1998; Öngür, An, & Price, 1998).

The intimate connections of the omPFC with limbic structures suggest a strong association with emotional functioning; whereas the connections with the brainstem and hypothalamus indicate mutual influence regarding visceral function and the autonomic nervous system (Hall, Livingston, & Bloor, 1977; Neafsey, 1990). By contrast, the dlPFC seems particularly well positioned to govern spatial attention and regulate the higher aspects of motor behavior (Petrides & Pandya, 2002).

The anatomical evidence highlights the difficult problem regarding the functionality of the prefrontal cortex. It shows evidence in favor of both the regional specialization hypothesis and the homogeneity hypothesis. In fact, the anatomical and functional evidence make it apparent that neither extreme position is tenable. The vast intrinsic connections suggest that regions are not strictly divided functionally; yet, certainly, distinct regions are initially focused on distinct processes, as the connections to postrolandic regions would indicate. We prefer a middle road, where there is both some regional specialization and some homogeneity, as first suggested by Stuss (2006). We hypothesize that initial processing for differing modalities is done at distinct local regions in the prefrontal cortex, but as demand for a particular task is increased (or there is a requirement for an orthogonal prefrontal process), additional prefrontal regions are recruited. Thus, processing may begin locally in a specific region of the prefrontal cortex, but it can spread to other regions if warranted by the task difficulty. This claim allows us to suggest that whatever computations are performed in the prefrontal cortex must be similar, even across disparate modalities. Therefore, going forward in this chapter, we will assume that the prefrontal cortex is a unitary structure performing a similar function for various modalities.

To address what singular computation the prefrontal cortex could perform that might encapsulate the diverse mental processes attributed to it, we first must examine two central psychological concepts: belief and doubt.

BELIEF, DOUBT, AND THE FALSE TAGGING THEORY

As suggested by Russell (1921), any scientific or philosophical inquiry into the nature of behavior is incomplete without understanding belief—what it is, how it happens, and what it does. Epistemological and psychological perspectives have asserted that most human actions are initially formed by a belief in an idea or a percept (Bogdan, 1986; Gilbert, 1992; but see Price, 1969). Thus, when an individual *accepts* or *believes* an idea or percept, the individual is prepared to act as though the idea or percept were true. Epistemologists have agreed that beliefs involve both the *mental representation* and the *positive assessment* of meaningful information (Gilbert, 1991). The representational component refers to merely the existence of meaningful information within a mental system, while the assessment component refers to the relation of that information to other information that already exists within the mental system. "Meaningful" indicates the pertinent representations for a present context; "information" can refer to either cognitive or perceptual information; and "positive" refers to a directional adjudication of truth, and not the more colloquial emotive associations of something advantageous or good. Therefore, a cognition or perception is believed when the information is represented in a mental system and when that representation disposes an individual to act as if it were true, given appropriate circumstances. In this definition, beliefs can work at a covert level, not relying on overt, conscious processing. This section will begin with a brief discussion of the belief substrate and then will move to the process of belief and doubt. Finally, we will present our model of how belief and doubt map onto neuroanatomical regions.

The rather vague notion of "meaningful information" encompasses a large component of mental representation in the brain (i.e., all cognitions and all perceptions). The elements of mental representation can generally be divided into perceptual elements and cognitive elements. A perceptual element is a mental representation of any perceptual modality, such as visual, auditory, or tactile perceptions. This includes imagery and perceptual memories that substantiate much of episodic memory. A cognitive element is a mental representation of an idea, proposition, knowledge, opinion, attitude, rule, or hypothesis, which substantiates much of semantic memory. At the simplest level, a cognitive element can refer to any type of associative learning (i.e., Pavlovian or instrumental), such as a stimulus-outcome association. Although these elements differ in phenomenology, they are thought to be distributed in similar regions throughout postrolandic association cortices (e.g., the superior temporal region), which have dense reciprocal connections to the prefrontal cortex (Bruce, Desimone, & Gross, 1981; Pandya & Barnes, 1987). One difficult question in epistemology and neuroscience is what the exact units of knowledge representation are and where the boundaries of these elements lie. However, delineation on this level may not be necessary or fruitful. What matters for our discussion is the relation of mental representation elements with one another (Festinger, 1957). Therefore, we will use the term "perceptual-cognitive representation" (PCR) to refer any relevant belief substrate.

Generally, PCRs correspond accurately to the common utility of the external environment, or the more colloquial term, "reality", of the outside world. Perceptions are usually faithful representations of reality, and cognitive elements correspond precisely to perception. Perceptual-cognitive representations create a "belief script" that is largely internally consistent and, at some point, can be grounded back to a perception (Price, 1969). However, PCRs can conflict, often creating doubt for a particular PCR (Festinger, 1957). Individuals can doubt perceptions, most prominently illustrated by perceptual illusions; however, perceptual elements are often highly resistant to doubt, whereas cognitive elements are less resistant to doubt.

Epistemologists have also agreed that there are degrees of belief ranging from (1) a suspicion to (2) an opinion, to (3) a colloquial "belief," to (4) knowledge (Price, 1969). Some have considered knowledge and belief as separate entities due to either a semantic rationale (Price, 1969) or an objective common truth for knowledge that allies to only certain domains (e.g., "knowledge" of perceptual objects such as a doorknob), whereas other domains lack this objective truth and should be considered as mere "belief" (Damasio, 2000; for a review, see Price, 1969). However, as suggested earlier, on rare occasions, individuals do doubt such perceptual representational knowledge (e.g., during an illusion, such as a mirage of a pool of water on a hot day). This implies that individuals' belief scripts "hang in the air like a cloud" (Price, 1969), connected only to fallible perceptions and the intrinsic consistency of the belief script. Philosophical arguments for subjectivism such as the brains-in-a-vat (Forbes, 1995; Wright, 1992) and the computer simulation argument (Bostrom, 2003) are consistent with this view. Therefore, in terms of the mechanics of neural processing for belief and doubt, we will consider knowledge as simply the highest form of belief, which is void of any doubt.

There have been two important philosophical models concerning *how* mental systems believe information (Gilbert, 1991). Descartes (1644/1984) proposed that comprehension and assessment are two distinct and sequential psychological processes. In this model, individuals first comprehend ideas and then are entirely free to accept (believe) or reject (doubt/disbelieve) them (Figure 24–2).

	Cartesian hypothesis	Spinozan hypothesis
Representation Stage	Comprehension	Comprehension & Acceptance
	↓	↓
Assessment Stage	Acceptance OR Rejection	Certification OR Unacceptance

Figure 24–2 Conceptual stages of the Cartesian and Spinozan belief hypotheses. Source: Adapted from Gilbert (1991) with permission.

Intuitively, it appears almost necessary that comprehension occurs before and separately from assessment. As Gilbert (1991) points out, few philosophers or scientists have challenged the Cartesian belief process, which still remains an underlying assumption in many models of mental systems. For example, modern computers process information using algorithms that have a strict division between representation and sequential assessment. Computers can represent information without assessing the information. The ability of information to exist within a computer without an assessment value of veracity associated with it indicates that computers act similarly to the process of belief described by Descartes (Gilbert, 1991).

However, one rare objection came from Spinoza (1677/1982), who rejected Descartes' distinction between comprehension and assessment and theorized instead that comprehending and accepting are the same operation. Spinoza argued that all ideas that are comprehended are represented in the mind as true, and this occurs before any analysis to determine the veracity of the ideas. Assessment is performed in Spinoza's model, but it happens only after comprehension and compulsory acceptance. As Gilbert (1991) has clarified and detailed, in Spinoza's model, if some of the recently comprehended and accepted ideas are compared to other information in the mental system and are deemed inaccurate, they are then unaccepted and become rerepresented as false (Figure 24–2). For example, given the proposition *brains are made of glue*, individuals who comprehend the proposition must initially believe it for an instant, but are then free to unaccept it. A Spinozan belief system, then, indicates that unacceptance (or doubt) is a distinct secondary psychological act in which the initial belief that is inextricably associated with comprehension is subsequently undone or altered. Positive assessment, then, in Spinoza's model simply involves not invoking the secondary psychological act, which allows an unhindered belief. Thus, Spinoza's framework indicates that disbelief, skepticism, and doubt are modifications of an untested initial belief by a secondary psychological process (Gilbert, 1991).

In a series of seminal experiments, Gilbert and colleagues (1990, 1993) examined the two hypotheses using the general principle of how modular systems break down. When a modular information system that vies for limited processing resources and has multiple exit capabilities competes with another process using the same store of resources, it will often bias toward yielding the products of early modules (Gilbert, 1991; Norman & Bobrow, 1975; Tversky & Kahneman, 1974). Resource depletion, then, should allow a system to represent propositions (a product of the first module), but should often prevent a system from assessing those representations (a product of the second module). Thus, for the two belief hypotheses, resource depletion would produce differing beliefs outcomes. Resource depletion should prevent a Cartesian system from either believing or disbelieving the propositions that it only comprehends, and it should prevent a Spinozan system from disbelieving the propositions that it both comprehends and believes. Gilbert and colleagues showed that when participants were given explicitly labeled but directionally valenced false information, resource depletion acted to sway social judgments toward the explicit false information (Gilbert, Krull, & Malone, 1990; Gilbert, Tafarodi, & Malone, 1993). Specifically, participants read crime stories and adjudicated a prison term for each criminal (Gilbert et al., 1993). The crime stories had explicitly labeled "true" information presented in white text and explicitly labeled "false" information presented in red text. Some of the participants were required to perform a distracting task (i.e., to press a button in response to a noise) while reading the crime stories. Participants who engaged in the distracting task (resource depletion) during reading were more likely to accept the red "false" information as true and incorporate it into their prison term judgments. In effect, resource depletion caused participants to believe the false information with which they made consequential social judgments. Resource depletion, therefore, acted to prevent the disbelief of propositions that it both comprehended and believed, supporting the Spinozan model. Subsequent research consistent with the Spinozan model has investigated belief change through fictional narratives (Appel & Richter, 2007; Green & Brock, 2002; Prentice, Gerrig, & Baillis, 1997), effects of warnings about false consumer claims (Skurnik, Yoon, Park, & Schwartz, 2005), and acquiescence on questionnaire responses (Knowles & Condon, 1999). Gilbert (1991, 1993) marshaled further evidence in support of the Spinozan model in diverse areas such as mental development, forced persuasions, attributions, psycholinguistics, mental evolution, and social psychological biases. The hypothesis has not been without its critics (Hasson, Simmons, & Todorov, 2005; Richter, Schroeder, & Wöhrmann, 2009). However, given the abundance and variety of evidence friendly to the Spinozan model, it presents as a plausible and testable theoretical tool.

As indicated by Gilbert (1991), both current and classical theories of mental representation suggest that "once the truth value of a proposition is assessed, the mental representation of the proposition must somehow be altered or 'tagged' to indicate that truth value—otherwise we would

have to reassess the validity of our knowledge each time we deployed it" (Gilbert, 1991, p. 108). Therefore, it is pertinent to address from a neuroanatomical perspective what brain region could mediate this "tagging," which in the Spinozan model is exclusively "false tagging" to representations that are already believed, or regarded as true.

Given the prefrontal cortex's unique anatomical linkages with diverse neural structures, it is ideally situated to mediate false tagging. All sensory modalities find representation in the prefrontal cortex (Öngür & Price, 2000), and the prefrontal cortex has dense connections with areas that are likely to mediate PCRs, such as the superior temporal region. Brainstem monoamine systems, visceral sensory inputs, and the autonomic nervous system connect strongly to the prefrontal cortex. Finally, the prefrontal cortex is closely connected to limbic structures such as the amygdala and the parahippocampal gyrus, suggesting reciprocal influence of emotional and memory systems. These inputs provide critical information about the status of the internal milieu and allow bodily reactions to alter that status. Therefore, the prefrontal cortex is continually informed about the state of the body and perpetually alters that state appropriately. This diverse connectivity implies that the prefrontal cortex may govern the tagging of signals to representations in postrolandic neural regions. Damasio (1994, 1996) has theorized that the prefrontal cortex is critical for the connection of bioregulatory or somatic tags (which include but are not limited to emotion and feeling) to the neural representations of salient experiences. In a similar vein, the FTT asserts that the prefrontal cortex is necessary for the false tag in the assessment component of belief. Belief is simply the existence of PCRs in postrolandic regions, whereas doubt, skepticism, and disbelief are mediated by false tags via the prefrontal cortex.

PRELIMINARY EVIDENCE

The FTT asserts that the prefrontal cortex is critical for false tags, which are somatic in origin, during the assessment of beliefs. The juxtaposition of a false tag on a PCR creates a dispositional doubt for the particular belief receiving the tag. Doubt for a specific belief can have a variety of effects, which are often realized as a reduction of behavior toward the belief. Therefore, the prefrontal cortex should be critical in situations where doubt, uncertainty, and ambiguity are high. In addition, individuals with altered prefrontal cortex structural integrity should have a "doubt deficit," a vulnerability to believe inaccurate information. This section will examine the direct evidence for these conclusions, using neuroimaging studies as well as research in developmental and brain-damaged populations. The prefrontal cortex in children is preferentially underdeveloped in comparison to other brain regions (Dempster, 1992; Giedd et al., 1999; Kostovic, Skavic, & Strinovic, 1988; Sowell et al., 1999), and there is a consistent improvement in the functioning of the prefrontal cortex from infancy to adulthood (Diamond, 2002). The FTT predicts that children, compared to adults, should be more gullible and susceptible to inaccurate beliefs. At the other end of the developmental spectrum, substantial evidence has shown that the structural integrity of the prefrontal cortex in older adults is preferentially diminished relative to other brain regions (Dempster, 1992; Pfefferbaum, Adalsteinsson, & Sullivan, 2005; Raz et al., 1997). The normal aging process results in a decline in frontal lobe functioning (Phillips, MacPherson, & Dalla Sala, 2002), especially for individuals beyond the sixth decade of life (Dempster, 1992; see West, 1996). Here the FTT predicts that older adults should be more vulnerable to inaccurate information, tending to believe without an appropriate level of doubt for a given item of information. Finally, we will examine the tendency toward belief and doubt in individuals with circumscribed brain damage to the prefrontal cortex. As with the two developmental populations, the FTT predicts that patients with prefrontal cortex damage (i.e., prefrontal patients) should have a doubt deficit, a tendency to believe information without normative skepticism.

Neuroimaging in healthy adults has shown prefrontal activations in situations where guessing behavior and doubt are accentuated (Elliott, Rees, & Dolan, 1999) and where ambiguity is increased (Simmons, Stein, Matthews, Feinstein, & Paulus, 2006). Moreover, the prefrontal cortex is also activated when doubt must be employed, such as when learned associations are contradicted (Fletcher et al., 2001; Goel & Dolan, 2001; Nobre, Coull, Frith, & Mesulam, 1999) or real-world beliefs are violated (Parris, Kuhn, Mizon, Benattayallah, & Hodgson, 2009).

One of the most evident characteristics in children is their credulity and tendency toward belief (a trait that likely has developmental and evolutionary advantages). Many philosophers and scientists have suggested that doubt or disbelief is acquired more slowly and with greater difficulty than belief (Flavell, 1985; Lundholm, 1936; Piaget, 1962). Gilbert (1991) has noted that Bain (1859) contrasted individuals' "primitive credulity" with their "acquired skepticism," suggesting that "we begin by believing everything; whatever is, is true" (p. 511). Indeed, research suggests that the last linguistic ability acquired in childhood is the "truth-functional negation," which is the ability to deny propositions (Bloom, 1970; Gilbert, 1991; Pea, 1980). Moreover, young children are notably gullible and suggestible, having a tendency to accept propositions uncritically (Bruck & Ceci, 1995, 1999; Ceci, Ross, & Toglia, 1987). However, as children age, axiomatic beliefs easily acquired in childhood, such as the popular imaginary figures Santa Claus, the Tooth Fairy, and the Easter Bunny, tend to decrease (Prentice, Manosevitz, & Hubbs, 1978; Prentice, Schmechel, & Manosevitz, 1979).

While elderly persons do not tend to believe in imaginary figures, there is evidence that they are more credulous than younger adults. Older adults are more susceptible to telemarketing scams, which after prolonged defrauding finally culminated in the Senior Citizens Against Marketing Scams Act (SCAMS Act) increasing the penalties for telemarketing crimes (Chen, 2007). Research has also indicated that older adults are more vulnerable to deceptive advertising (Denburg et al., 2007; Gaeth & Heath, 1987). Using Gilbert and colleagues' (1993) false information paradigm described above, Chen and Blanchard-Fields (2000) showed that older adults' social judgments were swayed by explicitly labeled false information *without* resource depletion. Thus, older adults believed and used the explicit false information during crime sentencing as much as young adults engaged in the distraction task. Chen (2002) replicated this result with a different paradigm using dispositional attributions instead of crime sentencing. Moreover, increased suggestibility in older adults (Cohen & Faulkner, 1989) has been correlated with impairment in source memory (Schacter, Kaszniak, Kihlstrom, & Valdiserri, 1991), which will be discussed in greater detail below.

There is also mounting evidence that patients with adult-onset focal damage to the prefrontal cortex are more suggestible than patients with focal damage to other brain regions. Recent research from our laboratory indicates that prefrontal patients are more vulnerable to deceptive advertising than healthy adults and brain-damaged comparison populations (Asp, Manzel, et al., 2012). Case studies of patients with prefrontal cortex damage have also described a persistent gullibility toward snake-oil salesmen and disreputable characters (Damasio, 1994). However, while these patients evince an overconfidence in speech and behavior (Damasio et al., 2012), suggesting an abnormal lack of doubt, a search for radical beliefs in these patients and in developmental populations may be futile. The vast majority of actionable beliefs are consistently regulated and sustained by individuals' reliable perception of the environment in which they interact. Even social communication, while more fallible than perception, tends to occur between individuals who transmit accurate information (Clark, 1984; Grice, 1975). Thus, even belief scripts in prefrontal patients with theorized doubt deficits usually correspond precisely to "reality" (one exception is their tendency toward confabulation, which is discussed in detail below) and are not often distorted by the physical or social environment in a systematic way. However, there is a set of beliefs that is discordant with physical "reality" yet is readily professed to all individuals: religious beliefs. Therefore, for scientific investigations religious beliefs may be particularly valuable in that they are common in the marketplace of ideas, they are held to varying degrees, and they are not directly falsifiable. On average, an individual who has a doubt deficit should have more religious belief and should be higher in religious fundamentalism.

Asp, Ramchandran, and Tranel (2012) found that prefrontal patients had higher fundamentalist beliefs and higher specific religious beliefs following brain injury than brain-damaged comparisons and medical comparison group (the latter group comprises patients who had experienced a life-threatening medical event that was nonneurological in nature; Figure 24–3). Across all the beliefs that were assessed, the prefrontal patients had the highest average belief endorsement relative to comparison

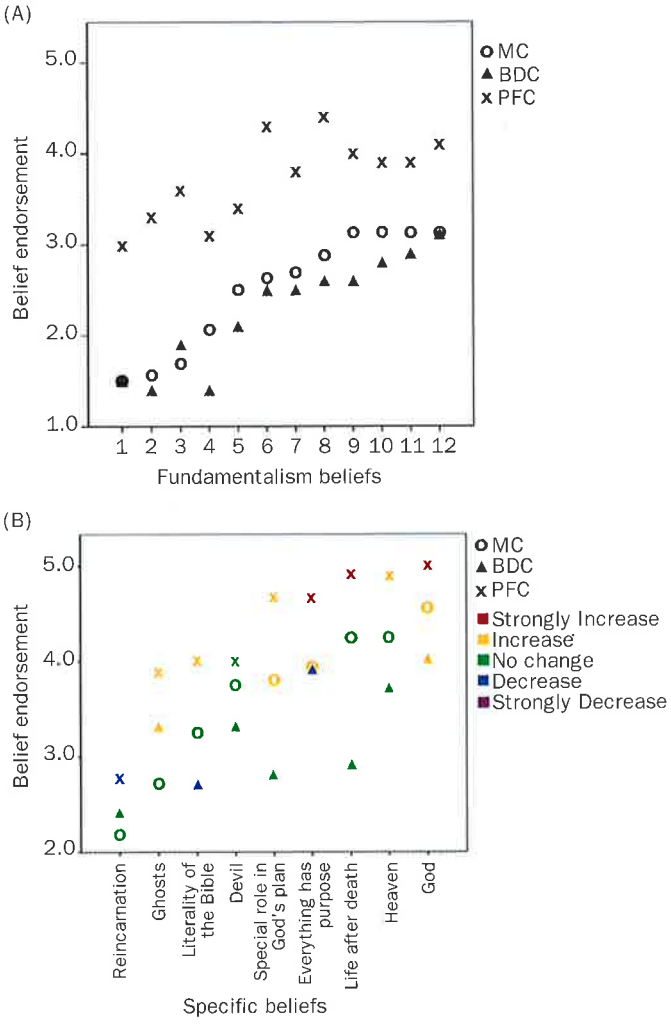

Figure 24-3 Group belief endorsement for individual religious beliefs. The *y*-axis represents belief endorsement, where 1 = low religious belief to 5 = high religious belief. The x-axis represents individual religious beliefs, arranged by increasing endorsement from medical comparisons. BDC, brain-damaged comparisons; MC, medical comparisons; PFC, prefrontal patients. (A) The group mean belief endorsement for each fundamentalism statement. (B) The group mean belief endorsement for specific beliefs including reincarnation, ghosts, literality of the Bible, the devil, a special role in God's plan, everything having a purpose, life after death, heaven, and God. Colors indicate reported changes in beliefs following a subject's medical event. Red and orange represent a mean increase in specific beliefs, green represents no change, and blue represents a mean decrease in beliefs. No group reported a strong decrease in any specific belief after their medical event. Prefrontal patients reported the highest religious fundamentalism beliefs, specific religious beliefs, and increases in beliefs.

patients. This pattern occurred in 6 (of 12) fundamentalist beliefs and in 3 (of 9) specific beliefs where the higher religious belief was to disagree. Therefore, this finding cannot be attributed to the tendency to acquiesce and agree with opinion statements. Prefrontal patients also reported more increases in specific religious beliefs following their brain damage than comparison patients (Figure 24–3). The results suggest that damage to the prefrontal cortex acts to increase religious beliefs. Experiencing a profound, life-threatening medical event per se did not account for the results; and brain damage per se, when located outside the prefrontal cortex, did not lead to the increased religious beliefs observed in prefrontal patients. Therefore, our results indicated that prefrontal cortex damage disrupted the false tagging mechanism. When prefrontal patients were exposed to religious propositions after brain damage, they were impaired at false tagging and doubting the more outlandish dogmas. In effect, this caused them to become more fundamentalist and hold more extreme religious views.

In accordance with this finding, religious belief is high in childhood, whereas religious doubt tends to increase in adolescence with age (Francis, 1986, 1989; Gibson, 1989; Simon & Ward, 1975). This formulation of a neural substrate for credulity in childhood may suggest a proximate cause for why cognitive representations of religious beliefs (also called religious "memes") continue unabated down generations (Dawkins, 2006). From an evolutionary perspective, it is quite advantageous for children to believe whatever parents and tribal elders tell them. However, the disadvantage of primitive credulity is an inability to discern good instruction (e.g., *Do not swim near the crocodiles*) from bad instruction (e.g., *Sacrifice a virgin at the next full moon*; see Dawkins, 2006). Cross-sectional studies have also shown that religious beliefs are greater in older individuals compared to younger ones (Argyle & Beit-Hallahmi, 1975; Hunsberger, 1985; Moberg, 1997; Schultz-Hipp, 2001). Interestingly, this may suggest that the tendency for religious beliefs to increase in old age is driven less by the comforting thought of an afterlife (Dawkins, 2006) but instead is substantiated by a decrease in frontal lobe functioning.

FALSE TAG ORIGINS, BLUNTED EMOTIONS, AND COGNITIVE DISSONANCE

False tags are derived from bioregulatory processes (such as emotion) and are necessary to increase doubt, thereby decreasing belief. The internal body state (of which emotions are a central component) is critical in the doubt process for all PCRs. Therefore, the FTT indicates that individuals with a disrupted false tagging mechanism will have problems using and experiencing these states, tending to show emotional abnormalities.

Patients with damage to the prefrontal cortex have a general emotional impairment, often presenting with blunted emotional responses, as if their affect has been neutralized (Beer, Heerey, Keltner, Scabini, & Knight, 2003; Damasio & Van Hoesen, 1983; Damasio et al., 2012; Stuss & Benson, 1984; Tranel, 2002). Damasio and colleagues (1990) discovered that prefrontal patients had impaired autonomic responses to socially meaningful stimuli, despite having normal autonomic responses to unconditioned stimuli. During the Iowa Gambling Task (where subjects choose from decks of cards, with resulting gains and losses of money; Bechara, Damasio, Damasio, & Anderson, 1994), prefrontal patients generated skin conductance responses (SCRs) when they lost a large sum of money, but the magnitude of the SCRs was never as high as that of normal comparison participants (Bechara, Damasio, Damasio, & Lee, 1999), suggesting that prefrontal patients may have a weakened ability to process the affective attribute of a negative emotional stimulus. Moreover, prefrontal lesion-derived emotional vacancy has been theorized to impair real-life decision making (Bechara, Tranel, Damasio, & Damasio, 1996; Damasio, 1994). The interaction between the monoaminergic systems mediated by the brainstem and the prefrontal cortex is also significantly linked with emotion and mood (Schildkraut, 1965), although it may be a rather indirect connection (Ruhé et al., 2007).

Damasio (1994, 1996) used the compromised ability of prefrontal patients to express emotion and to experience emotion in situations that should elicit an emotional response to argue that the prefrontal cortex is critical for somatic tags to the neural representations of salient experiences. Damasio (1994, 1996) proposed that the prefrontal cortex acts as a "convergence-divergence zone" containing a record of signals from neural areas that were active simultaneously and define relevant aspects of a given situation (see also Nauta, 1971). In his model, somatic states mark or tag salient experiences that are reexperienced when recalled from memory. The replayed somatic state is critical for advantageous decision making because it is this affective valence that biases choices between response options.

The FTT proposes a similar process whereby the prefrontal cortex mediates affective, bioregulatory tags that are superimposed on PCRs. However, the FTT is exclusive to somatic tags arising from body states induced by negative stimuli, such as a state resulting from negative feedback (addressed in further detail below). We define this state as "negative" which can be, on occasion, experienced consciously as an uncomfortable emotional state. "Positive" body states are important in the FTT as well, but only insofar as they influence negative body states.

Thus, in the FTT, when meaningful information, such as a novel proposition, is normally assessed, a negative somatic signal marks any discrepancies in the comparison of existing mental information to the new proposition. If there is a conflict, a negative state is

induced, and for propositions that are falsified, the negative marker is apposed to the propositional information, and a record of this apposition is stored in the prefrontal cortex. This record is dispositional and can recreate and reinstantiate a false tag when any part of the stimulus set is reexperienced or recalled from memory (see Damasio, 1994). The marker may be and often is covert, in that it is not necessarily perceived in the form of "feelings" and it may be largely processed unconsciously. The proposed somatic state is not mutually exclusive with various conscious moods or feelings, although emotional biases are predicted under this hypothesis. Thus, this model asserts that negative body states (such as a negative emotion) in fact determine what is doubted and, therefore, believed.

The general belief process of the FTT proposes what is in essence a largely unconscious single-unit cognitive dissonance theory. Arguably social psychology's most important and provocative theory, the theory of cognitive dissonance asserts that the presence of a cognitive inconsistency will evoke a negative emotional state that will motivate cognitive work aimed at reducing the cognitive inconsistency (Festinger, 1957). In the FTT model, upon comprehension (and compulsory acceptance) of a novel proposition aberrant to the extant belief system, a cognitive inconsistency is produced, which evokes a negative somatic state. "Cognitive work" is done by either tagging the negative state to the novel proposition, thereby falsifying it; tagging the negative state to existing mental information, which acts to falsify extant beliefs; or creating new beliefs (PCRs) aimed at reconciling the discordant proposition and the extant mental beliefs, thereby reducing the negative somatic state. While no extant study has directly examined cognitive dissonance in prefrontal patients, the FTT predicts that these patients have more compartmentalized minds; they fail to evoke a negative state and thus tend not to act in a manner that would reduce it. However, as will be discussed in the section on "Confabulation" below, other experimental paradigms and observations have revealed that prefrontal patients often believe inconsistent ideas without a normative internal assessment.

THE FALSE TAGGING THEORY AND NEUROPSYCHOLOGY

In the remainder of the chapter, we examine how the FTT comes to bear on some of the classical symptoms of frontal lobe damage and the relevance it has for certain psychiatric disorders. We begin with a look at perseveration and related phenomena after the onset of prefrontal lesions, with specific attention given to the role of response inhibition in prefrontal cortex functioning and the problems associated with this explanation.

PERSEVERATION

A classic and contemporary observation in primate ablation research is that frontal lobe lesions cause deficits in reversal learning or the suppression of inappropriate responses acquired over the initial course of learning (Clarke, Robbins, & Roberts, 2008; Izquierdo, Suda, & Murray, 2004; Jones & Mishkin, 1972; Mishkin, Prockop, & Rosvold, 1962; Rudebeck & Murray, 2008; Settlage, Zable, & Harlow, 1948). Recent studies utilizing probabilistic reversal learning tasks in patients with prefrontal cortex damage have confirmed this finding in humans (Fellows & Farah, 2003; Hornak et al., 2004; Rolls, Hornak, Wade, & McGrath, 1994). Reversal learning occurs when a subject must alter established response tendencies when outcome contingencies change, typically in a discrimination task. Thus, a subject is taught that responding to one stimulus produces a reward, whereas executing the same response to another stimulus produces a nonreward. After the subject learns to respond correctly, the experimenter switches the stimulus-outcome associations and the subject must learn to change its behavior. Neural activity related to the stimulus-outcome learning across reversals is evident in the prefrontal cortex of rats, monkeys, and humans (Hampton, Bossaerts, & O'Doherty, 2006; O'Doherty, Critchley, Deichmann, & Dolan, 2003; Schoenbaum, Chiba, & Gallagher, 1999; Thorpe, Rolls, & Maddison, 1983; Wallis & Miller, 2003). The deficits in reversal learning caused by prefrontal cortex lesions are characterized by perseveration, in which a subject inappropriately persists in the maintenance of a category or framework of activity. Perseveration has long been a hallmark of humans with frontal lobe lesions (Luria, 1965; Milner, 1963, 1964). This is exemplified by the Wisconsin Card Sorting Test (WCST; Berg, 1948; Grant & Berg, 1948), a clinical test that measures perseverations and was developed to provide an indicant of cognitive flexibility or the ability to shift cognitive set. It requires subjects to learn stimulus-outcome associations and then, when given negative feedback to their learned associations, measures their ability to unlearn and shift stimulus associations. Neuroimaging studies in healthy adults have shown involvement of the prefrontal cortex in the performance of the WCST (Berman et al., 1995; Lie, Specht, Marshall, & Fink, 2006; Monchi, Petrides, Petre, Worsley, & Dagher, 2001). Patients with frontal lobe lesions fail to achieved the normative amount of categories (new stimulus-outcome associations) and have more perseverative errors on the WCST (Barceló & Knight, 2002; Milner, 1963; Robinson, Heaton, Lehman, & Stilson, 1980); however, several studies have shown substantial variability in WCST performance across frontal patients (Anderson, Damasio, Jones, & Tranel, 1991; Drewe, 1974; Nyhus & Barceló, 2009). Nonetheless, the measure that is most sensitive to prefrontal damage on the WCST is the number of perseverative errors committed (Anderson et al., 1991).

Perseverative tendencies have often been attributed to the prefrontal cortex's role in inhibiting "prepotent" responding (e.g., Miller & Cohen, 2001; but see Schoenbaum, Roesch, Stalnaker, & Takahashi, 2009). However, there are several problems with this hypothesis. Typically, animals with prefrontal lesions are able to inhibit the same response before learning that they are unable to inhibit after reversal (Murray, O'Doherty, & Schoenbaum, 2007; Schoenbaum et al., 2009). Moreover, prefrontal lesions do not affect reversal of some naturalistic or innate response tendencies. Chudasama and colleagues (2007) allowed monkeys to choose between differently sized peanut rewards; however, the monkeys had to select the smaller reward in order to receive the larger one. Monkeys with prefrontal cortex damage inhibited their innate tendency to select the larger reward as well as control monkeys. Firm conclusions are often difficult to deduce from negative results, but these same monkeys with prefrontal cortex lesions showed perseverations on other tasks including a reversal learning task (Izquierdo & Murray, 2005; Izquierdo et al., 2004). This suggests that the prefrontal cortex is not critical for reversal learning and flexible responding because of the specific process of response inhibition (Murray et al., 2007). Instead, there must be a more general function served by the prefrontal cortex, which is critical for guiding behavior after contingency change and masquerades as simple response inhibition in some contexts.

One study in particular provides a vital clue to this underlying function. Cicerone and colleagues (1983) found that patients with frontal lobe tumors were impaired relative to patients with posterior tumors on a task that required subjects to generate hypotheses (or a mental representation of a stimulus-outcome association, i.e., a belief) regarding the relevance of certain stimulus dimensions and then modify the hypothesis on the basis of repeated feedback. Patients with frontal lobe tumors were particularly defective in the ability to eliminate an irrelevant hypothesis despite being informed that it was incorrect; however, they were able to maintain a positively reinforced hypothesis throughout the task. The FTT may supply an explanation for this deficit. If prefrontal cortex lesions impair the patients' ability to tag their beliefs as false, they would be unable to doubt incorrect beliefs, leaving the newly formed belief (and the ability to form alternative new beliefs) intact. In effect, this would cause a perseveration on inappropriate beliefs even in the face of contradictory evidence. Therefore, in contrast to the response inhibition hypothesis, where the prefrontal cortex is critical for simply stopping a prepotent behavior, the FTT requires a belief to be formed to some threshold before the prefrontal cortex becomes critical in doubting the specific belief, which only then is withheld.

Developmental populations also tend to have increased perseverations as children often make perseverative errors (Diamond, 2002; Luria, 1959; O'Sullivan, Mitchell, & Daeher, 2001). Children perseverate in tests of rule use, word learning, naming, and nonverbal tasks (Gerstadt, Hong, & Diamond, 1994; Johnson, 1994; Sophian & Wellman, 1983; Wertlieb & Rose, 1979). For example, 2-year-old children perseverate when sorting a series of items, even if they know enough to sort them correctly (Zelazo & Reznick, 1991). In a classical observation by Piaget (1954), infants tended to perseverate by searching for a toy in the last place they found it, not where it was last hidden. This is evident in Piaget's (1954) A-not-B search task, where infants find a hidden object at location A; following several successful searches at A, infants continue to search there even when they clearly see the relevant object being hidden at location B. The infant's failure in the A-not-B task has been confirmed by an analysis of infant eye movements (Diamond, 1991). These results suggest that infants can successfully create a PCR for a stimulus-outcome association (i.e., a belief) but are poor at falsifying that association relative to older subjects. Moreover, in a reversal learning task, younger children committed more perseverative errors than older children (Gollin, 1964). Indeed, several studies examining age-related norms of the WCST found that perseverative errors in children between the ages of 5 and 12 were significantly reduced with each subsequent year (Chelune & Baer, 1986; Rosselli & Ardila, 1993; Shu, Tien, Lung, & Chang, 2000).

Many studies of aging have indicated that older adults make significantly more perseverative errors on the WCST compared to younger adults (e.g., Daigneault, Braun, & Whitaker, 1992; Fristoe, Salthouse, & Woodard, 1997). In a detailed analysis of the WCST error types in older adults, Fristoe and colleagues (1997) found that older adults had a selective deficit in the use of feedback, and in particular negative feedback. After negative feedback, the probability of staying with a prior hypothesis (percentage of lose-stay) was significantly higher for older adults than for younger adults. This corroborated earlier evidence from Offenbach (1974) that older adults were poor at using feedback for the current hypothesis to guide subsequent behavior (negative feedback deficits are discussed in more detail below). Nagahama and colleagues (1997, 2005) found that cerebral blood flow was reduced in the prefrontal cortex in proportion to the increase in the number of perseverative errors with aging, supporting the notion that age-related increases in perseverative errors are linked to the prefrontal cortex.

One problem with reversal learning paradigms is that they confound the ability to use feedback from a previous stimulus-outcome association with the ability to learn a new stimulus-outcome association. It is difficult to delineate whether increased perseverations following prefrontal cortex damage are due to a deficit in using feedback to inhibit a stimulus-outcome association or if they are due to an inability to attend and acquire new stimulus-outcome

associations. However, experimental extinction paradigms do not have the additional requirement of acquiring a new stimulus-outcome association and have provided evidence that perseverations are due to the former function and not the latter. A seminal observation made by Pavlov (1927) in his conditioning paradigms was that when a stimulus is no longer paired with a reinforcer and is extinguished, the original learning is not destroyed. Pavlov discovered spontaneous recovery, where the simple passage of time partially dissipated the extinction learning and the original behavior appeared to be restored, suggesting that breaking the contingency does not simply remove the original learning. Recent research has bolstered this finding with multiple paradigms (Bouton, 2002; Rescorla, 2001) and indicates that the stimulus-outcome association in both Pavlovian and instrumental conditioning remains intact during extinction. Therefore, extinction reflects new learning that is quite specific to the particular stimulus-outcome association that has been extinguished (Rescorla, 2001).

In our conceptualization of the FTT, extinction learning is the application of false tags to the original stimulus-outcome association. Extinction learning is the gradual building of doubt for a specific association, which is accomplished via false tags mediated by the prefrontal cortex. Thus, following extinction, subjects doubt the specific old associations (or beliefs) rather than simply unlearning the associations, and this doubt proceeds to cause reduced responding. Consistent with our depiction, Rolls and colleagues (1994) found that prefrontal lesions hindered extinction learning, as patients produced more responses (or perseverations) to a stimulus without reinforcement than healthy comparison participants. Moreover, primate ablation research has observed that monkeys with prefrontal lesions are slower to extinguish responding (Butter, 1969; Butter, McDonald, & Snyder, 1969; Butter, Mishkin, & Rosvold, 1963). Of interest to our theorizing, Izquierdo and Murray (2005) replicated Butter and colleagues' results with the same prefrontal lesioned monkeys from Chudasama and colleagues' (2007) study of the peanut reversed reward contingency task described above. In fact, these monkeys were impaired at extinction learning directly following their successful completion of the peanut reversed reward contingency task (Chudasama, Kralik, & Murray, 2007), providing a difficult dilemma for the response inhibition hypothesis.

In the Pavlovian division of conditioning, many studies have indicated a strong link between the prefrontal cortex and fear extinction (e.g., Morgan, Romanski, & LeDoux, 1993; Quirk & Mueller, 2008; Sotres-Bayon, Cain, & LeDoux, 2006). Rats with prefrontal lesions tended to have increased spontaneous recovery (Quirk, Russo, Barron, & Lebron, 2000), and nonlesioned rats that showed less fear behavior (i.e., freezing) after extinction training had more prefrontal neuron responses (Milad & Quirk, 2002). In humans, prefrontal cortex activity during neuroimaging was preferentially enhanced during extinction (Gottfried & Dolan, 2004), and thicker prefrontal cortex has been correlated with greater fear extinction memory (i.e., lower SCR to the conditioned stimulus; Milad et al., 2005). However, some fear extinction results that were obtained using the lesion method may be confounded by the fact that prefrontal patients tend to have blunted emotions (Damasio et al., 2012) and show decreased autonomic responses to certain stimuli (Damasio, Tranel, & Damasio, 1990).

As will be discussed in greater depth below, prefrontal patients have deficits remembering the source of memories. These patients often have both temporal and contextual amnesia (e.g., Ciaramelli & Spaniol, 2009; Janowsky, Shimamura, & Squire, 1989); they can remember a specific memory item but are poor at correctly identifying when and in what context the memory occurred. Interestingly, this parallels two of the important phenomena where extinguished behavior can be restored: spontaneous recovery and contextual renewal (Bouton, 2002, 2004). Spontaneous recovery can be thought of as a type of temporal amnesia and contextual renewal (the original stimulus-outcome association is restored when the stimulus is tested in another context) as a type of contextual amnesia.

Some investigators have suggested that learning to inhibit a response may underlie experimental extinction results (e.g., Rescorla, 2001); however, others have suggested that the evidence for this is tenuous, as high responding in extinction does not guarantee better extinction learning (Bouton, 2004). Several alternative models indicate that each conditioned stimulus presentation arouses an expectation of the unconditioned stimulus, which is disconfirmed on each extinction trial. In the Pearce-Hall model (Pearce & Hall, 1980), the discrepancy is considered an event that reinforces new inhibitory learning that is subsequently superimposed on the original excitatory learning (see also Daly & Daly, 1982).

Results from the overexpectation paradigm are especially consistent with this expectation-violation account in both Pavlovian and instrumental conditioning (Lattal & Nakajima, 1998). In this task, animals are initially trained that several stimuli independently predict a reward. Then two stimuli are presented together, in compound, followed by a single reward. When animals are assessed later on each individual stimulus, they exhibit reduced responding to the compounded stimuli. This suggests that reduced responding results from the violation of summed expectations for the reward during compound training; i.e., the animal expects two rewards for the two stimuli but is given only one. The resulting discrepancy between actual and expected outcomes—a negative prediction error—weakens the associative representations, facilitating some extinction. Temporary inactivations of the prefrontal cortex in rats during compound training prevent later reduction in responding to stimuli (Takahashi et al., 2009). This finding presents another difficulty for the response

inhibition hypothesis because it predicts that prefrontal cortex reversible inactivation during the compound training phase would have no effect on subsequent performance in the probe test. The prefrontal cortex must be critical for *learning* about the discrepancy between the expectation of two rewards and the reality of one reward during the compound trials of the overexpectation task. Therefore, while the prefrontal cortex does play an important role in response inhibition, it appears that simply stopping a response is not its root function (Schoenbaum et al., 2009). In fact, the evidence suggests that the prefrontal cortex is necessary for extinction learning (and in our model false tags) arising from differences between actual and expected outcomes.

PREDICTION ERRORS AND NEGATIVE FEEDBACK DEFICITS

There is a growing consensus that the monoamine systems, and in particular the dopamine system, signal prediction errors, which calculate the difference between actual and expected outcomes (e.g., Hollerman & Schultz, 1998; Roesch, Calu, & Schoenbaum, 2007; Waelti, Dickinson, & Schultz, 2001). Dopamine plays a key role in switching during reversal learning (e.g., Smith, Neill, & Costall, 1999). Ridley and colleagues (1981) found that amphetamine injection (increasing dopamine levels) impaired reversal learning producing increased perseverative errors, while haloperidol (a dopamine antagonist) prevented perseverative responding during pretreatment in monkeys. Dopaminergic prediction errors drive both the acquisition of new learning and extinction learning by altering stimulus-outcome representations when contingencies change. Prediction errors are signaled by phasic activity in dopamine neurons in the ventral tegmental area (VTA) of the midbrain (as does the substantia nigra), which, as mentioned above, is diffusely connected to the prefrontal cortex. Dopamine neurons spike their activity when errors are frequent and outcomes unpredictable, but activation is progressively reduced as performance is consolidated and outcomes become more predictable (Hollerman & Schultz, 1998). Phasic suppression of firing in dopamine neurons occurs when an expected outcome is omitted (Hollerman & Schultz, 1998; Takahashi et al., 2009). Positive prediction errors occur when an unexpected outcome is instituted, producing spiking activity in dopamine neurons, and negative prediction errors occur when an expected outcome is omitted, producing a reduction of activity in dopamine neurons. Thus, the VTA should be critical for extinction learning. Takahashi and colleagues (2009) found that bilateral inactivation of the VTA (where phasic activity in dopamine neurons was not suppressed due to infusion of gamma-aminobutyric acid [GABA] agonists) during the compound training of the overexpectation task did not suppress responses in the probe test. In addition, unilateral VTA inactivation and contralateral prefrontal cortex inactivation during compound training also produced this pattern of results, suggesting that the connection between the VTA and the prefrontal cortex is critical to extinction learning.

The prefrontal cortex itself does not signal prediction errors, as it generally does not show stronger or weaker activity when outcomes are delivered or omitted unexpectedly (Schoenbaum et al., 2009; Takahashi et al., 2009). In our conceptualization, prefrontal false tags are the fundamental substance of extinction learning. They are superimposed on specific stimulus-outcome associations and act to reduce downstream responding. The fact that prefrontal neurons are relatively unresponsive to single unexpected events allows for a gradually increasing doubt for a particular stimulus-outcome association (i.e., a belief). This may be particularly adaptive for an animal in that it can doubt a specific belief yet behave in a way that is congruent with it. Thus, the behavior of an animal with the false tagging mechanism would be less reliant on the most recent outcome, and the animal could adapt its behavior to the *probability* of some event occurring.

Schoenbaum and colleagues (2009, 2010) have put forth a hypothesis suggesting that the orbitofrontal cortex is important for signaling outcome expectancies. In their view, the orbitofrontal cortex is crucial for using information about expected outcomes to update associative representations and guide behavior. Overall, the FTT is quite consistent with this perspective except for two distinctions. First, false tagging is a general mechanism for the entire prefrontal cortex rather than being exclusive to the orbitofrontal region; and second, the prefrontal cortex mediates extinction learning (i.e., false tagging) in response to only negative prediction errors rather than in response to both positive and negative prediction error learning. This perspective was alluded to by Schoenbaum's group (Takahashi et al., 2009), who originally suggested that the orbitofrontal cortex may not play a critical role in learning from positive prediction errors. Yet, in later theorizing (Schoenbaum et al., 2009) they did not make this distinction, suggesting that the orbitofrontal cortex is critical for learning from both positive and negative prediction errors but not for new learning. However, the division between learning using positive prediction errors and new learning without prediction errors remains tenuous and ill-defined. The FTT suggests that the prefrontal cortex is not critical for new learning or for positive error learning, as new learning is largely unaffected by prefrontal cortex damage (e.g., Gallagher, McMahan, & Schoenbaum, 1999).

Serotonergic systems mediated by the dorsal raphe also play an important role in prediction errors. Anatomical and pharmacological evidence has suggested that the serotonergic and dopaminergic systems may act as mutual opponents (Daw, Kakade, & Dayan, 2002). Sourbrié (1986), in an extensive review, indicated that the main function of

the central serotonergic neurons is behavior inhibition, where decreased serotonin transmission is associated with the increased performance of behaviors that are usually suppressed. Several studies have also shown that prefrontal serotonin depletion produces perseverative responding in reversal learning tasks (Clarke, Dalley, Crofts, Robbins, & Roberts, 2004; Clarke et al., 2005). More research is needed to delineate the role of the monoamine systems in false tagging and perseverative responding, as dopaminergic and serotonergic systems have a diverse and convoluted relation with one another, which differs in various neural structures (Daw et al., 2002; Marek, 2007; Meltzer, Li, Kaneda, & Ichikawa, 2003).

The aggregate evidence for the FTT suggests that damage to the prefrontal cortex should result in a selective deficit in the ability to use negative prediction errors or "negative feedback" to drive future responding. Wheeler and Fellows (2008) investigated the influence of positive and negative feedback on subsequent behavior with a probabilistic reinforcement learning task in patients with prefrontal cortex damage. These patients could learn the probabilistic discrimination normatively, and during a testing phase could also choose the previously (in the training phase) *rewarded* stimulus as often as the comparison group; however, the patients were much less likely to avoid the previously *punished* stimulus when given the stimulus in the testing phase. This indicates that prefrontal patients are selectively impaired at learning from negative feedback.

Social psychological studies first suggested that negative and positive outcomes influence behavior via distinct routes. Loss aversion, the intuition that a loss of $X is more aversive than a gain of $X is attractive, is thought to underlie various decision-making phenomena, including framing and endowment effects (Kahneman & Tversky, 1984). In a neuroeconomic paradigm, Tom and colleagues (2007) revealed that loss aversion correlated with activity in the prefrontal cortex, while potential gains were correlated with a broad network of activation in differing regions. Neuroeconomic research has also shown that normative loss aversion may be diminished in patients with prefrontal cortex damage. Shiv and colleagues (2005) examined the behavior of prefrontal patients and comparison participants in a gambling task where subjects were given the chance to bet on a series of coin flips that would each result in winning $2.50 or losing $1. Because each gamble has a positive expected value (in the long run, more gambles will gain more money), subjects who are sensitive to economic loss are at a disadvantage. Prefrontal patients gambled more often and earned more money than comparison participants, implying that the patients were less sensitive to or failed to learn from negative outcomes.

Recent evidence from reinforcer devaluation paradigms in rats and monkeys has also suggested that damage to the prefrontal cortex creates a selective learning deficit from negative feedback via *internal* cues as opposed to the external cues in standard extinction and reversal learning paradigms. "Reinforcer devaluation" refers to a procedure in which changes in a learned response are assessed after devaluation of the expected outcome. Typically, this is done with food by selectively satiating the animal on the outcome or by pairing it with illness (Holland & Straub, 1979). In contrast to reversal learning, in which the animal directly experiences pairing of the new outcome with the stimuli and responses, the effects of reinforcer devaluation do not involve a change in the actual outcome. Under these conditions, the animal must use the changing outcome value to update stimulus-outcome associations in order to appropriately guide behavior. This is easily accomplished by normal animals, which respond significantly less for a devalued outcome than for a nondevalued outcome (Murray et al., 2007). In contrast, rats and monkeys with prefrontal cortex lesions fail to alter learned responding after devaluation (Gallagher et al., 1999; Izquierdo et al., 2004). In a study using a developmental population, Klossek and colleagues (2008) found that young children (less than 2 years old) were also more resistant to outcome devaluation than older children. This suggests that the prefrontal cortex plays a critical role in integrating negative internal information with the original stimulus-outcome association. While this paradigm has not been performed in adult humans with prefrontal cortex damage, it is interesting to speculate on aberrant behaviors in prefrontal patients that may result from an inability to update devalued stimulus-outcome associations. Specifically, Anderson and colleagues (2005) discovered abnormal excessive collecting behavior (or hoarding) in patients with prefrontal cortex damage. If initial collecting allows a stimulus-reward association, which is then "devalued" by negative consequences, such as an inability to perform normal daily activities due to the clutter, then prefrontal lesions may cause hoarding because of a failure to alter learned responding after devaluation. Indeed, children (Burk, 1900; Olmstead, 1991) and older adults (Kim, Steketee, & Frost, 2001; Samuels et al., 2008) tend to have increased collecting behavior. However, this account is different than the traditional reinforcer devaluation paradigms in that it is not internal stimuli that initiate the devaluation.

Therefore, the prefrontal cortex is critical to alter future behavior following negative feedback. In our model, the prefrontal cortex uses tags derived from body states induced by negative stimuli (Damasio, 1994, 1996) to increase doubt for a specific learned association (i.e., belief), which only then often acts to reduce responding. Individuals who have a disrupted false tagging mechanism are able to form new beliefs normatively but are poor at falsifying beliefs when they are inconsistent. Thus, these individuals should have compartmentalized minds, where ideas are easily accepted but rarely compared with and doubted in reference to the extant belief script. Evidence

for this is presented in the following section, as prefrontal patients do tend to believe inconsistent ideas without a normative internal assessment.

CONFABULATION

To this point, the FTT has mostly dealt with memory encoding of meaningful information. However, if memory is a reconstructive process (Bartlett, 1932), and if both accurate and inaccurate memory elements are recalled during a memory search (Nadel & Moscovitch, 1997), the prefrontal cortex may act as a verifier of recovered memories by applying false tags (i.e., covert negative state representations) to inaccurate memory elements, thereby producing a veridical memory. The suggestion that the prefrontal cortex is necessary for editing and inhibiting inaccurate memories is certainly not novel (Burgess & Shallice, 1996; DeLuca & Diamond, 1995; Fisher, Alexander, D'Esposito, & Otto, 1995; Moscovitch & Melo, 1997; Parkin, Bindschaedler, Harsent, & Metzler, 1996; Schacter, Curran, Galluccio, Milberg, & Bates, 1996). In this perspective, damage to the prefrontal cortex would result in impairment to the mechanism that tags inaccurate memories as false and the symptom of confabulation would result. Pathological confabulation has been defined as "honest lying" (Moscovitch, 1989), because confabulating patients provide information that is obviously false without intending to lie. They truly believe their misreports and will produce consequential actions based on those beliefs. Their central deficit is an inability to compare inconsonant beliefs and rectify them. This section examines confabulation and related tendencies in patients with prefrontal cortex damage and developmental populations.

"Spontaneous" confabulation is a common categorization of confabulation that refers to an unprovoked outpouring of unbelievable autobiographical claims. Perhaps the earliest clinical description of spontaneous confabulation was from the seminal observations of the frontal lesion patient Phineas Gage by Harlow (1868/1993), who noted that "[Gage] was accustomed to entertain his little nephew and nieces with the most fabulous recitals of wonderful feats and hair breadth escapes, without any foundation except in his fancy" (p. 277). Substantial research has indicated a strong association between prefrontal cortex damage and confabulation (Dalla Barba, 1993b; Johnson, Hayes, D'Esposito, & Raye, 2000; Moscovitch & Wincour, 2002; Parkin, 1997; Schnider, Bonvallat, Emond, & Leeman, 2005; Stuss, Alexander, Lieberman, & Levine, 1978). Therefore, the FTT predicts that damage to the prefrontal cortex disrupts both memory encoding and retrieval. There is a deficit in the false tagging during initial comprehension (with compulsory acceptance) and assessment (which produces an increased fictile state), as well as a deficit in the false tagging during memory retrieval. This dual deficit explains why some patients will confabulate not only with recent events but also with remote memories acquired before brain damage (Moscovitch, 1989, 1995). It also addresses the increased suggestibility of confabulating patients, which early investigators surmised must play a prominent role in the genesis of confabulation (Ey, 1954; Korner, 1935; Pick, 1915; but see Berlyne, 1972). Confabulating patients are often unaware of their falsehoods, and when confronted with the truth, they tend to cling to their fallacy (Moscovitch, 1989) in a particularly enlightening perseveration. Perseveration is intimately related to confabulation (Baddeley & Wilson, 1986; Shapiro, Alexander, & Gardner, 1981), as confabulators tend to make increased perseverative errors on the WCST (Ciaramelli, Ghetti, Frattarelli, & Làdavas, 2006; Fisher et al., 1995; Moscovitch, 1989; Moscovitch & Melo, 1997). Moreover, it has been shown that longitudinal declines in confabulation correlate with decreases in perseverative errors (Kapur & Coughlan, 1980). Further, recent research has indicated that confabulation is specifically associated with extinction learning impairments (Nahum, Ptak, Leemann, Lalive, & Schnider, 2010; Nahum, Ptak, Leemann, & Schnider, 2009).

In accordance with this view, children can be induced to produce extensive false narratives that bear a striking resemblance to those of confabulating patients (Bruck & Ceci, 1995; Schacter et al., 1995). Children insist upon the accuracy of their stories even in the face of disconfirming evidence, suggesting a perseveratory tendency to the memory (Schacter, Kagan, & Leichtman, 1995).

MEMORY DEFICITS: TIME AND CONTEXT

Deficits in Temporal Memory

Research in spontaneous confabulators has indicated that the prefrontal cortex is necessary for temporal delineation of when memories occurred. On a continuous recognition task, Schnider and Ptak (1999) showed that spontaneous confabulators (with damage to the prefrontal cortex and basal ganglia) could not suppress irrelevant memories to previously encountered information. In this task, subjects saw several runs of a series of pictures, among which several (pictures) were repeatedly presented. Subjects were told to indicate picture recurrences within one run. The subsequent runs were composed of precisely the same picture series, arranged in different orders. Subjects were told to forget that they had already seen all the pictures from the previous runs and to indicate picture recurrences only within the current run. Thus, task performance depended on the ability to sense whether the memory evoked by the presentation of a picture referred to the "ongoing reality" of the current run or the "past reality" of the previous runs. Where healthy comparison participants and nonconfabulating amnesics maintained their performance on the latter runs to the level of the first run, spontaneous confabulator performance was markedly poor on the latter

runs. Moreover, spontaneous confabulators showed a pronounced increase in false-positive responses on latter runs (they tended to think that the pictures presented in the initial runs were repeated within the current run), which in the FTT model evince a deficit to tag evoked memories that do not pertain to ongoing reality as false.

The failure of spontaneous confabulators to differentiate between current and past events hints at another deficit that commonly occurs in patients with prefrontal cortex damage. Early observers of spontaneous confabulators noted that their confabulations tended to be real memories; however, they appeared out of the correct temporal order (Tiling, 1892; Van der Horst, 1932). These investigators attempted to explain confabulation as a complete loss of temporal signposts for personal experiences. In this view, confabulations were real experiences taken out of their proper chronological order (see also Dalla Barba, 1993a; Schnider, 2003; Talland, 1961). Moreover, Schnider and colleagues (2000) discovered that as patients recover and decrease spontaneous confabulations, temporal confusion (as measured by the continuous recognition task described above) also dissipates in parallel. A subsequent study using the continuous recognition task found that patients with prefrontal cortex damage who did not confabulate were also impaired on this task (Gilboa et al., 2006), suggesting that temporal memory deficits are a feature of prefrontal cortex damage, not of confabulation per se. Thus, even in prefrontal patients without notable confabulation, studies have found deficits in the memory for the sequence or temporal order in which items occur, while memory for the item itself remains intact (Butters, Kaszniak, Glisky, Eslinger, & Schacter, 1994; Johnson, O'Connor, & Cantor, 1997; Kesner, Hopkins, & Fineman, 1994; Mangels, 1997; Parkin, Leng, Stanhope, & Smith, 1988; Shimamura, Janowsky, & Squire, 1990; Swain, Polkey, Bullock, & Morris, 1998). The deficit in temporal ordering is not related to the severity of amnesia (Moscovitch & Winocur, 1995) and is consistent with the finding that prefrontal patients are impaired on tests of recency discrimination (Ladavas, Umilta, & Provinciali, 1979; Milner, 1971, 1974; Milner, Corsi, & Leonard, 1991). Impairments in temporal ordering from prefrontal lesions also correlate with increased perseverative errors on the WCST (Mangels, 1997; Shimamura et al., 1990). If a memory search is indeed "stupid," as multiple trace theory predicts (Nadel & Moscovitch, 1997), retrieving both accurate and inaccurate memory elements, then, in the FTT model, prefrontal lesions would not allow false tags to be applied to items in improper temporal contexts, producing temporal memory deficits.

Moreover, children are also poor at temporal ordering and sequencing even when they know enough to sequence items correctly (Zelazo & Reznick, 1991). Preschool children tend to report and behave as though they had known novel facts for a long time, even though they had actually acquired them only minutes earlier (Taylor, Ebensen, & Bennett, 1994). In addition, memory for temporal order is poorer in older compared to younger adults. Older adults have greater difficulty than younger adults in reconstructing the order of a list of words or pictures (Kinsbourne, 1973; Moscovitch & Winocur, 1995; Naveh-Benjamin, 1990) and in judging the relative recency of two items (LeFever & Kumkova, 1996; McCormack, 1982), which correlates with reduced performance on the WCST (Parkin, Walter, & Hunkin, 1995).

Deficits in Contextual Memory

Broadly, prefrontal patients tend to have deficits in "source memory" (Duarte, Ranganath, & Knight, 2005; Janowsky et al., 1989; Johnson et al., 1997; Parkin et al., 1988), a general term for the when, where, or associations with which a particular memory was encountered (Schacter, Harbluk, & McLachlan, 1984). Thus, in addition to having impairments in temporal attribution to memories, prefrontal patients also tend to have deficits in attribution of context to memories (Schacter, 1987; Smith & Milner, 1984). Ciaramelli and Spaniol (2009) found that confabulators with prefrontal cortex damage were especially poor in contextual memory, producing high false alarm rates. Prefrontal patients were prone to identify individual items with almost every context. In the FTT, source memory deficits are attributed to the inability to apply false tags to inaccurate memory elements. When a memory search produces an inaccurate memory element, no false tag can be applied to items in improper temporal or situational contexts. In effect, this creates a bias for the initially recalled memories to be verified as accurate and appropriate, resulting in temporally displaced confabulations and source memory deficits.

Bruck and Ceci (1995) have shown that children are especially vulnerable to source memory deficits, and as children age there is a marked developmental progression in their ability to remember the source of information (Gopnik & Graf, 1988; but see Schacter et al., 1995). In addition, declines in source memory for contextual details in older adults compared to younger adults have been found (Fabiani & Friedman, 1996; Henkel, Johnson, & De Leonardis, 1998; Schacter et al., 1991; Simons, Dodson, Bell, & Schachter, 2004; Spencer & Raz, 1994), which have been correlated with an increase in suggestibility (Cohen & Faulkner, 1989). Source memory deficits in older adults have also been correlated with increased perseverative errors on the WCST (Craik, Morris, Morris, & Loewen, 1990; Glisky, Polster, & Routhieaux, 1995).

Thus, the prefrontal cortex is critical in the appropriate *selection* of memory items after a memory search. Here the crucial clue for the FTT is that improperly selected memory items in prefrontal patients (with and without notable

confabulation) are believed and "feel right" (Gilboa, 2010). Therefore, this provides evidence that the doubt process and the cognition selection process are the same, as it is certainly possible for a dissociation to exist between the two processes. Instead, when evidence is offered against confabulators' inaccurate memories, they rarely doubt their inaccurate memories but will often "double down" and perseverate their confabulations by creating new secondary confabulations in an attempt to reconcile the inconsistencies (Gilboa, 2010).

Prefrontal patients (with and without notable confabulation) also tend to endorse more items not previously seen (foils) on yes-no recognition memory tests than patients with amnesia due to medial temporal lobe damage or normal subjects (e.g., Rapcsak, Reminger, Glisky, Kaszniak, & Comer, 1999; Schacter et al., 1996; Verfaellie, Rapcsak, Keane, & Alexander, 2004). In our conceptualization, when a foil is presented to patients with prefrontal damage, they are unable to use memory to false tag the foil's PCR. Patients with prefrontal damage would then believe that the foil was a target item. The failure of these patients to tag irrelevant stimuli (the foil) as false is an important clue regarding another symptom of prefrontal cortex damage. As will be discussed below, distractibility to irrelevant cognitive and perceptual information is a central problem following damage to the frontal lobe.

PLANNING DEFICITS

Deficits in temporal ordering shed light on a prominent feature of executive functioning: the ability to plan complex sequences of actions (Tranel et al., 1994). Although prefrontal patients are able to perform individual actions that constitute a sequence of events (Swain et al., 1998), the temporal organization for that sequence is impaired, which results in actions being performed in the wrong order or not at all. A number of studies have shown that prefrontal patients are impaired in planning (Duncan, 1986; Grafman, 1989; Stuss & Benson, 1986; Tranel et al., 1994). This has been shown in clinical tasks such as the Tower of London (Shallice, 1982; or Tower of Hanoi; Simon, 1975), which assess the ability to plan the moves necessary to rearrange an array of colored disks on pegs from a starting position to match a predetermined position. To perform the task successfully, subjects need to look a few moves ahead and choose the appropriate sequence in which subgoals are to be reached. Patients with damage to the prefrontal cortex are impaired on the Tower of London/Hanoi (Goel & Grafman, 1995; Owen, Downes, Sahakian, Polkey, & Robbins, 1990; Shallice, 1982), and research has indicated that they have particular difficulty in dealing with the first encounter of goal conflicts in these tasks (Morris, Miotto, Feigenbaum, Bullock, & Polkey, 1997). Deficits in "strategy application" or real-world planning tasks have also been discovered in patients with prefrontal lesions (Shallice & Burgess, 1991; Tranel, Hathaway-Nepple, & Anderson, 2007), which have been correlated with increased perseverative errors on the WCST (Knight, Alderman, & Burgess, 2002). In the FTT model, if a memory search retrieves both accurate and inaccurate potential actions for a given situation, and prefrontal patients are unable to false tag an inappropriate action for the correct sequence, the ability to plan complex sequences of actions will be impaired. The prefrontal cortex is critical for the *selection* of potential action representations to the relevant context. Thus, each potential action that is retrieved in a planning scenario is "believed" and "feels right/appropriate" for the context.

Young children are poor at the Tower of Hanoi task compared to older children (Klahr & Robinson, 1981; Welsh, 1991) and generally have a poor ability to manipulate any information held in their mind (Dempster, 1985). Moreover, declines in planning task performance are seen in older relative to younger adults. Several studies have found significant deterioration in performance on the Tower of London/Hanoi tasks due to normal aging (Allamanno, Della Sala, Laiacona, Pasetti, & Spinnler, 1987; Gilhooly, Phillips, Wyn, Logie, & Della Sala, 1999; Robbins et al., 1998). Research has also shown that older adults are poor at planning a sequence of actions in fictitious environments (Bisiacchi, Sgaramella, & Farinello, 1998), but their performance matches that of younger adults when executing an *externally imposed* strategy in such environments (Allain et al., 2005).

The prefrontal cortex is needed in novel and uncertain situations (e.g., Mesulam, 2002) but is not necessary when one can go on "automatic pilot" (Norman & Shallice, 1986; Reason & Mycielska, 1982). For instance, planning a task that has never been done, such as driving to a new job for the first time, would require the prefrontal cortex's false tagging mechanism, as each potential undesired route that is observed or retrieved from memory is given a false tag. But as an individual strengthens the PCRs for the task, such as using the most desired route to drive to work each day, the prefrontal cortex becomes less necessary and one can go on automatic pilot, not relying on the prefrontal false tags.

RESPONSE SELECTION AND RELATION TO THE SOMATIC MARKER HYPOTHESIS

Our discussion thus far indicates that, in the final analysis, prefrontal false tags are critical for appropriate PCR *selection* in several domains including cognitions, memory representations, potential action representations, and, as we will see below, perceptual representations. In effect, the doubt process is an elaborate biasing device against inappropriate and inconsequential PCRs to the current context. Thus, the prefrontal cortex acts to filter irrelevant information by negatively biasing these PCRs, which allows other PCRs to be believed and acted upon.

As mentioned above, Damasio (1994, 1996) has used prefrontal patients who have severe impairments in personal and social decision making to argue that their compromised ability to express emotion and to experience feeling directly results in their decision-making impairment. He suggests that a somatic signal marks potential response representations as good or bad and can be used as an alarm signal or an incentive signal (Damasio, 1996). The FTT can be conceived of as a more restricted version of the Somatic Marker Hypothesis, in that it proposes a unidirectional biasing (of the "alarm" type). However, it is also distinct from the Somatic Marker Hypothesis in that it also encompasses the entire prefrontal cortex and is not relegated to the ventromedial sectors. The next section will examine the role of false tags in attention, where the dorsal sectors of the prefrontal cortex have been centrally implicated.

DISTRACTIBILITY

Because the belief substrate is not simply cognitive representations but also includes perceptual representations, the FTT suggests that the failure of prefrontal patients to tag irrelevant stimuli as false extends beyond cognition to perception. Thus, the FTT may be relevant in the selection of perceptions for attentional focus. Indeed, another common symptom of prefrontal cortex damage is a persistent distractibility to extraneous perceptual information or an inability to hold focused attention (Damasio et al., 2012; Milner, 1964; Stuss & Benson, 1984; Stuss et al., 1982).

The concept of attention has always been intimately connected to the concept of working memory in neuroscientific research (e.g., Diamond, 2002), as a central question for theorists is whether the prefrontal cortex functions as a mnemonic, inhibitory, or attentional device (Braver, Cohen, & Barch, 2002). Thus far, we have dealt extensively with the inhibitory hypothesis of prefrontal cortex function but have neglected the mnemonic and attentional perspectives. Here we will examine these perspectives and the FTT's implications in regard to the attentional selection versus working memory debate of prefrontal cortex functioning.

In a seminal finding for the concept of working memory, Jacobsen (1936) discovered that monkeys with prefrontal lesions were severely impaired at delayed-response tasks where they needed to remember the location of a peanut for a few seconds. Investigators arrived at the natural conclusion that these monkeys could not remember what they had seen (Passingham & Rowe, 2002). The failure in delayed-response tasks has been traditionally attributed to a deficit in the ability to hold information in a working (or short-term) memory buffer. Baddeley and Hitch's (1974) influential multiple-component model of working memory proposed just such a buffer where the central executive could manage information and control attention. The concept gained further support when individual neurons in the prefrontal cortex of the monkey demonstrated sustained activity throughout the delay period of a delayed-response task (Fuster, 1973; Fuster & Alexander, 1971). Goldman-Rakic (1987, 1990) formally combined both notions and suggested that they were a cross-species manifestation of the same fundamental mental phenomenon (Postle, 2006b). The integration of Fuster's neuroscientific work and Baddeley's psychological work has given rise to the standard model of working memory, where the prefrontal cortex is critical for the storage buffer (or multiple domain-specific buffers; e.g., Baddeley, 2002) of working memory. However, recent research has challenged this view (D'Esposito, 2007; Postle, 2006b), and it has been suggested that the prefrontal cortex may not store memories, per se, but acts to gate out potentially distracting extraneous information that interferes with memory (Chao & Knight, 1995, 1998; Passingham & Rowe, 2002).

There are several lines of evidence suggesting that the prefrontal cortex does not store working memory representations in accordance with the standard model:

1. As Postle (2006b) points out, domain-specific buffers of working memory require ever-increasing dissociations in the prefrontal cortex, which, taken to its logical extreme, would require hundreds (or thousands) of domain-specific buffers. However, even the theorized spatial and object working memory buffer double dissociation remains elusive (Duncan & Miller, 2002). Instead, the majority of delay active prefrontal neurons do not discriminate spatial from object-related delay (Rainer, Asaad, & Miller, 1998; Rao, Rainer, & Miller, 1997), but adapt flexibly in a domain-independent manner to represent information that is currently relevant for task performance (Duncan & Miller, 2002).
2. Neuroimaging research has identified activation in the prefrontal cortex's area 46, which is critical to the standard model's storage when human subjects freely select among movements (Deiber et al., 1991; Frith, Friston, Liddle, & Frackowiak, 1991; Hyder et al., 1997; Jueptner, Frith, Brooks, Frackowiak, & Passingham, 1997), and a temporary lesion to the prefrontal cortex induced by TMS delays freely selected responses even without a memory load (Hadland, Rushworth, Passingham, Jahanshahi, & Rothwell, 2001). This indicates that the prefrontal cortex plays a central role in selection and not memory storage.
3. Many neuroimaging studies of delayed task performance do not find reliable prefrontal delay-period activity (Passingham & Rowe, 2002; Passingham & Sakai, 2004). Rowe and colleagues (2000) found delay-period activity in the prefrontal cortex only when their task placed high demands on attentional selection.
4. Passingham and Rowe (2002) have suggested that monkeys with prefrontal lesions may fail to perform

delay-response tasks not because they do not remember what they have seen, but because they are impaired at selecting among items in memory. The delay-response task is not without proactive interference (Mishkin & Delacour, 1975). During the setup for delay-response tasks, the monkey sees each side baited several times, and on any particular trial must select the side that has been baited most recently. Thus, impairment on the delay-response tasks may reflect a source memory or temporal ordering deficit, as subjects with prefrontal lesions are often poor at recency discrimination (e.g., Milner et al., 1991).

5. Finally, prefrontal lesions generally do not cause deficits in the maintenance of items in memory, but they produce problems when the information must be manipulated in some fashion (Chorover & Cole, 1966; D'Esposito & Postle, 1999; Ghent, Mishkin, & Teuber, 1962; Rushworth, Nixon, Eacott, & Passingham, 1997). Moreover, temporary lesions to the prefrontal cortex via TMS do not impair recollection of items from working memory but induce decreased performance when subjects must reorder and manipulate items (Postle et al., 2006; see also Feredoes, Tononi, & Postle, 2007; Hamidi, Tononi, & Postle, 2008; Luber et al., 2007).

Therefore, if the standard model is incorrect and the prefrontal cortex does not temporarily hold working memory representations, then perhaps the distinction between holding something in working memory for several seconds and focusing attention on something for several seconds is merely semantic (Diamond, 2002). This has led to the suggestion that working memory deficits in subjects with prefrontal lesions are the direct result of a decreased ability to focus attention or gate out extraneous perceptual and cognitive distracters (e.g., Postle, 2005, 2006a, 2006b). Several studies lend credence to this view, as it has been found that turning off the lights in the laboratory or mildly sedating the animal, which typically impair delay performance in healthy animals, improved performance in animals with prefrontal lesions (Bartus & Levere, 1977; Brutkowski, 1965; Malmo, 1942). Thus, decreasing the salience of irrelevant stimuli during delay improved the performance of animals with prefrontal lesions on tasks that have traditionally been explained via a working memory deficit. This presents another challenge to the standard model of working memory, as the salience of irrelevant stimuli should have no bearing on the ability (or inability) to hold memory representations in the prefrontal cortex. Prefrontal patients are also impaired in their ability to focus attention on task-relevant stimuli (Damasio et al., 2012; Knight, Hillyard, Woods, & Neville, 1981; Woods & Knight, 1986). Chao and Knight (1995, 1998) have confirmed that the prefrontal cortex is critical for gating distracting auditory and somatosensory information (monoaural clicks and brief electric shocks) during delay periods (see also Knight, Scabini, & Woods, 1989; Yamaguchi & Knight, 1990). Moreover, Desimone (1996) has used single-unit recordings to suggest that prefrontal neurons play a crucial role in protecting online information against distracters. The prefrontal cortex is activated in neuroimaging studies when subjects are required to ignore distracters during target detection (Coull, 1998). In addition, developmental studies indicate that children are more distractible than adults (e.g., Gumenyuk et al., 2001; Maurer & Lewis, 1998; Richards & Casey, 1992), and older adults are more distractible than younger adults (e.g., Chao & Knight, 1997; Connelly, Hasher, & Zacks, 1991; Healey, Campbell, & Hasher, 2008; Kim, Hasher, & Zacks, 2007).

One behavioral paradigm directly indicating that the prefrontal cortex is critical in the gating of irrelevant stimuli is the antisaccade task, which requires inhibition of reflexive glances to peripheral stimuli (Hallett, 1978; Hutton & Ettinger, 2006). Damage to the prefrontal cortex (predominantly the frontal eye fields, BA 8) results in an inability to suppress reflexive glances to the irrelevant information (Fukushima, Fukushima, Miyasaka, & Yamashita, 1994; Guitton, Buchtel, & Douglas, 1985; Walker, Husain, Hodgson, Harrison, & Kennard, 1998), and the prefrontal cortex is activated during the suppression of saccades in healthy adults (O'Driscoll et al., 1995). Performance on the antisaccade task improves continuously from 8 through 20–25 years of age (Fischer, Biscaldi, & Gezeck, 1997; Luna et al., 2001; Munoz, Broughton, Goldring, & Armstrong, 1998). Moreover, older adults are poor at the antisaccade task relative to younger adults (Butler, Zacks, & Henderson, 1999; Munoz et al., 1998).

The FTT provides an explanation for the increased distractibility of prefrontal patients. The prefrontal cortex false tags irrelevant stimuli, allowing focused attention and sustained working memory traces. Therefore, damage to the prefrontal cortex disrupts the ability to gate out or filter the irrelevant PCRs, leading to the increased distractibility. This perspective lends support to the emergent property view of working memory (D'Esposito, 2007; Postle, 2006b; see also Ruchkin, Grafman, Cameron, & Berndt, 2003) and offers a concise answer to the mnemonic, inhibitory, or attentional debate of prefrontal cortex functioning (Braver et al., 2002). As Diamond (2002) has indicated, subjects who have better performance on working memory tasks are better at (1) resisting distracting information (Conway & Engle, 1994; Conway, Tuholski, Shisler, & Engle, 1999; Hasher & Zacks, 1988), (2) performing inhibition tasks that lack large memory demands, such as the antisaccade task (Kane, Bleckley, Conway, & Engle, 2001), and (3) resisting proactive interference (May, Hasher, & Kane, 1999). The FTT reconciles this debate, providing a singular function for the prefrontal cortex across several modalities.

This view also addresses a tacit assumption made during Gilbert's belief study described above. Gilbert and

colleagues (1990, 1993) assumed that the attentional process and the belief assessment process competed for the same store of resources, and this competition decreased the performance of both processes (see Norman & Bobrow, 1975). However, there is no obvious reason why attentional processing should vie for the same resources of belief assessment processing. The FTT's view of the prefrontal cortex as a unitary structure that initially functions locally—which, with further demand, spreads to increased areas of the prefrontal cortex—does account for this assumption. The taxing attentional demand of responding to a distracter consumes some of the processing capacity of the prefrontal cortex needed for effective doubting of specific beliefs. Therefore, because proper false tagging for the two processes could not be supplied, individuals in the distracter condition tended to believe the false information relative to the nondistracter condition. Moreover, this may explain why divided attention in younger adults mimics aging (Craik, 1982), and also provides a clearer understanding of the poor performance of both younger adults under divided attention and older adults without divided attention in Gilbert's belief task (Chen, 2002; Chen & Blanchard-Fields, 2000).

It may also explain why, when individuals are in novel situations (such as the example of driving to a new job given above), distractions (such as talking on a cell phone while driving) are more difficult to deal with, and cause more errors and accidents, than when an individual can go on automatic pilot, without using the prefrontal cortex's false tagging mechanism, where individuals are more tolerant to distraction.

AN APPLICATION TO PSYCHIATRIC DISEASE

We have highlighted the case for false tagging largely using symptoms and deficits following damage to the prefrontal cortex; however, we theorize that there are other clinical populations that have disrupted false tagging mechanisms as well. This section will examine the FTT's implications for patients with delusions, focusing specifically on patients with schizophrenia.

Investigations into the phenomenology of pathological confabulations have noted that some prefrontal patients only produce temporal order confabulations, that is, real memories out of correct temporal order, but other patients produce more fantastic confabulations that are not real memories and tend to have a grandiose quality (Berlyne, 1972; Glowinski, Payman, & Frencham, 2008). Some investigators are reluctant to view confabulation as a source amnesia problem due to the implausibility of many patients' confabulations (e.g., Moscovitch, 1989). While it is not entirely evident why some confabulators produce fantastic confabulation or merely temporal order confabulations, Fisher and colleagues (1995) hint at a possible anatomical distinction. However, this distinction was not observed in other studies (e.g., Schnider, von Däniken, & Gutbrod, 1996) and remains a debated issue. Perhaps in the case of temporal order confabulations, the patient cannot false tag inaccurate memories only during memory retrieval but can false tag during encoding and normal ruminating; whereas during fantastic confabulations no false tagging for either memory encoding or retrieval can be performed. In fantastic confabulations, then, perhaps whatever cognition or perception that can be imagined is believed (Johnson et al., 2000). In this perspective, the patient has no way to falsify any rumination.

A clinical sign similar to that of fantastic confabulation is delusion, where patients cannot evaluate the accuracy of their cognitions or perceptions and make incorrect inferences about external reality (American Psychiatric Association, 1987). The clinical features often distinguish delusion (i.e., psychosis) from confabulation (i.e., frontal lobe damage; Fotopoulou, 2010; Kopelman, 1999). Malfunction of the prefrontal cortex is strongly implicated in clinical delusions (Coltheart, 2007; Ellis & Young, 1990; Young, Leafhead, & Szulecka, 1994; Young, Reid, Wright, & Hellawell, 1993). Benson and Stuss (1990) correlated various delusions such as reduplicative paramnesia, Capgras delusion, and spontaneous confabulation with frontal lobe damage in a series of case studies. Altered emotional functioning is prevalent in delusional populations (Fotopoulou, 2010), and disrupted dopaminergic-mediated prediction errors have been theorized as important contributors to delusions (Corlett, Taylor, Wang, Fletcher, & Krystal, 2010). Gilboa (2010) has indicated that delusional patients have a primary deficit in memory encoding as well as some mild retrieval memory impairments, suggesting that fantastic confabulations and delusion represent a broader false tagging impairment than temporal order confabulations. Therefore, in the FTT, patients with fantastic confabulations or delusions represent errant percepts or cognitions but fail to false tag the PCR, which results in such patients believing their errant percepts and cognitions.

Schizophrenia is a devastating psychiatric disorder, afflicting about 1% of the world's population (Sartorius et al., 1986). The signs and symptoms of schizophrenia are diverse, including "negative" symptoms (avolition, anhedonia, affective flattening, and inappropriate affect) and "positive" symptoms (formal thought disorder, disorganized speech, bizarre behavior, hallucinations, and delusions; Andreasen et al., 1995). Structural imaging has shown that patients with schizophrenia tend to have prefrontal cortex abnormalities such as reduced gray matter (Andreasen et al., 1994; Crespo-Facorro, Kim, Andreasen, O'Leary, & Magnotta, 2000); and functional imaging has revealed that less activation is found in the prefrontal cortex of patients with schizophrenia when challenged with executive function tasks (Andreasen et al., 1992; Berman, Zec, & Weinberger, 1986; Riehemann et al., 2001).

Substantial research and a long history of clinical observation have noted aberrant beliefs in patients with

schizophrenia. In our model, these patients have a disrupted false tagging mechanism. When errant percepts and cognitions are experienced, patients with schizophrenia are unable to falsify them. This deficit leads to a perseveratory tendency for many beliefs in the face conflicting evidence. Individuals with schizophrenia commonly present with delusions and have an increased rate of intrusion errors or "momentary" confabulations during story recall (Nathaniel-James & Frith, 1996). They are unable to distinguish plausible from implausible sentences and stories, indicating a lack of normative doubt, and cannot correct their own errors after listening to a tape recording of their own recall of a fable (Nathaniel-James, Foong, & Frith, 1996). Delusional individuals with schizophrenia are also impaired in their ability to integrate both pictorial and propositional disconfirmatory evidence with strong beliefs, even for information outside their delusional themes (Woodward, Moritz, Cuttler, & Whitman, 2006; Woodward, Moritz, Menon, & Klinge, 2008). Moreover, there is a positive correlation between religious delusions, psychoticism, and religiosity in individuals with schizophrenia (Feldman & Rust, 1989; Getz, Fleck, & Strakowski, 2001), who also have greater religious beliefs compared to the normal population (Brewerton, 1994; Kroll & Sheehan, 1989).

The FTT offers a clear rationale for why delusional patients believe cognitions and perceptions that are quite obviously false. Whenever normal individuals encounter a proposition or perception, their "belief index" is set to absolute belief and conviction. It is only during subsequent comparison of the new PCR with other PCRs in the belief script that normal individuals falsify and doubt the new aberrant PCR. The symptom of delusion would be elicited if this secondary process is disrupted, where individuals cannot doubt beliefs held with strong conviction. We argue that the prefrontal cortex is unable to apply false tags to PCRs in delusional populations such as those with schizophrenia. Therefore, when errant percepts and cognitions are experienced, they are believed. The remainder of this section will examine the symptoms and deficits of patients with schizophrenia, which, interestingly, parallel prefrontal patients' deficits.

Bleuler (1950) described stereotypy as one of the fundamental symptoms of schizophrenia, and numerous studies have shown an abnormally high frequency of perseverative errors on the WCST in individuals with schizophrenia (e.g., Berman et al., 1986; Buchanan et al., 1994; Wolkin et al., 1992). In addition, individuals with schizophrenia show reduced activation in the prefrontal cortex during the WCST compared to healthy comparison participants (e.g., Berman et al., 1986; Riehemann et al., 2001). In a detailed analysis of the WCST first four cards choices for patients with schizophrenia, Prentice and colleagues (2008) found that these patients had a specific deficit in using negative feedback appropriately, not an oversensitization toward rewards, and this accuracy on the first four cards predicted the overall WCST performance.

The inability to experience and use negative feedback in patients with schizophrenia has been investigated with several ERP studies. Patients with schizophrenia exhibited diminished error response negativity (ERN) amplitude relative to healthy subjects in a variety of experimental tasks, including Erikson-type flanker tasks (Kopp & Rist, 1999; Morris, Yee, & Nuechterlein, 2006), Stroop color-word naming (Alain, McNeely, He, Christensen, & West, 2002), go/no-go tasks (Bates, Kiehl, Laurens, & Liddle, 2002; Bates, Liddle, Kiehl, & Ngan, 2004), and picture-word naming (Mathalon et al., 2002). In a reversal learning paradigm, patients with schizophrenia also exhibited reduced feedback negativity (FBN), a related ERP component, which is observed when participants receive external feedback about erroneous responding or poor outcomes (Morris, Heerey, Gold, & Holroyd, 2008).

Patients with schizophrenia have increased false alarms on recognition memory tasks (Gold, Randolph, Carpenter, Goldberg, & Weinberger, 1992; Moritz, Woodward, & Ruff, 2003). Using Schnider and Ptak's (1999) paradigm testing the ability to inhibit irrelevant memories, which was described above, research has found that patients with schizophrenia who experience auditory hallucinations have pronounced increases in false-positive responses on the latter runs, in correspondence with confabulating brain-damaged patients (Badcock, Waters, Maybery, & Michie, 2005; Waters, Badcock, Maybery, & Michie, 2003). Patients with schizophrenia also have impaired memory for temporal order (e.g., Elvevåg, Egan, & Goldberg, 2000; Schwartz, Deutsch, Cohen, Warden, & Deutsch, 1991), are impaired at remembering the context of their memories relative to healthy comparison participants (Brébion et al., 2000; Moritz et al., 2003), and show deficits during planning tasks such as the Tower of London (Andreasen et al., 1992; Bustini et al., 1999; Morris, Rushe, Woodruffe, & Murray, 1995), which has been correlated with increased perseverative errors on the WCST (Bustini et al., 1999). Moreover, patients with schizophrenia are impaired at prefrontal-related attentional paradigms such as the anti-saccade task (Calkins, Curtis, Iacono, & Grove, 2004; Fukushima et al., 1994).

Emotional abnormalities are among the most striking features of schizophrenia. Blunted or inappropriate affect, anhedonia, alogia, and avolition are the central components of negative symptoms, where patients sometimes appear to have lost the ability to feel. An early demonstration of this phenomenon was performed by Dynes and Tod (1940), who showed that following an intramuscular injection of adrenalin, patients with schizophrenia failed to evoke the normative anxiety or fear emotional reaction, even though the somatic peripheral response was not different from that of normal subjects. While patients with schizophrenia rated unpleasant odors (Crespo-Facorro et al., 2001) and

unpleasant pictures (Paradiso et al., 2003) normatively, they failed to recruit subcortical limbic and paralimbic structures that were activated in healthy comparison participants, suggesting that they were not as emotionally aroused as the comparison group. Moreover, pharmacological or psychological treatment of a comorbid mood disorder tends to reduce delusions (Drury, Birchwood, Cochrane, & Macmillan, 1996; Serretti, Lattuada, Zanardi, Franchini, & Smeraldi, 2000). Dopaminergic and serotonergic systems, which are strongly associated with emotion, are also implicated in schizophrenia (Di Petro & Seamans, 2007; Salamone, Correa, Farrar, & Mingote, 2007; Swerdlow & Koob, 1987), as many antipsychotic drugs act to alter their availability.

Delusional patients often have prefrontal cortex abnormalities, persistent irrational beliefs, perseverations, deficits using negative feedback, source amnesia, and emotional abnormalities. The evidence presented indicates that there is a common defect between patients with delusions from a psychiatric disorder and patients with prefrontal cortex damage. We contend that both groups have a disrupted false tagging mechanism, which has resulted in a doubt deficit. However, there are differences between the groups. Prefrontal patients are often not delusional, and their florid confabulations tend to decline during the chronic stage of recovery. Undoubtedly, neural structures beyond the prefrontal cortex are also critical for the false tagging mechanism, such as the basal ganglia, basal forebrain, thalamus, amygdala, and brainstem nuclei, some of which have been implicated in psychiatric disorders. Future research will need to address how these structures interact with the false tagging mechanism and how distinct structural abnormalities can lead to persistent delusions.

CONCLUSION

The FTT is a parsimonious theoretical concept. It claims that belief is first, easy, inexorable, and substantiated by representations in the postrolandic cortex; by contrast, doubt is retroactive, difficult, vulnerable to disruption, and mediated by the prefrontal cortex. We believe that many of the classical symptoms following damage to the prefrontal cortex, such as perseveration, disinhibition, confabulation, and distractibility, can be explained by our theory. The FTT suggests that the prefrontal cortex performs the singular function of false tagging for disparate modalities, which compete for this resource in a flexible manner.

From an evolutionary perspective, the FTT allows us to speculate on how the anterior association cortex first evolved. If cognition is an evolutionary outgrowth of perception, the rudimentary ancestor of the prefrontal cortex may have evolved to discard irrelevant perceptual input and select stimuli for a response, perhaps using ancient extinction learning mechanisms present in simple learning systems (Hawkins, Clark, & Kandel, 2006). As cognition evolved, this initial false tagging may have been usurped to falsify stimulus-outcome associations and allow for probabilistic responding, where an organism would not need to rely on the most recent stimulus-outcome event to respond. Finally, as cognition and perception increased in complexity, the false tagging system expanded to influence the increased PCRs. The prefrontal cortex gained the ability to falsify and thereby select through *a process of elimination* the cognitive and perceptual information most relevant and important to the organism.

Although the FTT has abundant support from research, many questions remain. What are the boundaries of PCRs? What is the cellular mechanism for the belief threshold? What is the interaction between emotion and prediction errors? How do prediction errors influence false tagging on the cellular level? How do false tags influence PCRs on the cellular level? What is the involvement of attentional false tags and emotion? Is it qualitatively or quantitatively distinct? What is the contribution of false tagging from the basal ganglia and amygdala? Is there an anatomical distinction between temporal order confabulation and fantastic confabulation? What are the distinct prefrontal regions where the various components of false tagging perform initial processing? Evidence suggests that the superior medial prefrontal region is critical for attentional processing to perceptual representations (e.g., Stuss & Alexander, 2007), whereas the ventromedial prefrontal region is more critical for doubt processing to cognitive representations (e.g., Asp, Ramchandran, et al., 2012). How do the supplementary motor area, premotor area, and primary motor cortex fit into the false tagging picture? The circuitry of these structures with the basal ganglia is similar to that of the prefrontal circuitry with the basal ganglia, and the tendency of patients with frontotemporal dementia to have motor frontal release signs provides an intriguing parallel to false tags. In cases of pathological distractibility, such as attention deficit/hyperactivity disorder (ADHD), why is there often a hyperactive motor component? The connection of ADHD with the prefrontal cortex and the dopaminergic system is quite compelling (e.g., Jucaite, Fernell, Halldin, Forssberg, & Farde, 2005; Teicher, Ito, Glod, & Barber, 1996), and individuals diagnosed with ADHD who have pronounced hyperactivity are particularly poor at the antisaccade task (O'Driscoll et al., 2005). Certainly, the account presented in this chapter is an oversimplification of the complex neural interactions required for the false tagging mechanism and, as with most models, will need much refinement and elaboration.

These questions notwithstanding, we believe that the FTT has important implications for neuroscience and psychology. The model suggests that when the false tagging mechanism is inactivated, any rumination that is conceived is believed. Thus, the FTT may elucidate why we always believe the bizarre images and situations during dreaming (first commented on by Bagehot, 1871/1915). It

appears that no matter what outlandish notion is elicited, it is believed. Indeed, there is a selective deactivation of the prefrontal cortex during rapid eye movement (REM) sleep (Dang-Vu et al., 2005; Hobson, Pace-Schott, & Stickgold, 2000), a sleep stage strongly associated with dreaming. Moreover, there is a cessation of monoaminergic tonic activity during REM sleep that is thought to resensitize monoaminergic receptors (Hipólide et al., 2005; Jacobs & Fornal, 1993; Siegel, 2005; Siegel & Rogawaki, 1988; Tsai, Bergmann, Perry, & Rechtschaffen, 1993). Interestingly, the resemblance between confabulation, delusions, and dreams has been noted by several investigators (Scheid, 1934; Schilder, 1951; Whitty & Lewin, 1957); as Jung (1936, p. 79) remarked, "Let the dreamer walk about and act like one awakened, and we will have the clinical picture of dementia praecox." Research on this model may also shed light on other psychological areas such as déjà vu, sarcasm, lie detection, and humor; psychiatric disorders such as obsessive-compulsive disorder, ADHD, anorexia nervosa, and psychopathy; and social psychological biases such as the perseverance effect (Ross, Lepper, & Hubbard, 1975), the correspondence bias (Gilbert, 1991), the illusory truth effect (Hawkins & Hoch, 1992), and the truthfulness bias (Zuckerman, Depaulo, & Rosenthal, 1981).

Spinoza (1677/1982) defined emotions as states that make the mind inclined to think one thing rather than another. The notion that emotions determine beliefs has been a common assumption during much of human history (Frijda, Manstead, & Bem, 2000). For beliefs on issues of emotional importance to an individual, convincing someone to change his or her extant beliefs appears to be a virtually hopeless task. It is conventional wisdom that one should never discuss religion or politics at a dinner party. The underlying assumption is that the effort is futile and that individuals become emotional when arguing strongly held beliefs. The FTT suggests that attacks against ensconced beliefs do elicit negative emotion whose misattributions could prove to ruin a pleasant evening. As Abelson (1995, pp. 25–26) wrote:

> Throughout my academic career I have been fascinated by the capacity of holders of very strong attitudes to resist persuasive attempt at change. Public figures and ordinary folk alike often cling tenaciously to beliefs and attitudes that we, as know-it-all academics, are convinced are wrong-headed. Whether the attitudes concern life after death, gay rights, a perceived conspiracy to take over New Jersey, or whatever, we can argue until blue-faced without budging our State Representative or our Uncle Walter an inch.

The failures of rationalist approaches to alter the beliefs of disillusioned individuals may be elucidated by the FTT, as there appears to be a rich interplay between belief and emotion. This model suggests that emotion in fact determines what is believed, and what is believed determines emotion. As the ever-insightful William James (1905, p. 288) claimed, "In its inner nature belief, or the sense of reality, is a sort of feeling more allied to the emotions than anything else."

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