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Obsessive–Compulsive Disorder as a Disturbance of Security Motivation

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The authors hypothesize that the symptoms of obsessive–compulsive disorder (OCD), despite their apparent nonrationality, have what might be termed an epistemic origin—that is, they stem from an inability to generate the normal "feeling of knowing" that would otherwise signal task completion and terminate the expression of a security motivational system. The authors compare their satiety-signal construct, which they term *yedasentience,* to various other senses of the feeling of knowing and indicate why OCD-like symptoms would stem from the abnormal absence of such a terminator emotion. In addition, they advance a tentative neuropsychological model to explain its underpinnings. The proposed model integrates many previous disparate observations and concepts about OCD and embeds it within the broader understanding of normal motivation.

Kurt Gödel, the eminent 20th century mathematician, sought to live life as a "quest for rationality in all things" (Dawson, 1999, p. 76). In his work, he used logic even to reveal the hidden limits of logic: His famous incompleteness theorem showed that there exist statements in consistent mathematical systems that are true but unprovable, a result that was stunningly counterintuitive and even dismaying to the mathematicians of his time.

In quite a different way, Gödel's private life, too, illustrated the sometimes bizarre limits of rationality. For most of his life, this brilliant logician was plagued with senseless obsessions about the possibility of being poisoned—for example, accidentally via food or by gasses somehow escaping from his refrigerator. For many years, his wife had to serve as his food taster and coax him to eat; when she became seriously ill and unable to help him in this way, his obsessions about being poisoned led him to starve himself to death (Dawson, 1997).

Other preeminent intellects have also had their islands of seeming irrationality. Another good example is Samuel Johnson, one of the most important writers of the 18th century. This outstanding enlightenment figure, whose work has been described as "in defense of reason against the wiles of unchecked fancy and emotion" (Mahoney, 2000), was beset by many inexplicable compulsions, such as needing to touch every post in a street or step exactly in the center of every paving stone. If he perceived one of these acts to be inaccurate, his friends were obliged to wait, dumbfounded, while he went back to fix it (Stephen, 1900).

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Although such obsessions and compulsions can occur in a variety of disorders, they are most familiar as symptoms of obsessive–compulsive disorder (OCD). The main purpose of the present article is to advance a relatively novel hypothesis about the psychological processes that underlie such repetitive, seemingly inexplicable thoughts and actions. In brief, we hypothesize that such symptoms, despite their apparent nonrationality, have what might be termed an epistemic origin—that is, they stem from an inability to generate the normal "feeling of knowing" that would otherwise signal task completion and terminate the expression of a security motivational system.

To introduce our proposed theory, we first provide some background information about OCD. We then identify OCD symptoms as the behavioral output of an open-ended motivational system, explain why it makes sense to regard an emotion (the feeling of knowing) as the normal satiety signal for this class of motivated behaviors, and indicate why OCD-like symptoms would stem from the abnormal absence of such a terminator emotion. Finally, after comparing our satiety-signal construct, which we term *yedasentience,* to various other senses of the feeling of knowing in the psychological literature, we advance a tentative neuropsychological model to explain its underpinnings.

Description of OCD

OCD previously has been considered a rare psychiatric disorder, but recent studies estimate that in the general population, the lifetime prevalence of OCD is 1%–2%, twice that of schizophrenia or panic disorder (Rasmussen & Eisen, 1991). Symptoms consist of recurrent and persistent thoughts (*obsessions*) and/or repetitive, relatively stereotyped behaviors (*compulsions*) that the person feels compelled to think or perform but recognizes as irrational or excessive (Goodman et al., 1990).

According to Reed (1985), the content of obsessions may include thoughts involving (a) self-depreciation, self-denigration, and unworthiness, with reference to either perceived social norms or the individual's own standards; (b) undue concern with one's bodily functions; (c) fears, doubts, and preoccupations about dirt

and the spread of disease to self; and (d) fears of harm to a friend or relative and being responsible for the imagined event. The range of compulsive behaviors includes (a) excessive checking activities, characterized by repeated redoings of actions supposedly related to security, orderliness, or accuracy (Reed, 1985); (b) avoidance behaviors, which are "activities engaged in to avoid feared objects, places, or situations" (Reed, 1985, p. 37); and (c) washing and cleaning, generally of hands but sometimes also compulsive washing of clothes, teeth-cleaning, or the cleaning of possessions or parts of the home (Reed, 1985). As with obsessions, compulsions may involve more than one category of behavior. Some OCD patients (51%) have compulsions without obsessional thoughts (Rachman & Shafran, 1998; Rasmussen & Eisen, 1991). The most common subjective clinical features are doubts and indecision; the two most common compulsive behaviors are checking and washing (Henderson & Pollard, 1988; Rasmussen & Eisen, 1992).

OCD as a Pathology of Stopping

To an observer, OCD is a particularly fascinating disorder because it exemplifies a flagrant violation of our everyday view of rationality. OCD sufferers are well aware of external reality, generally recognize the absurdity of their obsessions and/or compulsions (a minority have poor insight; Foa et al., 1995), and prefer not to engage in them. Yet, despite this strong tie to reality, they knowingly continue to perform such activity at a tremendous cost not only to themselves but also to those around them (Hollander et al., 1996). Clearly, OCD is a stark demonstration that normal control of behavior can be overridden by some powerful noncognitive-based system(s).

A defining attribute of OCD is a sense of compulsion associated with the performance of ritualistic thoughts or actions. The psychological experience of compulsion is not well-defined (Reed, 1985), but nevertheless one can conceive of two broad mechanisms that would produce the intrusiveness and urgency characteristic of OCD symptoms. One is a pathological intensity of excitation of the particular thoughts, ideas, or actions. The other is a relative failure of the systems that normally terminate such thoughts, ideas, or actions.

At first blush, the notion that OCD symptoms spring from a pathologic intensity of excitation is a reasonable one, because it aligns positively with the prevalent concept of compulsion as a force that drives behavior against one's will. However, as noted by Reed (1985), this concept of compulsion is but a slight variant of the medieval belief in demons that invade the body and compel behavior against one's will. The modern version no longer holds that the force comes from outside the body but rather that it has an inner origin (Reed, 1985). But is it in fact the case that OCD patients describe their experience of compulsion in terms of an overpowering force? The study described below suggests that they do not.

Reed (1977b, 1985) interviewed over 60 OCD patients and analyzed their responses to the question: "What does the compulsive experience feel like?" Analysis revealed that by far the greatest majority of patients (70%) described the experiencing of compulsions in terms of some impairment of will power, as for example, "I keep wondering, and then I can't get it out of my mind. I know it's stupid, but I haven't got the will-power to push it out" (Reed, 1985, p. 127). Sixteen percent emphasized that their experience was one of stickiness, as for instance, "I can't move on because I can't convince myself that I've finished what I'm doing" (Reed, 1985, p. 127). In contrast, only 4% of the respondents laid major emphasis on the power of the compulsions, as for instance, "The idea is overwhelming. It just keeps coming back, and there's nothing I can do about it" (Reed, 1985, p. 128). On the basis of such findings, Reed concluded that "those who are trapped in a circle of repetitive behavior do not report that something forces them to *continue*, but that they lack something to make them *stop*" (Reed, 1977b, p. 384).

That the key problem is one of stopping seems reasonable also from the limited information available regarding the structure of OCD compulsive behavior. Available descriptions of OCD behavior suggest that most patients engage in few but extended bouts of compulsive behavior during the day (as opposed to bouts of behavior that are relatively normal in duration but repeated excessively often throughout the day; Neziroglu & Yaryura-Tobias, 1991). Such a behavioral profile is consistent with a dysfunctional stop mechanism rather than one that is involved in the activation of behavior.

Does the presumed OCD pathology in the mechanism of stopping extend to terminating all thoughts, ideas, or actions? Clearly not, because the patients' symptoms are not general but circumscribed in some manner. Typically, it is only certain types of thoughts that are problematic. Explaining this specificity is a major challenge for psychological theories of OCD, to which we now turn.

Conceptualizations of OCD as a Cognitive Disorder

Previous conceptualizations of OCD have often focused on the hypothesis that there is an underlying disorder of cognition. Here we focus on Reed's (1985) attempt to identify the core problem in OCD and relate it briefly to other cognitively oriented explanations. As will eventually become clear, our own proposal is a modification and extension of some of Reed's ideas.

Largely on the basis of an analysis of the form of obsessional thoughts, Reed (1968) suggested that the clinical symptoms of OCD might all be regarded as manifestations of, or reactions to, one central cognitive phenomenon—namely, a "functional impairment in the spontaneous organization and integration of experience" (p. 382). He argued that this experiential impairment stems from the patient's cognitive problem in the defining of categories, in the determination of boundaries and limits, in the establishment of criteria, and in the allocation of class members (Reed, 1985). We normally form such classifications spontaneously, but according to Reed (1968, 1985), the obsessional individual cannot do so. Consequently, obsessional people look for external ways to classify their experience, ways that may include not only artificial overstructuring of input and maladaptive overdefining of categories and boundaries but also the use of rituals as arbitrary "time-markers" or "crypto-decisions" (Reed, 1968, 1985). In other words, the doubt and indecisiveness so characteristic of the clinical picture is, according to Reed (1968, 1985), a direct manifestation of patients' cognitive inability to define and put closure to an experience, and their obsessional cognitive style and rituals are, in turn, attempts to compensate for this inability. Later writers (e.g., Pitman, 1989) refer to the inability to experience closure as a failure in "the sense of task completion."

Some experimental tests support this hypothesis. For instance, Milner, Beech, and Walker (1971) showed that even though signal detectability and response bias are the same in obsessional and nonobsessional people performing an auditory signal detection task, their performance is different when they are allowed to hear the trial again before rendering judgment as to whether the tone was played. Individuals with obsessional symptoms ask for significantly more repeats of a trial than those without obsessional symptoms, consistent with the prediction that obsessional individuals will demand more information to arrive at a decision than nonobsessional individuals. Similarly, Reed (1977a) showed that decision difficulty experienced by the obsessional individual is inversely related to the amount of structuring available in the task itself. Deductive tasks present little difficulty because the task itself provides inherent logical closure. Open-ended tasks, which demand an inductive or intuitive approach, favor indecisiveness and uncertainty because they have little external structure to define closure and consequently elicit an overproduction of competing hypotheses that characterize the overcompensating obsessional cognitive style.

One noteworthy feature of Reed's (1985) hypothesis is the identification of OCD as a cognitive disorder, in which "obsessional difficulties reflect . . . maladaptive ways of thinking, of reasoning, and of attending to, assessing, processing, and assimilating information" (Reed, 1985, p. xiii). A cognitive perspective was a radical departure from the then prevalent view (first promulgated by Freud) of OCD as a disorder of affect or anxiety; Reed noted, however, that his perspective was in the tradition of 19th century French writers such as Pierre Janet (see Janet, 1903). Other recent explanatory models of OCD have also been strongly cognitive; for example, a major line of theorizing has implicated dysfunction in the metacognitive regulation of one's own stream of thoughts (Purdon & Clark, 1999). Accordingly, Salkovskis (1985, 1989, 1998), Rachman (1997, 1998), and Wells (1997) have suggested a causative role for various dysfunctional beliefs that OCD patients appear to have about the meaning and implications of their conscious thoughts—for example, the belief that thinking something bad is virtually the same as actually doing it (thought–action fusion).

A Reconceptualization of OCD in Terms of Motivation

There is, however, a second very important feature of Reed's (1985) hypothesis, one that leads more directly into our own proposal. It is the heuristic power obtained from reducing the obsessional problem to a specific deficit, namely, an inability to obtain closure in an unstructured (open-ended) situation or task (Reed, 1983). We retain Reed's (1985) essential notion that OCD patients suffer from failure to put closure on experience, but we put a restriction on the domain of these experiences and ascribe the core deficit to a noncognitive process.

Although Reed's (1985) emphasis on closure was extremely insightful, the attribution of the core problem to the domain of cognition was, in our view, incorrect. The main reason for the inadequacy of a cognitive explanation is the specificity of OCD symptoms. If it were indeed true that patients suffered from a broken cognitive module (used for classification of information), then they should have profound intellectual difficulty with very many everyday tasks. Such is not the case, however. Although some neuropsychological studies indicate that OCD patients may show limited and highly specific neurocognitive deficits, especially on tests tapping complex visual–spatial functions (Bolton, Raven, Madronal-Luque, & Marks, 2000; McNally, 2000; Purcell, Maruff, Kyrios, & Pantelis, 1998), the relationships of such deficits to the origins and course of the disorder is unclear (Summerfeldt & Endler, 1998). Indeed, outside of their particular obsessional thoughts and compulsive rituals, OCD patients seem remarkably normal; some may even rank among those with superior talents, Samuel Johnson being one outstanding example. Even more problematic for the notion of a general cognitive impairment is that despite their variegated and idiosyncratic content, OCD symptoms do possess a thread of continuity across most patients. As noted before, the content of most obsessional thoughts, ideas, or actions revolves around the issue of security or safety, either of the self or of others (Salkovskis, 1985). In summary, they are concerned with self-preservation or preservation of the species, to paraphrase an expression used in another context by Paul MacLean (MacLean, 1973). A general cognitive processing deficit would not restrict the content of OCD symptoms to such a specific concern but would generate a multitude of symptoms with little invariance in content.

In distinction to the cognitive framework is a biological psychiatry perspective on OCD. It is characterized by two currents. One, which we review later, is the appreciation that however complex OCD symptoms may appear to be, they probably result from a dysfunction in a neural substrate that involves a cortical–basal ganglia circuit. The second is the use of ethological concepts to interpret OCD symptoms (Rapoport, 1989a). OCD behaviors are seen to resemble "fixed-action patterns" (Lorenz, 1970). As such, OCD symptoms are posited to reflect the inappropriate release of specific behavioral packages preprogrammed through evolution and biologically hardwired in brain (basal ganglia) circuitry (Rapoport, 1989a; S. Wise & Rapoport, 1989). In the words of Rapoport and Fiske (1998, p. 160): "Such specificity in the 'hard wiring' of human behaviors as basic to everyday functions as cleaning, checking, and ordering, or concern about dangers to self or others, suggests a biological basis for universal categories of certain thoughts and rituals."

Given the universality of OCD symptoms and their circumscribed focus on biologically primitive concerns regarding self-preservation and preservation of species, one may suggest that OCD symptoms constitute the expression of a special (*security*) motivational system. A motivation with security as its goal is not among the five special motivations recognized classically in psychology, which are hunger, pain, sex, maternal, and exploratory motivations (Hebb, 1966). However, ethologically oriented psychologists (e.g., Adams, 1979; Bolles & Fanselow, 1980; Gilbert, 1989; Marks & Nesse, 1994; Masterson & Crawford, 1982; Trower, Gilbert, & Sherling, 1990) have advanced the notion of a motivationally fundamental "defense system" concerned with the detection and amelioration of both physical and social potential threats to security. In general terms, *motivation* refers to the tendency of the whole organism to be active in a selective and organized way, and *special motivation* refers to that tendency that is also biologically primitive and necessary for species survival (Hebb, 1966). Thus, *security motivation* refers to a set of biologically based (hardwired), species-typical behaviors directed toward protection from danger of self and others, suggesting the operation

of a tendency that is biologically primitive and necessary for species survival. Furthermore, the species-typical behaviors for protection of self or others include behaviors characteristic of OCD, namely, cleaning, checking, and hoarding, and presumably also the associated thoughts and ideas. In the next section, we lay out the main working characteristics of such a security motivation system.

The Security Motivation System

An evolutionary perspective suggests that certain sets of behaviors have come to be organized within domain-specific, fairly independent systems or modules, which addressed specific adaptive problems and thereby contributed to fitness (Pinker, 1997; Tooby & Cosmides, 1990, 1992; Trower et al., 1990). For example, even the human capacity for language has been viewed in this way, as a core innate system of rule schemas or grammatical computations that is calibrated and elaborated by experience (Hauser, Chomsky, & Fitch, 2002). Fodor (1983) argued that perceptual input systems in particular tend to be modularly organized, to enable the rapid processing of information of potential relevance to survival. Such modular systems are innately specified and hardwired, highly tuned to certain kinds of input, comparatively automatic and autonomous, and *encapsulated* (i.e., relatively isolated from information developed by other systems). The security motivation system is such a mental module, but to Fodor's reasoning about the perceptual input side, we would advance similar arguments about the behavioral output side. In particular, to enable quick action for alleviating risk, the security motivation system requires specifically tuned outputs associated with risk assessment and reduction, outputs which are relatively automatic, autonomous, and encapsulated—thus avoiding interference from other systems concerned with less immediately pressing agendas.

Research by ethological psychologists and ecologists on how animals assess and respond to the risk of predation strongly supports such a concept of a security motivation system. As Lima and Bednekoff (1999) remarked,

> Temporal changes in the risk of predation are a fact of life for most animals, and hundreds of studies demonstrate that animals respond quickly and adaptively to changes in risk of predation. . . . As a consequence of this variation in risk, animals are generally faced with the problem of how to best allocate feeding and antipredator efforts across different risk states. (p. 656)

More specifically, this research suggests the following major working characteristics of a security motivation system:

1. The system is tuned to detecting potential danger and does so on the basis of often subtle and indirect cues. Blanchard and Blanchard (1988, p. 64) noted that "the risk-assessment pattern is . . . related to defense, but it occurs in the context of unclear or partial threat stimuli and has the primary goal of acquiring information needed either to make the defense pattern effective or make it unnecessary." Likewise, Curio (1993) distinguished innate releasing mechanisms for recognizing predators from those for decoding hidden risk:

> Apart from assessing overt risk, prey animals make use of various risk-assessment mechanisms even in the *absence* of any overt predator cues. . . . These hidden-risk mechanisms are already known to be highly diverse and to consist, for example, of an assessment of unclassifiable risk, of risk permanence . . . after the predator's disappearance, and of the risk posed by the presence of an especially vulnerable prey conducive to attack. (pp. 225–226)

In summary, compared with real and present danger, potential danger requires different perceptual processing and different responses, and there exists a class of behaviors directed at the assessment and alleviation of such potential danger.

2. The system is readily activated, responding to even a slight chance of danger, and once activated, it has a long half-life, being slow to deactivate despite changes in the environment that feed into the appraisal process (Curio, 1993; Marks & Nesse, 1994; Masterson & Crawford, 1982). This easy-to-turn-on, hard-to-turn-off quality makes sense evolutionarily, because repeated false alarms are much less costly than even a single failure to prepare for upcoming danger. In addition, the system may be activated by threats to offspring and to members of the animal's social group, as well as to the animal itself (Curio, 1993).

3. The system is oriented toward action. For example, Curio (1993, p. 137) noted that "an ongoing antipredator behavior can profitably be seen, in part, as *manipulating* and *probing* the behavior of the predator." Behaviors such as checking and surveillance serve as preemptive actions, because predators rely on surprise, as well as active ways to gather information. As Masterson and Crawford (1982) pointed out, "the activation of a motivation system excites or primes motor acts relevant to the motive. . . . a drive is accompanied by a *readiness* for motivationally relevant innate actions" (p. 670).

4. The security motivation system can be distinguished from other systems that protect the animal from noxious events, such as what has been termed the "pain motivation system" (Bolles & Fanselow, 1980) or "alarm reactions" (Masterson & Crawford, 1982). Of particular importance is the distinction between the security motivation system, which is oriented around prevention, and what Öhman and Mineka (2001) have termed the "fear module," which mediates fear learning, such as escape and avoidance. Öhman and Mineka (2001) remarked,

> Potentially disastrous events . . . may be heralded by subtle cues. For example, to the attentive observer, a predator may announce its presence by faint sounds or odors. By using the contingency between such cues and the potentially deadly consequence, the central motive state of fear . . . [can] be conditioned to the cue. (p. 483)

In contrast to the fear module, the security motivation system does not rely on actual encounters with the "potentially deadly" consequences but operates through other, subtler unconditioned stimuli signaling hidden risks, often in the absence of any overt danger, as detailed earlier. Another distinction is that, as also noted earlier, security-motivated behavior is often directed toward probing and acquiring information rather than simply avoiding noxious stimuli. Likewise, wariness and/or anxiety is the emotional state of the security motivation system rather than fear, which is the affective state of the fear module (Masterson & Crawford, 1982). In this way, the security motivation system, part of what Trower and his colleagues (Trower et al., 1990) labeled "the defense system," may also be distinguished from what they call "the safety system," which is posited to work through positive reinforcement and affect afforded by safety cues (see also Chance, 1984).

The Open-Ended Nature of the Security Motivational System

To the extent that OCD reflects a special motivational system, the study of OCD mechanisms can be informed by questions and findings in the extensive literature on motivation. For instance, are the factors that determine the beginning and end of motivated activity the same ones that start and stop OCD activity? Is the security motivational system different in an important way from the other special motivational systems, and is such a difference relevant for OCD? These particular questions are of special pertinence to the hypothesis advanced here and are addressed next.

A change in responsiveness to a constant stimulus in the environment is one attribute that defines motivation. There are distinct mechanisms that invoke the change in responsiveness for different motivational systems. For example, in the case of loss of water, hunger for water (thirst) is the invoked tendency that dominates behavior and directs the organism's activity to seek and consume (drink) water, which terminates thirst. Because ultimately the internal physiological norm is restored, hunger motivation is a homeostatic mechanism, and the ingested substance that terminates motivated activity is known as a "consummatory stimulus" (Hinde, 1970). Hunger motivation is a closed loop system, with specified start and stop events, though not all stimuli that compose these events are known.

Unlike hunger, sexual motivation is not considered homeostatic because copulation is not known to correct deviations of a vitally regulated substance. Nonetheless, sexual motivation is a powerful organizer of activity, crucial for preservation of the species, and another closed system with a real consummatory stimulus to terminate it (i.e., contact with a mate).

Although other special motivations share, to a varying degree, attributes of either hunger or sexual motivation, the proposed security motivation is distinct in a crucial respect because it does not have reality-based consummatory stimuli and is, in effect, an open-ended system. Regardless of how security motivation starts, its termination has a built-in problem. Consider an organism that has observed a predator at a distance—how long should it continue to check for impending danger? There is no straightforward rule because the inability to see the predator is no guarantee that it is gone. It would make sense for checking to continue for some time, well beyond the last sighting.

The problem is exacerbated if we consider that the species-typical activity of checking for predators or other sources of harm is generally invoked when there are no real stimuli to stimulate the senses—that is, motivated activity is directed toward potential sources of danger. As an example, Trower et al. (1990, p. 19) noted that "animals emerging out of their burrows will sample the local environment for the presence of threat." Here, external confirmation that there is no potential danger is logically impossible, and for this reason we claim that reality-based consummatory stimuli for security motivation do not exist. As such, the task engaged by the security motivation system has no external structure and is open-ended in the sense used by Reed (1985). What then terminates it? We propose that termination of motivated security activity is induced by an internally generated feeling of knowing, a crucial idea to which we now turn.

Termination, Satiation, and Feeling of Knowing

When a severely dehydrated person drinks rapidly for a few minutes, thirst motivation disappears long before the fluid can be absorbed by the gut and correct the chemical changes that generated the thirst (Denton, McKinley, & Weisinger, 1996). Thus, even though the consummatory stimulus that inhibits water-directed activity is the drinking of water, the specific stimuli that actually terminate thirst motivation are not the same ones that invoked it. The phenomenological sign to stop drinking is a feeling of satiation. This feeling is distinct from the feeling of thirst, as disorders of either exist with life-threatening consequences (Denton, Shade, Zamarippa, Egan, Blair-West, McKinley, & Fox, 1999). Of importance, this feeling may not only represent the subjective correlate of the stop signal but, in fact, constitute the critical mechanism that turns off the internal motivation. Similarly, we hypothesize that an internally generated feeling of knowing provides not only a phenomenological sign of goal attainment but is also the physiological mechanism that actually shuts down security motivation. This kind of mechanism would be especially critical in an open-ended motivational system, in which reality-based consummatory stimuli do not exist. By the same token, it must follow that without environmentally supplied cues, stimulation of the feeling of knowing comes directly from performance of the behaviors evoked by security motivation. Such an arrangement may be a special instance of the more general proposition advanced by Glickman and Schiff (1967) that mere engagement in motor activity is reinforcement, a notion to which we return later.

We label the terminator signal as a feeling of *knowing,* rather than of *safety* or some similar term, for three reasons. First, there exists a psychological literature on feelings of knowing (see below), and our use of the term shares features with the usage there. Second, a feeling of safety suggests a mood state induced by environmental stimuli, but that is contrary to our hypothesis of an internally generated phasic signal of goal attainment in the absence of consummatory stimuli in the environment. Finally, a feeling of knowing captures the essential implication of the hypothesized terminator signal: a subjective conviction functionally separate from knowledge of objective reality.

Restatement of the Core Problem in OCD

We now have the necessary ingredients to reformulate Reed's (1985) hypothesis of the core problem: namely, an open-ended security motivational system that requires an internally generated feeling of knowing to signal goal attainment and shut down the motivation. Let us re-examine the main features of OCD and relate them to this framework.

One of the most striking features of OCD is the inability to feel reassured by seemingly obvious and compelling information from the senses. The perceptions of OCD patients, although objectively sound, do not seem to sink in or feel right; indeed, some older terms for OCD include *folie de doute* (French), *Zweifelsucht* (or *Grübelsucht;* German), and *follia del dubbio* (Italian)—all of which translate as "the doubting (or questioning) mania" (Baldwin, 1901). Rapoport (1989b) described the predicament of OCD sufferers as follows:

> The doorknob must be turned again and again; the light switched on and off, on and off. These acts bring immediate information, yet it

> doesn't get through. They can't say, "Yes, I have checked this out and now I know that the door is locked." (p. 238)

Accordingly, OCD appears to stem from a particular disturbance in subjective convictions about reality: Concerning the problematic content, OCD sufferers know objectively but cannot believe subjectively. For example, even though the compulsive hand washer knows objectively that his or her hands look clean, he or she cannot readily generate the subjective conviction that they are truly clean and so continues to wash.

According to the framework we are proposing, OCD patients are haunted by the subjective sense that things are wrong because of the following set of events. First, their particular concerns and behaviors were invoked by a very potent special motivation that handles basic threats to existence (e.g., predation). Second, this motivational system is open-ended with no external consummatory stimuli and so inherently unhooked from immediate environmental control. Third, because of this lack of a terminating signal in the environment, goal completion is normally signaled by an endogenously generated terminator (experienced as a feeling of knowing), but OCD patients cannot generate this emotional signal or it is inadequate to inhibit the invoked motivation.

Our model is similar to Reed's (1985) in identifying, as the core deficit, a failure to put closure on an experience, but we constrain it to experiences invoked by a biologically primal motivation for protection of self and others and consider that the failure to put closure on experience does not stem from cognitive inability but from the breakdown in a satietylike mechanism that normally generates a feeling of knowing. The feeling of knowing ultimately derives its power as a terminator from primal, compelling emotions having to do with basic threats to existence. In this sense it has a close parallel with, for example, the phenomenon of thirst (Denton et al., 1996). Water deprivation elicits a primal compelling emotion of thirst that can entirely occupy the stream of consciousness. Denton, Shade, Zamarippa, Egan, Blair-West, McKinley, Lancaster, and Fox (1999, p. 5308) pointed out that severe thirst is less easily ameliorated cognitively than some other emotions: "Thirst . . . is interoceptor-driven and initiated through mechanisms in the phylogenetically ancient brain, as with hunger for air. The relative inaccessibility of the primal emotions to the higher amelioration may reside in this fundamental of brain organization."

Refinement of the Concept of a Feeling of Knowing

In the cognitive literature, the term *feeling of knowing* has been widely used to refer to the intuitive sense that one knows some piece of information even though at the moment one cannot yet bring it to mind (e.g., Nelson, Gerler, & Narens, 1984). We retain this distinction between the subjective sense of knowing and one's objectively verifiable knowledge. The notion of a feeling of knowing is also akin to psychological processes that are hypothesized to underlie fundamental intuitions or insights, such as the sense of number, which serves as a basis for mathematical thinking (e.g., Dehaene, Spelke, Pinel, Stanescu, & Tsivkin, 1999). However, the feelings of knowing involved in OCD have to do with the subjective sense of what is real (Rapoport, 1989b). There is a long history to regarding such subjective intuition about reality as an important factor in psychopathology. For example, Jaspers (1913/1963, pp. 93–94) pointed out that "conceptual reality carries conviction only if a kind of presence is experienced"—that presence, he argued, being a primary, irreducible phenomenon. Furthermore, he noted, "our attention gets drawn to it because it can be disturbed pathologically and so we appreciate that it exists" (p. 94). In a similar vein, William James (1890) remarked

> *In its inner nature, belief or the sense of reality, is a sort of feeling more allied to the emotions than anything else. . . . The true opposite of belief,* psychologically considered, *are doubt and inquiry, not disbelief.* In both these states the content of our mind is in unrest, and the emotion engendered thereby is, like the emotion of belief itself, perfectly distinct, but perfectly indescribable in words. Both sorts of emotion may be pathologically exalted. (pp. 283–284)

James identified the pathological excess of the emotion of doubt as "the questioning mania," an earlier term for OCD, as mentioned above.

More generally, Damasio (1994) has argued that our sense of what is reasonable and real, and even our capacity for rationality, is undergirded critically by emotion. In his somatic-marker hypothesis, he proposes that somatic states or signs experienced as feelings are an "indispensable foundation for rationality" (Damasio, 1994, p. 200) in that they direct and shape everyday thoughts and decision making. In support of this hypothesis, he and his colleagues have shown that the impoverished decision-making capacities of some frontal patients seem to be due to the lack of somatically mediated feelings of knowing rather than any difficulty with logical skills (Bechara, Damasio, Tranel, & Anderson, 1998; Bechara, Damasio, Tranel, & Damasio, 1997; Bechara, Tranel, Damasio, & Damasio, 1996). The implication is that, unlike the classic Western assumption, feeling is not opposed to rationality but part of it.

What we mean by the feeling of knowing in the present context shares features with these concepts but is also quite different. Rather than the feelings of knowing that serve as a guide or director of thinking, as in intuition, we are proposing that a particular feeling of knowing serves as an essential terminator of a species-specific motivation, namely, one concerned with protection from harm. To distinguish this meaning from the broader usage of the term *feeling of knowing,* we coined the term *yedasentience* (Woody & Szechtman, 2000), from the Hebrew *yeda* [knowing] and the Latin *sentire* [to feel]. The core hypothesis we are advancing may then be stated as follows:

> An internally generated feeling of knowing (termed *yedasentience*) provides a phenomenological sign of goal attainment and has as its consequence the termination of thoughts, ideas, or actions motivated by concerns of harm to self or others. Failure to generate or experience this feeling produces symptoms characteristic of OCD.

Our proposal is interestingly related to Zald and Kim's (2001) speculation concerning the possible impairment of sensory-specific satiety in OCD: They noted that OCD patients "in essence fail to reach a point at which they feel 'satiated' in their safety" (p. 59). In summary, yedasentience serves as a satiety signal. Thus, the security motivation system has two distinct emotional states: Anxiety is part of a go signal, and yedasentience is the stop signal. The absence of anxiety is not yedasentience, just as the absence of thirst is not the feeling of satiety.

Conceptual Sketch of a Motivational Model of OCD

Our conceptual model of OCD as a dysfunction of security motivation is summarized in Figure 1. The model posits the operation of four major functional components (shown across the middle of the figure) and three major routes of feedback (shown above and below). The first component is the Appraisal of Potential Danger, which evaluates incoming environmental stimuli in the context of the organism's experiential history and intended actions. If results indicate a potential threat to self or others, this component outputs an excitatory signal to a second component, Security Motivation. Stimulation of this subsystem activates a motivational state with a protracted half-life. In particular, the output of the Appraisal subsystem may change quite quickly, given change in external stimuli (including Safety Cues), context, and plans. In contrast, even an excitatory signal of short duration from the Appraisal subsystem to the Security Motivation subsystem is hypothesized to activate the latter system for a fairly extended time period. This hypothesis follows directly from the idea that security motivation cannot be under the direct control of factors like external stimuli, because such stimuli are unreliable indicators of the lack of danger.

When activated, the Security Motivation subsystem generates a set of coordinated outputs that serve to energize and focus the actions of the organism on attaining a specified goal. Among the activated outputs is an Anxiety-related feedback to the Appraisal component; this positive feedback signal provides the organism with a further interoceptive cue indicating potential danger and forms part of a loop that acts to sustain the appraisal. In addition, another activated output is an excitatory signal to a third major component, Security-Related Programs, which coordinates and executes species-typical motor and cognitive programs for the protection of self or others. These programs are instructions for performance of acts such as checking or washing; the selection of the appropriate program is dictated by the informational signal from the Appraisal subsystem. Engagement in the performance of the activated program(s) yields the next functional component, Motor and Visceral Output. This behavioral output provides the feedback, via Yedasentience, that serves as a stop signal to inhibit both the activity of the Security Motivation and the Appraisal subsystems. Accordingly, without a Yedasentience output signal, the Security Motivation subsystem would continue to be active, yielding a persistent drive to perform actions related to the protection of self or others and so yielding the behavioral profile characterizing OCD. Finally, Motor Output is also postulated to have a possible effect on the Appraisal subsystem through the enhancement of Safety Cues, a slower mode of inhibitory feedback due to the hypothesized long half-life of Security Motivation once it is activated. This role of Safety Cues recognizes the coacting effects of a Safety System, as posited by Trower and his colleagues (Trower et al., 1990), which is otherwise quite separate from the Security Motivation System.

In Figure 1, the Xs on the pathways show potential sites of blockage that would yield OCD. Consider first the hypothesized blockage from Motor and Visceral Output to Yedasentience, which would interfere with negative feedback effects on the Security Motivation and Appraisal subsystems. Because of the lack of the Yedasentience signal to the Security Motivation subsystem, species-typical behaviors would fail to inhibit the subsystem's stimulation of the innate programs subserved by the basal ganglia. Thus, with no terminator for these programs, they would persist for

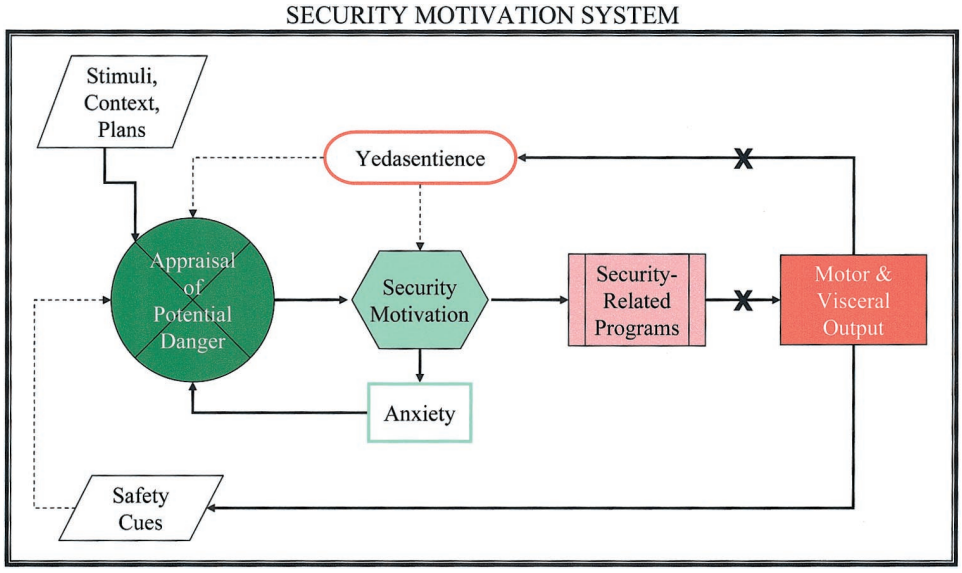

Figure 1. A conceptual model of the Security Motivation System and sites of dysfunction producing obsessive–compulsive disorder (OCD). Solid arrows indicate excitatory stimulation, and dashed arrows indicate inhibitory stimulation. Yedasentience output does not act on environmental input but rather on the Appraisal of Potential Danger and the Security Motivation processors to inhibit their activity. Exposure through motor output to safety stimuli provides inhibitory stimulation to Appraisal of Potential Danger. The Xs mark potential sites at which a blockage would yield OCD.

abnormal lengths of time. Likewise, because of the lack of the Yedasentience signal to the Appraisal subsystem, performance of species-typical behaviors would fail to dampen the sense of potential danger in the normal fashion. Thus, these behaviors would have abnormally little corrective effect on perceptions of potential danger.

Also shown in Figure 1 is a second hypothesized blockage: Security-Related Programs might fail to generate appropriate Motor and Visceral Output. This possibility would produce more generally disabling effects because of the failure to initiate the species-typical behaviors that the overall system uses as inhibitory feedback. Absent would be not only negative feedback via Yedasentience but also negative feedback via the inhibitory effect of Safety Cues on the Appraisal subsystem. We would propose that this type of blockage may correspond with the pure-obsessional type of OCD, in which compulsive behaviors are absent and which is especially resistant to treatment (Emmelkamp & Kwee, 1977; Salkovskis & Westbrook, 1989; Steketee, 1993; Steketee & Cleere, 1990; Stern, 1978).

Neurobiology of OCD

There are five kinds of findings that suggest there may be an identifiable neurological basis of OCD. First, the symptoms of OCD often appear to be associated with several neurological disorders (postencephalitis parkinsonian syndrome [Schilder, 1938], Sydenham's chorea [Swedo et al., 1989], bilateral necrosis of the globus pallidus [Laplane et al., 1989], Huntington's chorea [Cummings & Cunningham, 1992], and Gilles de la Tourette's syndrome [Pauls, Towbin, Leckman, Zahner, & Cohen, 1986]), suggesting that OCD may have a genetic and/or structural basis related to these disorders. Second, PET studies show that changes in glucose activity in the orbitofrontal cortex and caudate nucleus correlate with the presence and disappearance of OCD symptoms (Baxter, 1992; Baxter et al., 1992; Benkelfat et al., 1990; Swedo et al., 1992). Other neuroimaging studies also implicate the orbitofrontal cortex in OCD (Adler et al., 2000; Kim et al., 2001; McGuire et al., 1994; Rauch et al., 1994; Saxena, Brody, Schwartz, & Baxter, 1998). Third, OCD responds to pharmacological treatment, being ameliorated by serotonin (5HT) reuptake blockers (DeVeaugh-Geiss, 1991) and made worse by a 5HT1B receptor agonist, metachlorophenyl-piperazine (Goodman, Price, Woods, & Charney, 1991). Fourth, severe cases of OCD may be improved with psychosurgery, in particular with anterior capsulotomy or cingulotomy (Baer et al., 1995; Chiocca & Martuza, 1990; Dougherty et al., 2002; Jenike et al., 1991; Kettl & Marks, 1986). Finally, a susceptibility marker that may predispose some individuals to develop OCD has been identified (the D8/17 antigen on the surface of peripheral blood mononuclear cells); D8/17-positive individuals develop OCD as a result of their autoimmune response to Group A beta-hemolytic streptococcal infection, a response that is believed to yield antibodies that cross-react with basal-ganglia antigens and produce tissue damage (Swedo et al., 1997).[1]

Considering several of the foregoing observations, it is not surprising that the basal ganglia is prominent in all current neuroanatomical models of OCD (Baxter et al., 1992; Insel, 1992; S. Wise & Rapoport, 1989). Originally, an appreciation for its importance in OCD stemmed from insights that the basal ganglia may be a repository of innate motor programs (MacLean, 1978) and that OCD rituals may be examples of such species-typical programs (Swedo, 1989). Drawing on a conceptual organization of the basal ganglia as the nodal point of converging but segregated closed-loop circuits involving cortex–basal ganglia–thalamus–cortex pathways (Alexander, Delong, & Strick, 1986), S. Wise and Rapoport (1989) proposed that pathological activation of such circuits would produce reverberating activity and result in a persistent discharge of the innate programs characteristic of OCD. Subsequent neuroanatomical OCD models have all maintained this conceptual schema, focusing particularly on the orbitofrontal cortex as a nodal point and providing more elaborate details on the circuit diagram and its functions (Baxter et al., 1992; Insel, 1992; Modell, Mountz, Curtis, & Greden, 1989; Saxena, Bota, & Brody, 2001; Saxena & Rauch, 2000).

Despite the widespread interest in the theoretical implications of overactivity of the orbitofrontal cortex in OCD, Zald and Kim (2001) cautioned that it may be an effect of rather than the cause of OCD. They pointed out that in both OCD patients and control subjects, activity in the orbitofrontal cortex (OFC) is a normal correlate of silent ruminative thinking (Cottraux et al., 1996), and thus "increased resting OFC metabolism in OCD . . . [may simply reflect] a greater engagement of obsessive ruminations in OCD patients" (Zald & Kim, 2001, p. 61).

Neural Underpinnings of the Model

Figure 2 shows our suggestion for a neuroanatomical circuit of the proposed Security Motivation System. The circuit diagram incorporates previous proposals regarding neuroanatomical models of normal motivation and of neural dysfunction in OCD but is distinct in highlighting feedback connections from the brainstem to shut down circuit activity. Current neuroanatomical models of both motivation (e.g., Brown & Pluck, 2000; Everitt & Wolf, 2002) and OCD (e.g., Modell et al., 1989; Rauch et al., 2001) are built with functional loops involving cortico-striato-pallido-thalamo-cortical connections suggested by Alexander et al. (1986) and elaborated by others (Groenewegen, Wright, Beijer, & Voorn, 1999; Haber & Fudge, 1997; Haber, Fudge, & McFarland, 2000; Haber, Kunishio, Mizobuchi, & Lynd-Balta, 1995; Haber & McFarland, 1999; Joel & Weiner, 1994, 2000; Penney & Young, 1983). Although the motivation and the OCD circuit models originated in separate literatures, the models bear great similarity to each other. Our framework of OCD as a disturbance of normal motivation suggests that the neural convergence of the two literatures is expected and illuminating.

Our neural schema of the Security Motivation System is composed of several cascading circuits that subserve the four functional components identified in Figure 1 (middle row) and which we label correspondingly as the *Appraisal of Potential Danger Loop,* the *Security Motivation and Affect Loop,* the *Security-Related Programs Loop,* and the *Brainstem Output Network.* In addition, specific connections between these loops and inputs to them provide the Anxiety, Yedasentience, and Safety Cues signals.

Our labels for the four major circuits correspond closely to neuroanatomic systems discussed by other authors. Specifically,

[1] It is interesting in this regard to note that Gödel contracted a prolonged case of rheumatic fever at age 8 (Dawson, 1999).

the present *Security Motivation and Affect Loop* is similar to what others have called the "limbic striatum loop," and the *Security-Related Programs Loop* is similar to the "motor striatum loop" (e.g., Joel & Weiner, 2000). Likewise, what we call the *Appraisal of Potential Danger Loop* corresponds well to what are generally viewed as the "limbic cortex inputs" (e.g., Everitt & Wolf, 2002) to the limbic striatum loop. The role we assign to the *Brainstem Output Network* includes its widely recognized function as the station of the final motor pathways, but an important and novel aspect that it has in our proposed model is to provide inhibitory feedback to the foregoing circuits. In particular, whereas previous neural models of OCD have focused on reverberating activity within one of the basal ganglia loops to account for the disorder, we locate the dysfunction in a failure of the Brainstem Output Network to provide an inhibitory signal to terminate the activity of these loops.

Below we elaborate on the proposed neural schema and consider each of the four functional circuits in succession:

Appraisal of Potential Danger Loop

The kind of information processed in the Appraisal of Potential Danger Loop is no doubt quite diverse and includes not only sensory input about the current environmental conditions but also data regarding plans and future intentions. For this reason, the afferents to the Appraisal of Potential Danger Loop probably originate in most regions of the cerebral cortex. However, within the Appraisal of Potential Danger Loop itself, the computations would be relatively limited in scope, involving assessment of whether the interaction of current and intended conditions yields a deviation from a state of safety and, if so, yielding an output signal of potential danger to self or potential danger to others. Because the within-loop computations evaluate not only declarative knowledge but also the emotional valence associated with stimuli and events, we propose that the neuroanatomical substrate of the Appraisal of Potential Danger Loop consists of several interconnected limbic regions crucial for processing of motivational stimuli: the hippocampus, the amygdala, the bed nucleus of the stria terminalis, and the medial orbital prefrontal cortex (see Figure 2, dark green boxes). Although these regions are well-recognized as crucial way stations in the activation of defensive motivation and affect (e.g., LeDoux, 2002), we suggest for three reasons that they probably constitute also the gateway into security motivation.

First, an extensive literature points to the amygdala as the integrative hub in the identification of imminent threat and the activation of a defensive reaction (e.g., LeDoux, 2002). Of relevance to the argument here, the activation of a defensive reaction can be conditioned to environmental stimuli, and such learning and conditioned control are still crucially dependent on the amygdala and the indicated limbic connections (LeDoux, 2002). Considering a likely continuum from unconditioned through conditioned to potential danger, the corresponding functional circuits should be topographically adjacent to one another. Such topographic organization is consistent with, for instance, the observed "spread of allied reflexes" induced by nonspecific arousal (MacDonnell & Flynn, 1966; Szechtman, 1980; Teitelbaum, 1967, p. 64) and the notion that "different parts of . . . [an anatomically defined] continuum . . . are likely to act on information in a similar fashion, but functional shifts could emerge as a consequence of topographical variations in information that reaches this structure" (Heimer, Harlan, Alheid, Garcia, & Deolmos, 1997, p. 984).

Second, patients with damage to the ventral medial prefrontal cortex or the amygdala perform poorly on tasks that depend on the emotional evaluation of future (as opposed to immediate) outcomes (Bechara, Damasio, & Damasio, 2000; Bechara, Damasio, Damasio, & Lee, 1999; Bechara et al., 1997, 1998), a condition which the authors of that study (Bechara et al., 2000) described as a "myopia for the future" but which equally aptly can be conceptualized as a deficit in appraisal of potential danger.

Finally, the proposed loop has the requisite neuroanatomic attributes for an integrative gateway to activate security motivation. On the input side, the loop connects extensively with sensory, associative, and autonomic–affective brain areas (Rolls, 2000; Zald & Kim, 1996). Similarly, on the output side, the loop connects extensively with the ventral striatum (a key area in the proposed motivation circuit) via hippocampal, amygdalal, and medial orbital prefrontal cortex projections, as well as by virtue of the position of the medial orbital prefrontal cortex as a nodal point in both the appraisal and motivation loops (see Figure 2).

Security Motivation and Affect Loop

The neuroanatomic circuit of the proposed Security Motivation and Affect Loop is very similar to the motivational circuit described by Everitt and Wolf (2002) and implicated by those authors in mediating the addictive effects of psychostimulant abuse. A similar circuit was also identified by Brown and Pluck (2000) and labeled as the "affective" striato-thalamo-cortical component in their neural model of motivation and goal-directed behavior. That the proposed neural basis of security motivation is equivalent to other motivations should not be surprising, given that a common set of limbic regions had been implicated in every motivation (MacLean, 1985; Robbins & Everitt, 1996), and no a priori rationale exists to suggest otherwise for security motivation. Indeed, the task of a motive circuit (Kalivas & Nakamura, 1999) is generic—to sustain goal-directed activity until the object is reached and to potentiate appropriate motor and sensory responsiveness. Motivational specificity should arise by virtue of having a subset of possible neural circuits potentiated, a subset selected in the case of security motivation by the output of the Appraisal of Potential Danger Loop. Similarly, we suggest, motivation should arouse an associated affect, with the type of stimulated emotion being dependent, again, on the motivation-triggering conditions. In the case of an activated security motivation, we suggest that the associated experiential feeling is anxiety (or wariness; Masterson & Crawford, 1982) and is mediated by limbic striatum projections to the bed nucleus of the stria terminalis (see Figure 2), based on evidence of its role in anxiety-potentiated acoustic startle (Davis & Shi, 1999; Davis, Walker, & Lee, 1997; Lang, Davis, & Öhman, 2000). Chiocca and Martuza (1990) also noted the importance of the limbic system in mediating the anxiety component of the disorder; likewise, Pitman (1989) commented on similarities between OCD symptoms and compulsivelike behavior of animals with limbic system perturbation.

The Security Motivation and Affect Loop has design features of the generalized basal ganglia–thalamocortical circuit (Alexander et al., 1986). As noted by Penney and Young (1983), the structure of the cortico-striato-pallido-thalamo-cortical feedback circuit sug-

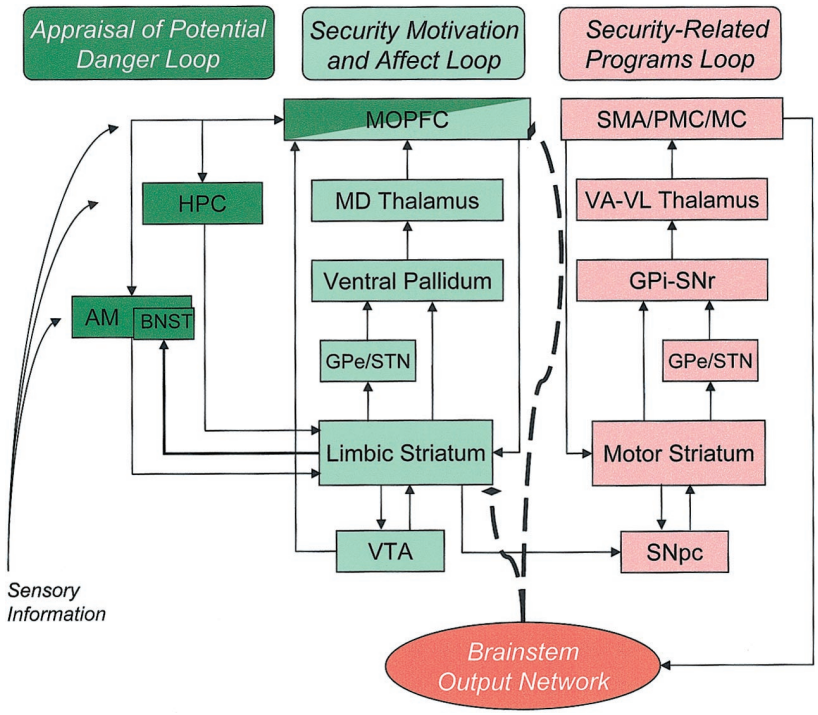

Figure 2. A neural circuit model of the Security Motivation System. Each of the four distinct subcircuits (loops) subserves one of the functional components in Figure 1 and is identified by corresponding colors. The dashed line indicates possible sites of yedasentience feedback inhibition. MOPFC = medial and orbital prefrontal cortex; SMA = supplementary motor area; PMC = premotor cortex; MC = motor cortex; HPC = hippocampus; MD Thalamus = mediodorsal thalamic nucleus; VA-VL Thalamus = ventroanterior and ventrolateral thalamic nucleus; GPi = internal segment of the globus pallidus; SNr = substantia nigra pars reticulata; AM = amygdala; BNST = bed nucleus of the stria terminalis; GPe = external segment of the globus pallidus; STN = subthalamic nucleus; VTA = ventral tegmental area; SNpc = substantia nigra pars compacta.

gests that an important functional property of the circuit is the capacity for reverberating activity, a desired property for processes that depend on prolonged and sustained activation, as is the case for motivation. Equally important, the closed-loop structure of the circuit connecting topographically related regions suggests that the sustained activation is functionally selective by virtue of potentiating a subset of neural pathways. For this reason, Penney and Young (1983) observed that the striatal node of different basal ganglia–thalamocortical circuits may be where "the basal ganglia select and maintain species-specific behaviors" (p. 79).

In a similar fashion, the present neural model considers the limbic basal ganglia–thalamocortical loop as a circuit that selects and sustains activity, although not of behavior directly but rather of motivation, which in this case is security motivation. Accordingly, in our model, the neural route to behavioral output includes a cascade of additional circuits described below.

Security-Related Programs Loop

MacLean's (1978) studies of brain mechanisms underlying innate patterns of display behaviors provided the basis for the current concept that the striatal complex does not control the generation of individual motor components but instead codes, at a more abstract level, the program by which constituent movements are organized into a species-typical pattern of behavior. In agreement, Aldridge and Berridge (1998) recently found that neurons in the dorsolateral neostriatum were activated by the overall sequential structure of grooming behavior, leading them to suggest that this neostriatal region may implement the "action syntax" of species-specific "instinctive" movement sequences. Although the evidence is good for the neostriatum as a region specialized to program or implement the action syntax of species-typical behaviors (Aldridge & Berridge, 1998; Berridge, Fentress, & Parr, 1987; Berridge & Whishaw, 1992; Cromwell & Berridge, 1996), the details of such circuitry are not known. Nonetheless, following the lead of Penney and Young (1983) and others (S. Wise & Rapoport, 1989), we posit that species-typical programs (which in our case are for security-related behaviors) are implemented in a basal ganglia–thalamocortical circuit, which, in light of the studies by Berridge and colleagues (Aldridge & Berridge, 1998; Berridge & Whishaw, 1992; Cromwell & Berridge, 1996), may be identified as the motor loop that includes the dorsal (motor) striatum.

Several neuroanatomical mechanisms have been suggested that would permit a cascade of activity from one basal ganglia–thalamocortical circuit to another (Groenewegen et al., 1999; Haber & Fudge, 1997; Haber et al., 1995, 2000; Haber & McFarland, 1999; Joel & Weiner, 1994, 2000). In our model (see Figure 2), we indicate the neural progression as proceeding via one of the series of "spiral" connections (Haber et al., 2000) that link, in a

topographically arranged pattern, midbrain dopamine neurons and the striatum. However, other possibilities should not be discounted, including intervening loops in the cascade from the limbic to the motor loop.

Brainstem Output Network

Although we are currently unable to specify the details of the brainstem anatomy involved, our neural model ascribes two important roles to the brainstem. First, and relatively obvious, the output of the basal ganglia motor programs must utilize the brainstem nuclei to produce behavioral responses. Second, and more critical for our model, the brainstem also generates crucial feedback, giving rise to yedasentience, which inhibits the security motivation and the appraisal loops.

Because yedasentience is an affective signal, it may seem strange to locate its origins in the brainstem. As Berridge (2003, p. 36) remarked, "conjunction of the words 'affective' and 'brainstem' might seem contradictory to those who hold a dogmatic view of the lower brain as merely reflexive." Nevertheless, a range of recent work strongly implicates the brainstem in the generation of emotion and feelings (Panksepp, 1998; Parvizi & Damasio, 2001). Consistent with this work, we propose for the reasons below that yedasentience is subserved by brainstem (midbrain, pons, and medulla) circuits and possibly includes ascending serotonergic projections.

Considering that security motivation is instigated by potential danger, and hence reality-based goal stimuli do not exist, what then stimulates yedasentience? We posit that activation of yedasentience is produced by performance of species-typical acts. That is to say engagement in behavior, in and of itself, is the condition that stimulates yedasentience. This idea has roots in a decades-old biological theory of reinforcement. Specifically, Glickman and Schiff (1967) were struck by their observation that without reinforcing stimuli, animals engage in investigatory behavior, and this behavior is composed of motor patterns that are characteristic of a species but vary widely from species to species. To explain what processes, in the absence of reinforcing biological stimuli, could maintain such investigatory repertoires, Glickman and Schiff proposed an evolutionary framework for reinforcement. According to them, reinforcement evolved as a mechanism that uses species-typical behavioral repertoires to manage contact with appropriate stimuli, and hence, a sufficient condition for reinforcement is the facilitation of neural pathways mediating species-typical motor behavior. Glickman and Schiff supported their thesis with evidence from brain stimulation and lesion studies that showed the same brain system mediated performance of species-typical acts and the reinforcing effects of brain stimulation. Although forebrain systems could modify the performance of the species-typical acts, Glickman and Schiff reviewed evidence from ablation studies indicating that the circuits for the elementary components of these motor repertoires are fully organized at the level of the brainstem (e.g., as shown for grooming; Berridge & Whishaw, 1992).

We follow here Glickman and Schiff (1967) and suggest that the relevant satietylike feedback is generated by a brainstem structure coincident with, or closely related to, circuits there subserving species-typical motor acts of protection of self or others, acts that are characteristic of OCD compulsions. Although the feedback signal originates in the brainstem, it is possible either that yedasentience also originates here or that the feedback signal becomes yedasentience only when security motivation is inhibited in the limbic system.

In addition, we suggest that the satietylike signals to the appraisal and security motivation loops may be conveyed from the brainstem by serotonergic pathways. The suggestion for a serotonergic pathway is based on the following four considerations.

First, there is evidence that serotonin may act as a satietylike terminator signal. For instance, sexual behavior ceases when serotonin release in the lateral hypothalamus increases (Lorrain, Riolo, Matuszewich, & Hull, 1999). Appetite and feeding are similarly reduced by enhanced serotonin activity (Blundell, 1991). Moreover, exhaustion from voluntary exercise (a focused endeavor that, like security motivation, has no external consummatory stimuli) is associated with an increase in brain serotonin (Bailey, Davis, & Ahlborn, 1993; Blomstrand, Perrett, Parry-Billings, & Newsholme, 1989; Dishman, 1997; Heyes, Garnett, & Coates, 1988), suggesting that this neurotransmitter system may provide the signal to stop such intense motor activity. Conceivably, a serotonergic pathway for the satietylike signal may reflect but a more general rule that central serotonergic neurons are involved in behavioral suppression (Soubrie, 1986) and inhibition of information flow (Spoont, 1992), effects that are often antagonistic to those of dopamine systems normally involved in facilitating active behavior (Antelman & Szechtman, 1975; Kapur & Remington, 1996; Robinson & Berridge, 1997; R. A. Wise & Bozarth, 1987).

Second, yedasentience, by virtue of shutting down the security motivation loop, removes also the anxiety output (see Figure 1), suggesting an anxiolytic effect. Serotonin may be anxiolytic in conditions associated with motivated behaviors of defense (Graeff, Viana, & Mora, 1997), and in this respect, a serotonergic feedback signal is also consistent with an expected relief-of-anxiety effect.

Third, the anatomical distribution of serotonergic neurons is consistent with projections to the cortex and the limbic system (Soubrie, 1986), as proposed in Figure 2. However, at the present time there is no strong rationale for pointing to either the dorsal or the median raphe as the more likely projection neurons (Spoont, 1992).

Finally, an inhibitory feedback signal that is serotonergic is consistent with the beneficial effects of serotonin reuptake inhibitors in OCD (DeVeaugh-Geiss, 1991) and the proposed yedasentience dysfunction in OCD.

Implications of the Model

We have shown that our proposed model of OCD as a disturbance of security motivation integrates a wide range of core features of the psychology and underlying biology of OCD. In this section, we turn to some of the more speculative but intriguing implications of this relatively novel explanatory framework.

First, it would be interesting to explore to what extent the full spectrum of OCD behaviors can be subsumed under the umbrella of a security motivation system and its organizing theme of potential threats to security. The concept of such a system implies that we are built to recognize fragments of real threats (undoubtedly elaborated through learning) and that those fragments evoke searching and checking rather than the more commonly discussed defensive responses, such as escape. Consider, for example, the

OCD behavior of a compulsion for symmetry, which may at first not seem to have much to do with potential danger. Nonetheless, if the environment is made symmetric and orderly, deviations are more readily detected, facilitating the checking for changes that may signal potential danger. That is, as long as symmetry is maintained, there is no disturbance, and things remain "the way they ought to be." Such behavior seems to parallel the way animals seek out a thorough familiarity of their home range as a major aspect of their antipredator checking behavior (Curio, 1993). A similar argument might be advanced for the compulsion to count things (which may have parallels with the ways animals keep track of all the offspring in their litter). In contrast, a distinction needs to be developed for related (and sometimes comorbid) problems of compulsivity, such as tics and Tourette's syndrome (Leckman & Riddle, 2000), which do not seem to imply a security motivation system and thus do not seem to fit OCD.

Second, the identification of OCD as the dysfunction of a security motivation system should provide promising and generative links with animal models of the disorder. Our conception of OCD implies that under some circumstances, nonhuman animals should show OCD-like, maladaptively repetitive behavior, and this indeed has been an active area of research (e.g., Dodman, Moon-Fenelli, Mertens, Pfueger, & Stein, 1997; Pitman, 1989). For example, Szechtman, Sulis, and Eilam (1998) have investigated a drug-induced model of OCD, in which chronic treatment of rats with the dopamine agonist quinpirole induces compulsive checking behavior that is partly attenuated by clomipramine. The presently proposed theory of OCD indicates specifically where to look to explain compulsive behavior—namely, dysfunction in a satiety mechanism that connects the performance of security-related behaviors as inhibitory feedback to a subsystem that generates and sustains security motivation.

Third, we may ask how our model of OCD, which focuses largely on compulsive behavior, addresses the cognitive features of OCD. To begin, note that unwanted cognitive intrusions or obsessions are part of normal experience over the lifespan (Rachman & De Silva, 1978; Salkovskis & Harrison, 1984; Thomsen, 1999), and clinical obsessions are on a continuum with these normal experiences (Salkovskis, 1985). Accordingly, our model focuses not on the origins of unwanted, intrusive thoughts but on the inability to turn them off.[2] Because of the encapsulated (Fodor, 1983) nature of the security motivation system, it is relatively isolated from corrective input from other systems, including higher cognitive processes (cf. Öhman & Mineka, 2001). As a result, OCD sufferers experience two dissociated senses of knowing—they simultaneously both know (intellectually) and don't know (emotionally)—and they find paradoxically that the latter is largely immune to the influence of the former. As Pitman (1989) observed,

> a paradox of compulsions is that the sufferer recognizes their senselessness but is powerless to do anything about them. . . . At one level, the compulsive checker "knows" that he has turned off the gas; his memory of the action is intact. At another level, however, he is plagued by doubt and does not "feel" that he's turned it off, so that he's compelled to go back to check the stove again and again. (p. 193)

Pitman (1989) interpreted this critical feature of OCD as an inability of "the memory system to curb the habit system" (p. 193). Similarly, Rauch and his colleagues (Graybiel & Rauch, 2000; Rauch et al., 1997) advanced an intriguing proposal, that in OCD there may be a defect in the implicit (automatic) learning system for which the patient tries to compensate (unsuccessfully) with intensification of the explicit (conscious) learning system. However, it may not be necessary to propose any deficit in learning mechanisms in OCD. Instead, according to the present model, the underlying problem is lack of closure—the inability to turn off security motivation, which drives security-related thoughts, through the normal route of performing specific security-related behaviors. The predicament for the OCD patient is a deceptively counterintuitive one: Problems in thought cannot readily be corrected through more thought (higher cognitive processes), even with great effort. This is because, in the terminology of the present model, yedasentience is not an output of volitionally directed higher cognition; instead, it normally stems from enacted motor behavior.

Thus, although problems in learning and thought develop in OCD, we propose that they are secondary elaborations of this primary deficit. In addition, such problems should not develop in domains that are unrelated to potential danger—no one obsesses about the possibility of making someone happy. However, although secondary, these cognitive problems are not trivial. For sufferers of OCD, their peculiar thoughts and actions undoubtedly constitute what Zimbardo (1999) has termed "discontinuities" in experience, and as Zimbardo has amply demonstrated, these discontinuities drive processes of explanation that may seriously exacerbate the person's difficulties (see also Jacobs & Nadel, 1999, for similar ideas applied to panic disorder).

Another perspective on these secondary cognitive difficulties is suggested by Damasio's (1994) ideas about the crucial role of feeling in decision making and the limits of rationality as a compensation. For example, he described a frontal patient who, when asked to schedule a next visit, spent half an hour carefully searching out and weighing all logical possibilities before Damasio finally stopped him. Rather than viewing this flagrant obsessiveness as a primary deficit, however, Damasio argued that it represented the failure of feeling to inform implicit, automated mechanisms of decision making and the inevitable limits of pure reason as a substitute. In a similar vein, we propose that obsessiveness in OCD may partly represent the attempt to use rationality to compensate for the dysfunction of a more primary, automatic mechanism—namely, yedasentience-mediated inhibitory feedback on security motivation, normally resulting from the simple execution of security-related behaviors. Simply put, to some extent OCD sufferers may appear to think too much, because behavior, and the feelings it would normally have evoked, does not work for them. In this connection, we have already suggested that pure-obsessional patients experience no yedasentience from security-related behaviors at all; this complete absence, together with the problem-compounding nature of a purely rational computative substitute strategy, may explain why they are particularly resistant to treatment, as mentioned earlier.

Fourth, we can very briefly sketch some implications of our model for psychological treatment strategies. To begin, why would

[2] Dysfunction in the start mechanism for such thoughts would seem to relate to generalized anxiety disorder; in contrast, our proposal is that OCD has to do with dysfunction in the stop mechanism.

exposure therapy work for OCD? According to the present model, such exposure works by reducing the sensitivity of the system to stimuli—essentially, stimulus devaluation (cf. Marks & Nesse, 1994). This extinction helps to prevent the security motivation system from getting turned on, so that its inability to turn off normally in OCD becomes less of a problem. Indeed, after successful exposure therapy, the former OCD patient may well have a subnormal threshold of response to certain cues of potential danger, because his or her original condition was one of underactive stopping rather than overactive starting.

Far more speculatively, the model may suggest the possibility of other treatment strategies. For example, ritualistically elaborating or increasing the difficulty of the security-related behavior may increase its capacity to produce yedasentience and help terminate the motivational state, shortening the duration of the behavior. This strategy is somewhat akin to "ordeal therapy" (Haley, 1984). Likewise, we may ask if it is possible to substitute another feeling (e.g., another somatic marker) for the missing feeling of yedasentience, or is the system too encapsulated to allow this?

Finally, we turn our attention briefly to considering what sort of future data would support the model and what sort would refute it. The model would be supported by data showing that individuals with OCD, compared with controls (such as patients with other anxiety disorders), have a problem with stopping or satiation rather than initial sensitivity or motivation. For example, they should not work harder for access to the relevant security-related stimuli (e.g., water for washing and access for checking). Such findings would parallel those for other dysfunctional satiety mechanisms: For instance, rats with ventromedial hypothalamic lesions become fat not because of heightened motivation to eat—they do not, in fact, work harder to obtain food—but because of loss of the satiety mechanism (Whalen & Simon, 1984). In contrast, our model would be clearly refuted by the demonstration that there is no encapsulated security motivation system, or by the inability to find physical structures unique to this system. If there is no security motivation system, then OCD cannot be a disturbance of its functioning.

Conclusion

Considering OCD as a disturbance of security motivation builds on many previous observations about the disorder but casts them in a new light by reinterpreting them within the broader understanding of processes of normal motivation. Much previous work on OCD has asked, in essence, what kinds of defective reasoning could produce this baffling pattern of behavior. Although not denying a role for higher cognitive factors, our proposed model draws on a range of ideas from biological psychiatry and the psychology of motivation to locate the core deficit elsewhere. The inability to put closure on experience in OCD is not so much a problem in higher cognition as a deficit in its emotional underpinnings: In the OCD patient, the performance of security-related behaviors fails to generate the feeling state that would normally shut down security motivation. The resulting abnormally persistent motivation undoubtedly drives various cognitive elaborations, but we propose that these are secondary to a more basic deficit in knowing. Moreover, this deficit in emotion-based knowing has a plausible neuropsychological basis, which builds on previous proposals about the biological underpinnings of OCD.

Drawing attention to the epistemic nature of OCD, S. Wise and Rapoport (1989) characterized OCD patients as being in a Berkeleian "nightmare," disbelieving their senses and thus "tied to their immediate sensory systems, needing continuous reaffirmation that their hands are clean, the door is locked, and so on" (p. 341). Although we strongly concur with the idea that OCD has an epistemic origin, the present model implies that the OCD patient's inability to believe should be characterized somewhat differently. Rather than doubting their senses, OCD patients essentially doubt their own behavior. They disbelieve their behavior because it fails to produce the normal internal feedback that should have released them from the grip of activated security motivation. In addition, because of the open-ended nature of the security motivation system, which disconnects it from immediate environmental control, there is indeed a Berkeleian predicament for OCD patients: They cannot look to the environment to compensate for the terminator that they cannot generate endogenously. Thus, their recourse is to repeat the behavior over and over, in an attempt to overcome a dysfunctional feedback mechanism and eventually dampen the driving motivation.

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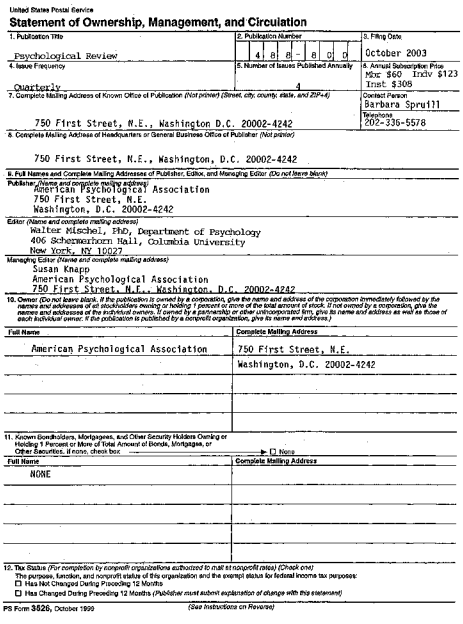

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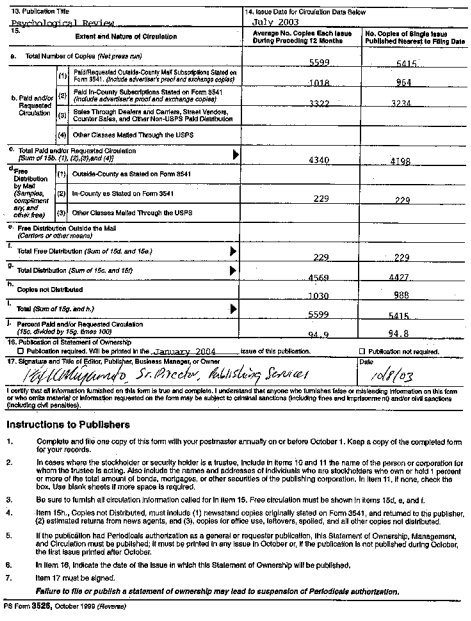

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