

Using Hypnosis to Develop and Test Models of Psychopathology

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We illustrate some of the unique ways in which hypnotic suggestions can be used to develop and test models of psychopathology. In particular, we describe a study of hypnotic hallucinations, focusing on its value for probing the underlying nature of hallucinations, including their neural underpinnings. We discuss how this work led us to view OCD behaviour as having qualities opposite in some ways to those of hallucinations—an insight that subsequently led to the development of a new theory of OCD, which we briefly review. Finally, we describe an experimental test of the plausibility of this theory using appropriate hypnotic suggestions.

INTRODUCTION

Hypnosis has been used instrumentally to develop and test models of a number of psychopathologies. In the 1960s, Reyher (1961, 1962) used hypnosis to experimentally evaluate the role of psychodynamic conflict and thereby induce analogs of psychopathology. Periodically over the years, hypnosis has been employed as a model for delusions (e.g., Kihlstrom & Hoyt, 1988; McConkey, Szeps, & Barnier, 2001), functional blindness (e.g., Bryant & McConkey, 1989, 1999; Sackheim, Nordlie & Gur, 1979), and functional memory disorders (e.g., Barnier, 2002; Kihlstrom & Schacter, 1995). Zimbardo used hypnosis to evaluate a model of paranoia (Zimbardo, Anderson, & Kabat, 1981). Recently, Oakley has used hypnosis to model functional paralysis (Halligan, Athwal, Oakley, & Frackowiak, 2000; Oakley, 1999, 2006), and Barnier and her colleagues have used hypnosis to model identity and misidentification delusions (Barnier et al., 2008; Cox & Barnier, 2009). Nonetheless, the instrumental use of hypnosis as an empirical approach to model psychopathology is hardly common, and so it is worthwhile to begin by considering the question: *Why use hypnosis for this purpose?*

One reason is that hypnotic phenomena have qualities that closely resemble some symptoms of psychopathology. Appropriate suggestions in highly hypnotizable participants produce, to use John Kihlstrom's words, "conviction bordering on delusion" and "involuntariness bordering on compulsion" (Kihlstrom, 2008, p. 21). In addition, by using hypnosis we can produce the

phenomena of interest on demand. Rather than waiting for a patient to spontaneously experience a hallucination, we can generate one in the lab and study, for example, its associated brain activity (Oakley & Halligan, 2009). Finally and most importantly, by using hypnosis we can independently manipulate factors of putative importance to theories of psychopathology, thus exploring whether these factors have the role that a theory says they do.

To illustrate these themes, we will now highlight some of our own work over the last decade. In this work, our use of hypnosis has evolved from modeling hallucinations to modeling compulsions, even as our thinking has interconnected the two conditions.

STUDY OF AUDITORY HALLUCINATIONS VIA NEUROIMAGING

Highly hypnotizable participants undergoing the experience of a hallucination have a compelling experience of the reality of a given perception, even though the perceived stimulus is actually not present in the outside world. Some of our work has addressed the issue of how such experiences can occur, and, more specifically, which brain mechanisms underlie such false convictions of reality. This work also provides insights into the processes that normally provide the foundation for our sense of external reality, which may break down in some kinds of psychopathology.

With Ken Bowers and Claude Nahmias some years ago, we addressed these issues by conducting a positron emission tomography (PET) study

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of hypnotic auditory hallucinations (Szechtman, Woody, Bowers, & Nahmias, 1998). Our rationale for using hypnosis in this experiment was instrumental: we wanted to use hypnosis to facilitate the study of the nature of hallucinations, rather than focusing on the nature of hypnosis itself. Thus, our design compared two groups of highly hypnotizable participants: (1) carefully selected individuals shown to be consistently capable of producing vivid auditory hallucinations, and (2) individuals who were equally high in overall hypnotizability, but shown to be incapable of producing such hallucinations.

After prescreening all participants for high hypnotizability on the Harvard Group Scale of Hypnotic Ability, Form A (Shor & Orne, 1962) and the Waterloo-Group C Scale of Hypnotic Susceptibility (WSGC; Bowers, 1993, 1998), we evaluated their capacities to experience vivid hallucinations in individual clinical assessments. Each assessment included a variety of hypnotically suggested hallucinations, such as music, voices, and singing. Hallucinators and non-hallucinators differed not only during this assessment, but on two other occasions, i.e., whether or not they passed the auditory hallucination item during the WSGC; and whether or not they hallucinated during the PET study itself.

It is important to point out that this type of design is different from the far more typical one that compares highly hypnotizable participants with their low-hypnotizable counterparts. There are considerable individual differences in ability profiles among highly hypnotizable individuals,

consistent with hypnotizability being a multidimensional characteristic (Laurence, Beaulieu-Prévost, & du Chéné, 2008; Woody & Barnier, 2008; Woody, Barnier, & McConkey, 2005). Although the underlying causes for this multidimensionality are not yet known, its implication for instrumental studies of uncommon phenomena like hallucinations is very clear: it is an error to consider all high-hypnotizable participants to be equivalent. If we want to study hallucinations, we need to make sure we find a group of high-hypnotizable participants who can actually hallucinate (rather than, for example, simply imagine vividly). In addition, in accordance with experimental logic, the best control group for these hallucinators would be participants who are as much like them as possible in all other respects. This group comprises equally high-hypnotizable participants who cannot hallucinate (rather than low-hypnotizable participants).

Our experiment was designed to incorporate particular theory-based, within-subject comparisons. Our theoretical framework derived from Bentall (1990), who conceptualized hallucinations as internally generated events that are misattributed to an external source. Accordingly, we asked the following question, *Which brain regions are involved in distinguishing whether an auditory perception comes from the external world or not?* For this purpose, we measured changes in regional cerebral blood flow (rCBF) under four conditions: (1) while resting; (2) while hearing a recorded message; (3) while imagining the same message; and (4) while hallucinating the message. With this design, we can answer the following question: *How is brain activation during hallucination like hearing, but different from imagining (and baseline resting)?* [Note that rather than trying to reduce a hypnotic phenomenon to an act of imagination, as in Spanos' (1991) well-known work from a social-cognitive perspective, we were explicitly trying to contrast the two.]

Following a brief hypnotic induction, each participant was given the four conditions twice, the second time in reverse order. In the *hearing* condition, participants were informed that a tape recorder would be turned on, and they were asked to pay attention to a recorded message. This message consisted of the phrase, "The man did not speak often, but when he did, it was worth hearing what he had to say," repeated several times. In the *imagining* condition, participants were informed the tape recorder would not be turned on, and they were asked to imagine the same man's voice repeating the same phrase over and over, as vividly as possible. Finally, in the *hallucination* condition, participants were administered exactly the same instructions as for the *hearing*

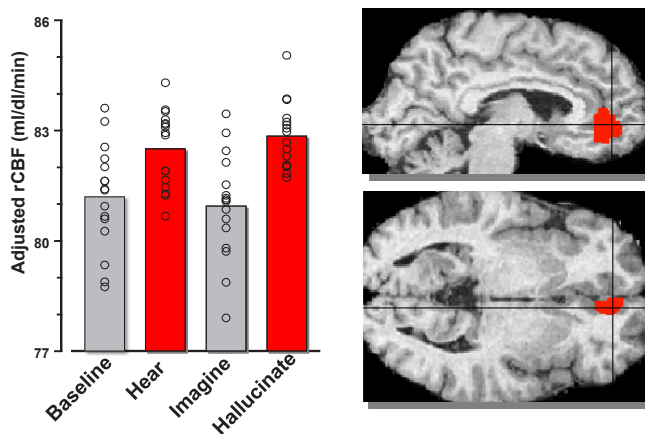


FIGURE 1. Right ventral anterior cingulate region activated in hallucinators by both hearing and hallucinating compared to both imagining and baseline conditions. Right panel: Projections of this region on sagittal (upper) and transverse (lower) MRI templates, with crosshairs at the maximum voxel ($z = 4.60$) located at the coordinates $\{6,48,0\}$ according to the Talairach atlas (Talairach & Tournoux, 1988); the threshold for significant voxels was set at $p = 0.001$ and the indicated region is comprised of 207 voxels. Left panel: The adjusted rCBF response at $\{6,48,0\}$ for each experimental condition (averaged across the two replications). Reprinted with permission from Szechtman et al. (1998).

condition—namely, that the tape recorder would be turned on, and they should pay attention to the recorded message. But after the initial sound of the tape recorder being turned on, no message was played. There were no further suggestions in this condition, and participants were not told that it had anything to do with hallucination. Following each trial, participants rated the externality of the sound (from 1, “entirely inside their own head,” to 10, “entirely external”) and its clarity (from 1, “virtually inaudible,” to 10, “clear and vivid—as real as real”).

For the hallucinators, the re-experience of the taped message was apparently very compelling (even though there was no sound). In the debriefing, it proved impossible to convince some of them that the message had not actually been replayed. In contrast, some of the non-hallucinators were kind enough to let us know that there might possibly have been a problem with our tape recorder. Because there has been so much emphasis in some circles on the role of expectancies in hypnotic responding (e.g., Benham, Woody, Wilson, & Nash, 2006; Lynn, Kirsch, & Hallquist, 2008), it is interesting to note that both hallucinators and non-hallucinators fully expected to hear the taped message again, but only the hallucinators managed to hear it.

The method of Statistical Parametric Mapping (Friston, Frith, Liddle, & Frackowiak, 1991) enabled us to look for brain locations activated during hearing and hallucination but not during imagining and baseline. For the eight hallucinators, this four-fold contrast yielded the region of the right anterior cingulate (Brodmann area 32), as depicted in the right panel of Figure 1.

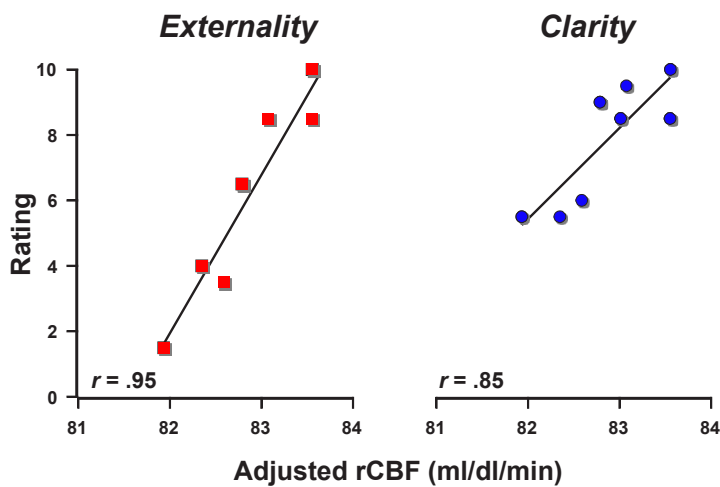


FIGURE 2. Association between adjusted rCBF response at {6,48,0} and ratings of externality and clarity of the voice in the hallucinating condition, $p < 0.05$. The adjusted rCBF and the ratings are both averaged for each participant across the two trials of the hallucination condition. Reprinted with permission from Szechtman et al. (1998).

As shown in the left panel of this figure, during hallucination the adjusted blood flow (rCBF) in this region was as elevated above baseline as it was during *hearing*, whereas during *imagining* there was no such elevation.

Furthermore, as shown in Figure 2, rCBF response in this region during *hallucination* was very strongly related to hallucinators’ subjective ratings of externality ($r = .95$) and clarity ($r = .85$).

In contrast, for the six non-hallucinators, the same comparison of *hearing* and *hallucination* conditions with imaging and baseline revealed no significant activation in the right anterior cingulate. The difference between the hallucinators and the non-hallucinators at this site was statistically significant, which implies that activation of the right anterior cingulate is crucially implicated in the experience of a hallucination. For the non-hallucinators, the fourfold contrast did yield significant activation in one region, (the auditory association cortex: right Brodmann area 22), and the non-hallucinators also differed significantly from the hallucinators at this site. This difference between the two groups implies that activation of the auditory cortex alone is not sufficient for a hallucination.

The results of some comparisons between pairs of conditions helped flesh out these differences. The comparison of *hearing* to *hallucination* conditions showed activation over a much wider area of the temporal lobes in the hallucinators than in the non-hallucinators. Thus, with regard to the auditory cortex, the hallucination task was less similar to hearing for the hallucinators than for the non-hallucinators. In addition, the comparison of *hearing* to *baseline* showed significant differences in the brain activation of hallucinators and non-hallucinators. *Hearing* activated more extensive regions (including the auditory cortex and the right rostral anterior cingulate) for the hallucinators than for the non-hallucinators. In this regard, it is interesting to note that the pattern of brain activity during *hearing* shown by non-hallucinators was essentially the same as that shown by unselected, un hypnotized participants (Szechtman et al., 1992). Thus, hallucinators appear to show an unusual tendency to process auditory events in more extensive brain regions than other people. This tendency is consistent with the research on individuals with “fantasy-prone personalities” (Wilson & Barber, 1983; Lynn & Rhue, 1988), who are highly hypnotizable, hallucinate readily, and show high sensitivity to sensory stimuli.

The main result of this study, implicating the anterior cingulate in the experience of a hallucination, fits intriguingly with a variety of other research findings. Hallucinations in schizophrenic

patients appear to be associated with activity in the anterior cingulate, although perhaps somewhat more dorsally than in our hypnotic hallucinators (Cleghorn et al., 1990, 1992; Silbersweig et al., 1995). In addition, surgical lesions in the anterior cingulate can produce faulty reality monitoring, in which imagined events are believed to be real (Whitty & Lewin, 1957). Finally, the critical role of the anterior cingulate in our study is consistent with many other findings concerning the neural mechanisms underlying hypnotic effects (Oakley, 2008). For example, neuroimaging studies of hypnotic analgesia implicate the anterior cingulate (e.g., Crawford, Gur, Skolnick, Gur, & Benson, 1993; Crawford et al., 1998; Rainville, Duncan, Price, Carrier, & Bushnell, 1997).

But what can activation of the anterior cingulate tell us specifically about the underlying nature of hallucinations? The anterior cingulate serves as a critical interface between emotional and attentional systems inasmuch as it is closely associated with emotional experience and regulation—especially the assessment of the motivational significance of stimuli (Damasio, 1997; Devinsky, Morrell, & Vogt, 1995). It is also an essential component of the anterior attentional system (Posner & Petersen, 1990; Morecraft, Geula, & Mesulam, 1993). Thus, the interconnection between emotion and attention seems strongly implicated.

The line of interpretation we favour links our research to ideas that Rapoport (1989, p. 240) has termed the “biology of knowing” and Damasio (1994, p. 196) the “neurobiology of rationality.” Similar to these researchers, we proposed that what may be necessary for a convincing subjective sense of the reality of some perceptions is a crucial “feeling of knowing.” By affecting such feelings of knowing, hypnosis may produce a dissociation between subjective convictions and reality (Woody & Szechtman, 2007).

Cognitive psychologists have used the term “feeling of knowing” to designate the intuitive sense of knowing something, even though at the moment it cannot be brought to mind (e.g., Nelson, Gerler, & Narens, 1984). More generally, what one “knows” *subjectively* may be quite distinct from what one knows *objectively*—for example, one continues to experience the subjective conviction that one of the two lines in the Müller-Lyer illusion is obviously longer than the other, even as one verifies objectively that they have the same length.

One effect of hypnosis is to foster such subjective feelings of knowing, and these subjective convictions may then serve as the kernel around which plausible content is readily elaborated. Thus, with regard to a hypnotic hallucination, once the conviction arises that something is out

there, the situation provides very clear expectations (i.e., the taped message) through which the accompanying percept may be constructed.

Other hypnotic phenomena may also readily arise from alterations in feelings of knowing. In particular, negative hallucinations can be explained as stemming from the *absence* of a feeling of knowing. To illustrate, in one study (Spanos, Flynn, & Gabora, 1989), highly hypnotizable participants were given the suggestion that they would see only a black sheet of paper and then shown a clear “8.” Some participants reported seeing nothing on the paper, but when later pressed by a second experimenter almost all of them correctly guessed what the number was. Spanos (1991) interpreted these results in terms of compliance in the service of playing a social role, whereby these participants were being “less than forthright” (p. 337) in their initial claim of not seeing anything. Nonetheless, an alternative interpretation related to hypnotically altered feelings of knowing is that these participants’ behaviour was fully consistent with *not feeling as if they knew*. Thus, hypnosis may provide particular access to the subjective, rather than rational, underpinnings of mental processes.

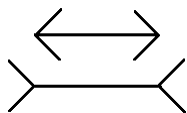
If we regard alteration in feelings of knowing as a general explanation for hypnotic responses, an interesting question is what the non-hallucinators in our study (who were equally high in overall hypnotizability) may lack, so that they do not hallucinate. Because our study was about hallucinations, rather than hypnotizability per se, it did not directly address this question. Nonetheless, one possibility is that there are distinct classes of feelings of knowing, and highly hypnotizable individuals differ in their respective propensities to alter these different types of feeling (Woody & Szechtman, 2000). For instance, the crucial feeling in hypnotic motor responses, which is involuntariness, is quite different from the crucial feeling in a hypnotic hallucination, which is the sense of external reality (see also Woody & McConkey, 2003).

THE EMOTIVE UNDERPINNINGS OF REALITY

It has long been pointed out that such subjective intuitions about reality may have a crucial role in mental life. For example, Jaspers (1963, p. 93–94) made the following observation:

Conceptual reality carries conviction only if a kind of presence is experienced. . . . Our attention gets drawn to it because it can be disturbed pathologically and so we appreciate that it exists.

MÜLLER-LYER ILLUSION



The lower horizontal line looks subjectively longer than the upper one, even after one has verified with a ruler that they are the same length.

YEDASENTIENCE
 From the Hebrew *yeda*, knowing, and Latin *sentire*, to feel; an internally generated feeling of knowing

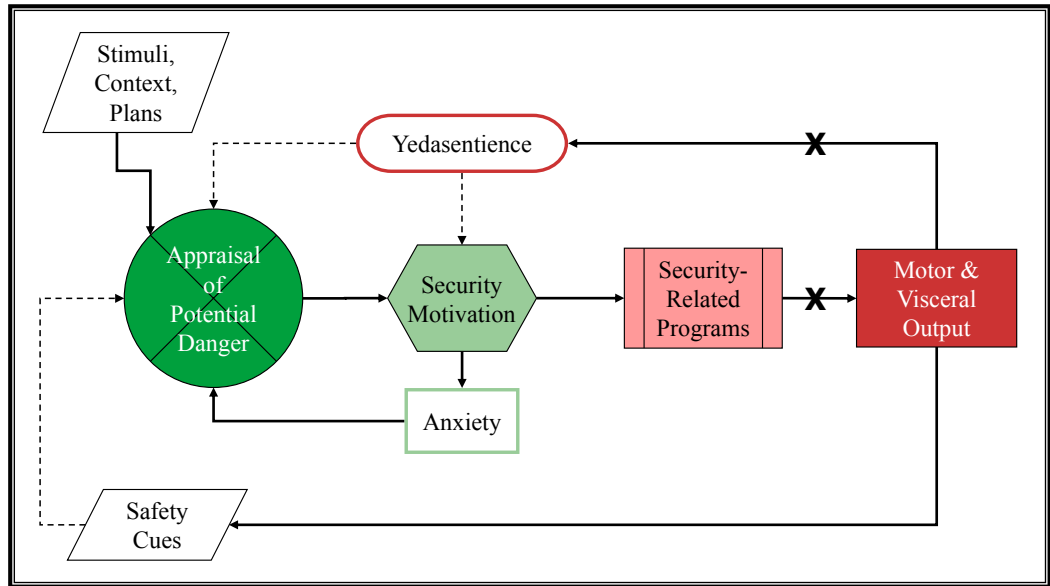


FIGURE 3. A conceptual model of the Security Motivation System and sites of dysfunction producing OCD. Solid arrows indicate excitatory and dashed arrows inhibitory stimulation, respectively. Yedasentience output does not act on environmental input but rather on the Appraisal of Potential Danger and the Security Motivation components to inhibit their activity. Exposure through motor output to Safety Cues also provides inhibitory stimulation to Appraisal of Potential Danger. Reprinted with permission from Szechtman & Woody (2004).

Similarly, William James 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 opposites 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. (James. 1890, p. 283–284, italics in the original)

Consistent with James’ opposition of the feeling of belief versus the feeling of doubt and inquiry, we were particularly intrigued with the idea that obsessive-compulsive disorder (OCD) has a striking similarity to a negative hallucination. Specifically, Rapoport (1989, p. 96) has conceptualized OCD as a condition in which crucial feelings of knowing are *absent*:

A major feature, and a strange one, of Obsessive-Compulsive Disorder is the inability to be reassured by the senses. Obsessives have lost their ability to “know” certain simple things that we all take for granted. [These patients] are asking “How do you know” about things that we find ourselves hard put to explain: Is the grass really green? Are my eyes blue? “Why,” I say, “we just *know*, that’s all.”

Indeed, difficulty in accepting the simplest observations led the French to call OCD the “doubting disease.” Rapoport argued that OCD patients’ perceptions, although objectively sound, are not accompanied by the subjective convictions that would normally be present:

[They] appear to say “knowledge comes from the senses only”; therefore, the test of . . . truth will be in what they can hear, see, and touch. Hence 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.” (Rapoport, 1989, p. 238)

To summarize, in hallucination, the external stimulus is absent, yet the feeling of knowing is present; whereas in OCD, the external stimulus is present, yet the feeling of knowing is absent. That is, despite sensory evidence that most people would find logically compelling, the OCD patient is not convinced because, we argue, he or she cannot generate a feeling of knowing. (For a discussion of how the absence of a feeling of knowing may also explain other psychopathologies, such as Capgras delusion, see Woody & Szechtman, 2000.)

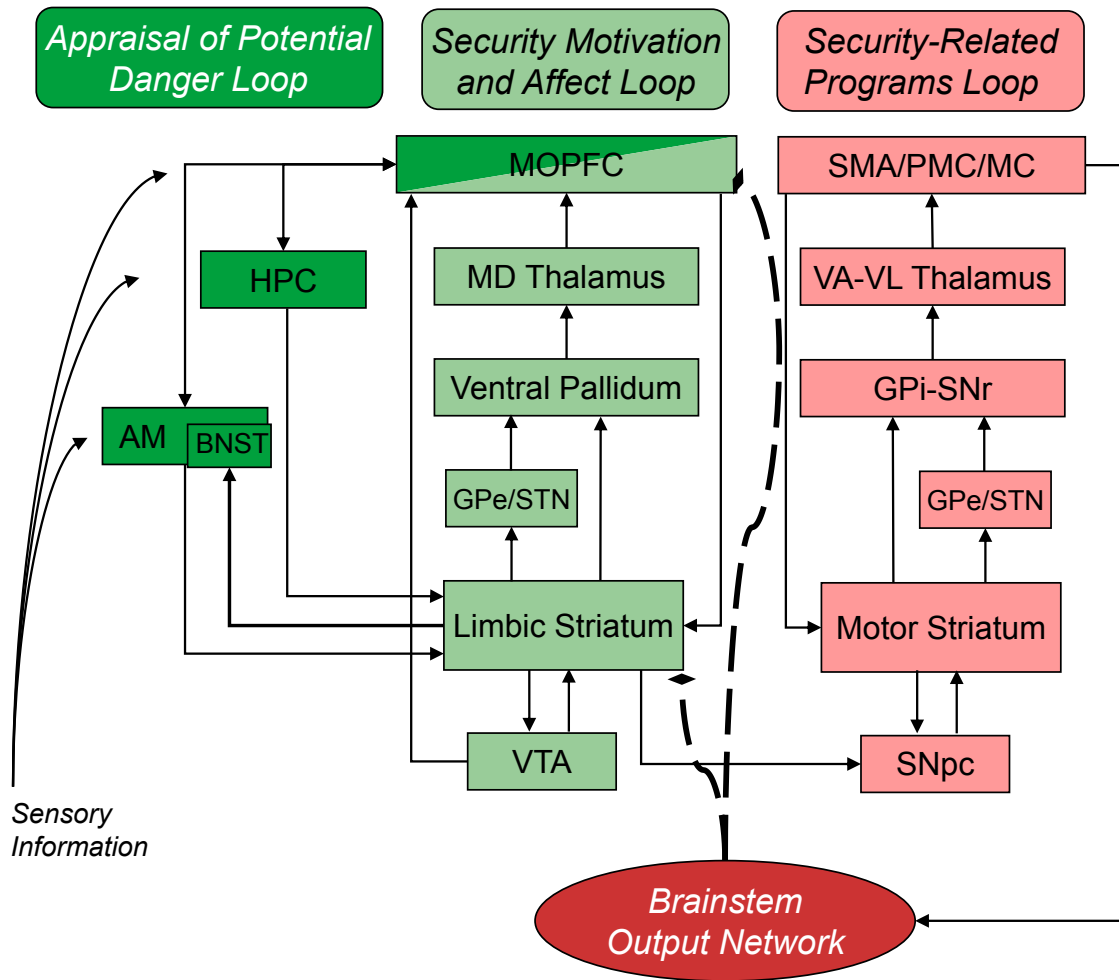
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A NEW THEORY OF OCD

The power of subjective, feeling-based knowing to overrule rational, logical knowing is dramatically evident in the lives of some people who, ironically, are pre-eminent for their rational achievements. Kurt Gödel, the eminent 20th century mathematician, sought to live life as a “quest for rationality in all things” (Dawson, Jr., 1999, p. 76). In his revolutionary “incompleteness” theorem, he used masterful logic to reach conclusions that astounded his contemporaries. Yet in his personal life, this brilliant logician was plagued with senseless obsessions about the possibility of being accidentally poisoned by something in his food or by gasses coming out of his refrigerator. To coax him to eat, his wife had first to taste his food herself. When she became ill and unable to continue in this role, his obsessions were so severe that he inadvertently starved himself to death.

Another interesting example is Samuel Johnson, the 18th century writer and outstanding Enlightenment figure, whose life’s work was “in defense of reason against the wiles of unchecked fancy and emotion” (Mahoney, 2000). Nonetheless, his personal life was full of inexplicable compulsions, such as needing to touch every post in a street or step exactly in the centre of every paving-stone. If one of these senseless acts had not been done exactly right, his friends had to wait while he went back to fix it (Stephen, 1900).

Such obsessions and compulsions can occur in a variety of disorders, but they are the hallmark of OCD. Partly as an outgrowth of our reflections on the foregoing work with hypnotic hallucinations, we were led to advance a new theory about the psychological processes that underlie OCD (Szechtman & Woody, 2004; Woody & Szechtman, 2005). In brief, we hypothesized that



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FIGURE 4. A neural circuit model of Security Motivation System. Each of the 4 distinct subcircuits (loops) subserves one of the functional components in Figure 3 and is identified by corresponding colour. The dashed line indicates possible sites of yedasentience feedback inhibition. Abbreviations: AM, amygdala; BNST, bed nucleus of the stria terminalis; GPe = external segment of the globus pallidus; GPi, internal segment of the globus pallidus; HPC, hippocampus; MC, motor cortex; MD, thalamus, mediodorsal thalamic nucleus; MOPFC, medial prefrontal cortex and orbital prefrontal cortex; PMC, premotor cortex; SMA, supplementary motor area; SNpc, substantia nigra pars compacta; SNr, substantia nigra pars reticulata; STN, subthalamic nucleus; VA, ventroanterior thalamic nucleus; VL, ventrolateral thalamic nucleus; VTA, ventral tegmental area. Reprinted with permission from Szechtman & Woody (2004).

such symptoms are caused by the inability to generate the normal “feeling of knowing” that would otherwise signal task completion. For example, even though compulsive hand washers know objectively that their hands look clean, they cannot readily generate the subjective conviction that they are truly clean, and so they continue to wash.

More specifically, we proposed that the symptoms of OCD stem from the dysfunction of a biologically primal security motivation system. Activation of this system, which originally evolved to manage protection from potential harm to self and others (Szechtman & Woody, 2004; Woody & Szechtman, 2011), gives rise to OCD concerns and behaviours, such as cleanliness and washing. In addition, the security motivation system differs from most other motivational systems in that it is open-ended, with no terminating signal in the environment. Instead, the engagement in security-related activities in themselves normally generates the terminator signal, which is experienced as a feeling of knowing. For example, when people wash their hands in response to potential danger of contamination, what normally terminates this activity is not that their hand washing has met certain logical, objective criteria; instead, they stop when it feels right and done. However, OCD patients have a crucially reduced capacity to generate this endogenous emotive terminator.

In short, what is missing for an OCD patient is a particular feeling of knowing that serves as an essential terminator of a species-specific motivation concerned with protection from potential danger. We gave this specific emotive signal the name *yedasentience*, from the Hebrew *yeda*, knowing, and Latin *sentire*, to feel, and stated our core hypothesis 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 (Szechtman & Woody, 2004, p. 116).

Figure 3 lays out our conceptual model of OCD as a dysfunction of security motivation, consisting of 4 major functional components (shown across the middle of the figure) and 3 major routes of feedback (shown above and below).

The first component, *Appraisal of Potential Danger*, evaluates environmental stimuli in the context of the organism’s plans. If results indicate a potential threat to self or others, an excitatory signal is passed to the second component,

Security Motivation, which generates a set of coordinated outputs that serve to energize and focus the person’s actions. One of these outputs, labeled *Anxiety*, provides positive feedback to the *Appraisal* component. This feedback signal provides a further interoceptive cue indicating potential danger and forms part of a loop that acts to sustain the appraisal. Another output is an excitatory signal to a third major component, *Security Related Programs*, which co-ordinates and executes species-typical motor and cognitive programs for protection from potential danger such as washing or checking. Performance of these activated programs generates the next functional component, *Motor and Visceral Output*. This output provides the feedback, via *Yedasentience*, which is the stop signal that shuts down the activity of the *Security Motivation* and the *Appraisal* subsystems. *Motor Output* also has a possible effect on the *Appraisal* subsystem through the enhancement of *Safety Cues*, a slower mode of inhibitory feedback due to the protracted half-life of *Security Motivation* once it is activated.

In the figure, the X’s on the paths show potential sites of blockage that would interfere with the feedback of the *Yedasentience* signal to the *Security Motivation* subsystem. Without the *Yedasentience* feedback, species typical behaviours such as washing and checking would fail to inhibit the subsystem’s stimulation of security-related programs, and thus these programs would persist for abnormal lengths of time. Likewise, because of the lack of the *Yedasentience* signal to the *Appraisal* subsystem, performance of species typical behaviours would have an abnormally weak corrective effect on perceptions of potential danger. Thus, the blockages indicated by the X’s would yield the behavioural profile of OCD.

Figure 4 shows our proposal for a neuroanatomical circuit of the security motivation system. It consists of cascading circuits that subserve the four functional components, labeled correspondingly as the *Appraisal of Potential Danger Loop*, *Security Motivation and Affect Loop*, *Security Related Programs Loop*, and *Brainstem Output Network*. Although most of the details of this neural model are not central to the present article, the model does usefully illustrate how our theory can be integrated with a wide range of work in behavioural neuroscience (see Woody & Szechtman, 2011, for further information).

Given the crucial role of the anterior cingulate in our hypnotic hallucinations experiment and the theme of feelings of knowing to link hallucinations and OCD, it is worth noting that in Figure 4 the anterior cingulate is included in the *Appraisal of Potential Danger Loop*, as part of the medial prefrontal cortex and orbital prefrontal

cortex (MOPFC). Moreover, Fiddick (2011) has reviewed a wide range of research to argue that the anterior cingulate plays a primary role in the assessment of potential threat (as distinguished from immediate hazards). With regard to OCD more specifically, one intriguing line of evidence is that cingulotomy has been used as an effective treatment for otherwise untreatable, severe OCD (Dougherty et al., 2002).

To summarize briefly, our theory posits that individuals with OCD suffer from the inability to turn off security motivation through the normal route of performing specific security related behaviours. These patients cannot readily generate the crucial emotional sense of knowing that, for example, their hands are clean, even though they may know this intellectually.

Because we approached OCD from a very different starting point than other psychopathologists, our theory is quite distinct from previous theories of OCD in several major respects. In particular, it proposes the existence of a special motivational system focusing on management of potential danger; it emphasizes the role of preventative behaviour and the primal feelings normally engendered by such behaviour; and its core hypothesis about OCD is a deficiency in the ability to terminate an activated state of security motivation through engagement in security-related behaviour. Other theories of OCD tend to focus mainly on cognitions as causes of the disorder, and they tend to focus more on the hypothesis that OCD represents a starting problem, in which individuals are too sensitive to particular cues (e.g., Purdon & Clark, 1999; Rachman, 1997).

STUDY OF THE SECURITY MOTIVATION THEORY USING HYPNOSIS

Recently, we have been engaged in several different approaches to testing this theory. One of them is of particular interest in the present context because it involves the instrumental use of hypnosis (Woody, Lewis, Snider, Grant, Kamath, & Szechtman, 2005). In this study, we wanted to evaluate the question of whether dysfunction of a feeling of knowing is a plausible mechanism for OCD behaviour. Our approach was to block this feeling hypnotically and see if the blockage produces OCD-like behaviour—specifically, prolonged washing. In other words, can we temporarily create in non-patient individuals OCD-like behaviour?

As an aside, we had some difficulty in obtaining ethical approval for this study. The ethics board had two concerns: first, they thought the study would not work; and second, they worried that we might produce lasting OCD in some of our participants. Both of these concerns proved to be incorrect.

In this experiment, we compared high hypnotizable participants, who had scored 9 or above on the Waterloo–Stanford Group C Scale (Bowers, 1993, 1998), to low hypnotizable counterparts, who had scored 3 or less. Because blocking changes in feeling should only be possible for high-hypnotizable participants, the lows serve as an important control for demand effects.

One of the experimentally manipulated factors involved eliciting the sense of potential harm. We did this by asking participants to think of something that could be contaminated with germs and imagine that they had touched it. In contrast, participants in the control condition imagined an innocuous activity, such as reading a book or watching TV.

The other experimentally manipulated factor involved blocking the changes in feeling that would normally occur during washing. We did this with the following suggestion (Woody et al., 2005):

As you know, usually when you wash your hands there is a feeling of satisfaction that comes with it. . . . However, now when you wash your hands, you will find that you do not experience that feeling of satisfaction. There will be a lack of satisfaction as you wash your hands.

The main dependent variable was the duration of subsequent hand-washing behaviour. Hand washing took place at a sink installed with an automatic faucet and an automatic soap dispenser, both activated by proximity of the hands.

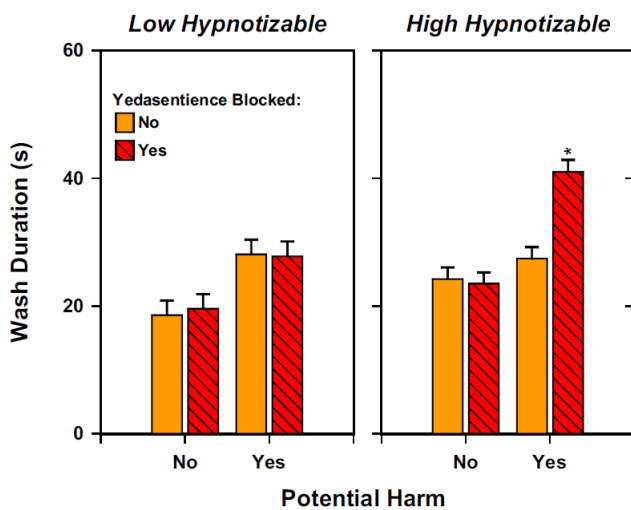


FIGURE 5. Adjusted Mean Washing Duration as a Function of Hypnotizability, Potential Harm, and Blocking of Yedasentience. .Note: mean with an asterisk is significantly different from every other mean, $p < 0.05$. The combination of Potential Harm and blocked Yedasentience yielded prolonged hand washing in the highly hypnotizable participants, compared to all other conditions. Reprinted with permission from Woody et al. (2005).

Before hypnosis, participants had engaged in an initial hand washing to familiarize themselves with the sink set-up. A video camera mounted over the sink recorded the number and duration of all washing episodes.

The security-motivation hypothesis of OCD implies that the combination of potential harm and blocked yedasentience should yield prolonged washing. In addition, because only high hypnotizables should be capable of blocking yedasentience, our prediction was a three-way interaction involving potential harm, blocking of change in feeling, and hypnotizability.

In the results, this interaction was indeed highly significant. Figure 5 shows the corresponding means.

Blocking yedasentience significantly increased wash duration only in the predicted cell, where potential harm had been suggested to high-hypnotizable participants (the bar to the far right). In contrast, blocking yedasentience had negligible and insignificant effects on wash duration when potential harm was not suggested to high-hypnotizable participants and whether or not potential harm was suggested to low-hypnotizable participants. This pattern of results confirms the main hypothesis of the study.

It turns out that participants are quite good at vividly imagining having touched something that may be contaminated. On five-point scales we had them rate the levels of disgust and anxiety they had felt after being asked to think of contact with a potentially contaminating stimulus. Both

high- and low-hypnotizable participants reported substantially and significantly elevated disgust and anxiety, compared to the control condition. A significant Hypnotizability by Potential Harm interaction also indicated that high-hypnotizable participants responded somewhat more strongly to the imagined stimulus.

Also on a five-point scale, participants rated the level of satisfaction they had experienced when subsequently washing their hands. Analysis of these data yielded a significant Hypnotizability by Yedasentience interaction, and Figure 6 shows the associated means.

For the high-hypnotizable participants, blocking yedasentience significantly reduced their experience of satisfaction while washing their hands; whereas for the low-hypnotizable participants, this effect was negligible and statistically insignificant. Thus, as anticipated, only the high-hypnotizable participants could effectively enact the suggestion to block yedasentience.

The experiment also included a more covert index of how participants were feeling: namely, their heart rate. We submitted the heart rate data to a mixed-model analysis of covariance, using baseline heart rate as the covariate. In addition to the between-subject factors, there was also the within-subject factor of Trials, with three times of measurement: Trial 1 was measured just after the hypnotic induction; Trial 2 was measured just after the suggestion of an emotional experience (e.g., a situation of potential harm); and Trial 3 was measured just after the completion of hand washing. The analysis yielded a significant two-way interaction of Trials by Potential Harm, and Figure 7 shows the relevant means.

The mean for Trial 2 (Before Wash) in the *potential-harm* suggested condition is significantly higher than each of the other means, which in turn do not differ significantly from one another. Thus, the suggestion of an experience of *potential harm* increased participants' heart rates, whereas the *control* suggestion of a neutral experience did not. In addition, this *potential-harm*-related increase returned to baseline once the participants had been allowed to wash their hands. (Note that it makes sense for the experimental factor of blocking yedasentience not to be involved in this effect: its manipulation took place between Trial 2 and Trial 3, and heart rate at Trial 3 was measured after the completion of handwashing, when participants had been able to take as long as they wanted to clean their hands.)

In summary, all participants, regardless of their level of hypnotizability, tended to react to their images of potential harm with disgust and anxiety, as well as elevated heart rate and increased washing time, and this elevated heart rate

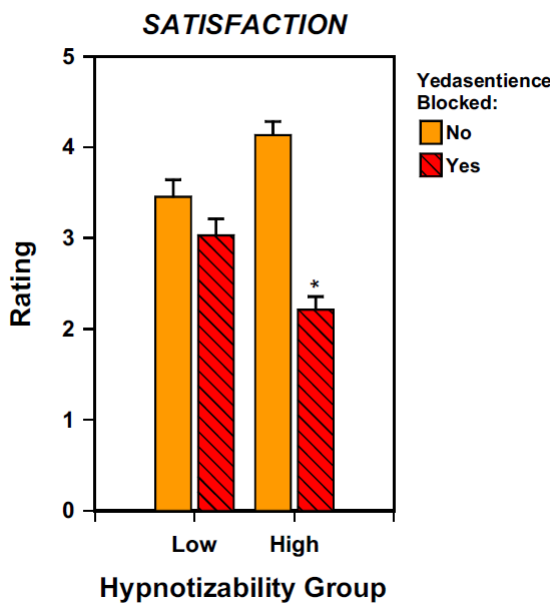


FIGURE 6. Satisfaction as a Function of Hypnotizability and Blocking of Yedasentience. Note: mean with an asterisk is significantly different from the adjacent mean for No Blocking of Yedasentience, $p < 0.05$. Blocking Yedasentience significantly reduced self-ratings of satisfaction during the hand-washing in the high-hypnotizable group, but not in the low-hypnotizable group. Reprinted with permission from Woody et al. (2005).

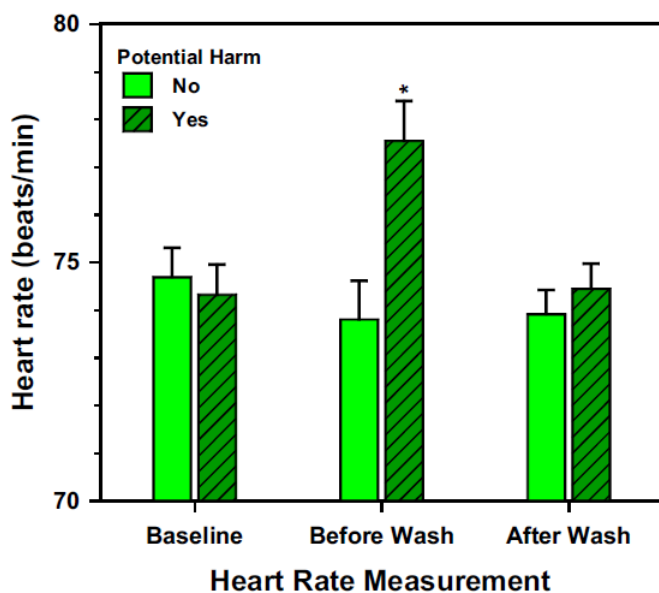


FIGURE 7. Heart Rate as a Function of Time of Measurement and Potential Harm. Note: mean with an asterisk is significantly different from every other mean, $p < 0.05$. The suggestion of Potential Harm increased participants’ heart rates compared to the control suggestion. This increase dissipated once the participants had washed their hands. Reprinted with permission from Woody et al. (2005).

returned to baseline when they had washed their hands. These self-report, heart rate, and behavioural data indicate that participants experienced a situation of potential harm in a vivid and involving way, which is an essential precondition for evaluating the effects of the subsequent blockage of yedasentience.

For the high-hypnotizable participants, the yedasentience-blocking suggestion reduced their rated satisfaction from hand-washing and, more importantly, had the predicted effect of substantially prolonging the duration of their hand-washing in the predicted condition of potential harm. This crucial result lends credence to our hypothesis that a deficit in the feeling of knowing is a plausible explanation for OCD-like behaviour. In addition, the result laid the groundwork for our subsequent experiments, not using hypnosis, that have established the properties of the Security Motivation System (Hinds et al., 2010).

CONCLUSION

To close, let’s return to the overarching theme of using hypnosis to develop and test models of psychopathology. We have seen that by using hypnosis in appropriately preselected participants, it is quite possible to dissociate subjective experience from the objective input available to the senses. In addition, independently manipulating these subjective convictions can provide important

clues about their role in various kinds of psychopathology, for symptoms as diverse as hallucinations and compulsive behaviour.

Skeptics may question whether such hypnotic recreations of clinical disorders really capture something about the essence of the disorders, rather than merely imitating their surface features. To some extent, this skepticism may stem from lack of knowledge about hypnosis and a poorly informed suspicion that hypnotic responses may merely be faking. Recent, high profile work on the neural mechanisms underlying hypnotic effects (e.g., Cojan et al., 2009; Mendelsohn, Chalamish, Solomonovich, & Dudai, 2008) will likely help to dispel some of this skepticism.

There is a broader kind of skepticism that can be expressed as follows: *How can we ever be sure that a hypnotic model fully replicates the clinical condition it is modeling?* This concern, we believe, stems from a misunderstanding of what a model is. Indeed, the same misunderstanding occurs in response to animal models of psychopathology (Szechtman & Eilam, 2005). Chapanis (1961) pointed out that any model is “only an analogy, a statement that in some ways the thing modeled behaves ‘like this’” (p. 188). Even an excellent model is only a likeness, not a replica. For example, there are many aspects of the hypnotic hallucinations in our experiment that are unlike schizophrenic hallucinations, such as their eliciting conditions and content. Likewise, the hypnotic suggestions in our washing experiment do not come close to fully reproducing OCD. Given these unavoidable limitations, Chapanis argued that models should be evaluated differently from theories: “Models, in a word, are judged by criteria of usefulness; theories, by criteria of truthfulness” (p. 119). Good models need to be generative, rather than true; they need to stimulate novel insights and better research.

With this perspective in mind, we can offer a couple of suggestions about using hypnotic models:

1. Pose clear, specific research questions that are plausibly addressed by the model in question. For example, each of the models covered in this article was guided by a central, very specific research question. Because a model is never a full substitute for the phenomenon being modeled, there are likely many other important questions that a particular model cannot plausibly address.
2. Keep in mind that “the worst error committed in the name of models is to forget that at best a model represents only

a part—and usually only a small part—of the thing being modeled” (Chapanis, 1961, p. 126). Experimental results from a hypnotic model need to be evaluated as part of a fuller picture of converging evidence collected in other ways.

Nonetheless, carefully crafted hypnotic suggestions can serve to manipulate theoretically important factors that are difficult to manipulate in other ways. For example, in our washing study, the yedasentience-blocking suggestion served, in effect, as a temporary and reversible “lesion,” corresponding to the hypothesized blockage in the

theoretical model, as shown in Figure 3. Because a more direct manipulation of such a blockage may be impractical, hypnosis may provide a neuroscience tool of considerable potential importance.

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