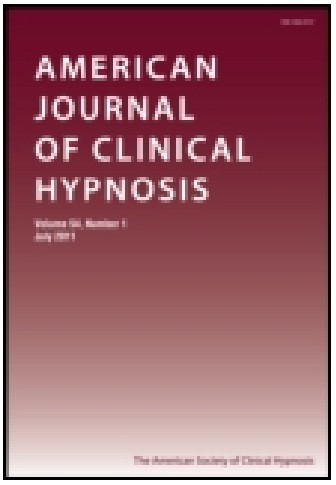

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COMMENTARY

Hypnosis, Neuroplasticity, and the Plastic Paradox

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This special issue on hypnosis and the brain raises the question of the relationship between hypnosis and neuroplasticity—explicitly in a few of the articles and implicitly in others. Neuroplasticity, as I define it, is that property of the brain that allows it to change its structure and function in response to mental experience and activity. Neuroplasticity takes place in the brain, not in the mind, but it is triggered by the mind. I say so, because, we shall see, it is easy to slip into using the terms mental plasticity and neuroplasticity interchangeably. But we have long known that we can "change our minds"; what is new is the discovery that the mind can change the neural tissue; hence, "neuroplasticity." Even before neuroplasticity became widely accepted in the lab, studies of the physiological state of the brain during hypnosis were attempted. Such is the case with most studies in this issue. But now that we understand that the brain is neuroplastic, we must understand not only the state the brain enters in hypnosis but also how the hypnotic process itself alters the brain neuroplastically. So, in my brief comments, I will explore whether the studies in this issue shed any light on how hypnotic induction might foster brain neuroplasticity. It should be noted that in this endeavor, we all stand on the shoulders of Ernest Rossi, prophetic in so many of these scientific matters and, to my knowledge, the first to describe hypnosis as engaging neuroplasticity, having laid out a model in his book, *Psychobiology of Gene Expression: Neuroscience and Neurogenesis in Hypnosis and the Healing Arts* (Rossi, 2002). There he described how the brain enters neuroplastic states in trance.

When I completed my first book on neuroplasticity, *The Brain That Changes Itself* (Doidge, 2007), I was often asked by people "Is there anything I can do to increase

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my plasticity?" When I got that question, I initially had mixed emotions; I had just marshaled a lot of evidence that one's brain was, not as was commonly believed, fixed with its circuits formed and finalized in childhood but was plastic from cradle to grave. I had shown that this was clinically very significant. So the question "Yes, but can I make my brain even more plastic?" seemed an insatiable one that missed the point. Sometimes I would reply, "But the brain's *modus operandi* is to work by changing itself. You already have an embarrassment of riches. Just get out of your brain's way and let it do its thing."

Of course, I realized where the question was coming from: so many human behaviors are rigid. People were saying "How can this be so, if the brain is plastic?" Using Alvaro Pascal-Leone's analogy in my book (Doidge, 2007) and elsewhere, I explained that plasticity is like snow on a hill in winter. If we wish to go down that hill a first time, because the hill is a virgin hill, and because the snow is plastic and pliable, we can take many different paths down that hill. However, being human, if we have a good run the next time we go down that hill, we will be inclined to take a path very similar to the first if we found it very rewarding. And if we keep repeating that, we will develop tracks in the snow, and eventually ruts—precisely because the snow is plastic and pliable and easily molded—and find ourselves unable to stray from the initial path. This is a metaphor for the fact that brain plasticity is competitive, and once we build up circuits in the brain, they get very good at doing what they do and out-compete other potential circuits and ways of doing things.

Thus, plasticity can give rise initially to flexible behaviors, and over time, if repeated, to rigid behaviors as well. In other words, the brain is always plastic even if our behaviors are rigid. And if they are, we then tend—to use the psychoanalytic term—to project this rigidity back onto the brain itself and assume it too is rigid. I called this insight "the plastic paradox" and made the point that one of the reasons we did not appreciate the degree of plasticity the brain has, in fact, is because plasticity gives rise to so many rigid behaviors and remains hidden beneath them.

Many of the conditions that I described in that first book (e.g., chronic pain, poor posture from strokes, focal dystonias, accents, bad habits, obsessional worrying, rigid character neuroses, and defenses) were products of neuroplasticity, giving rise to rigid behaviors or ways of thinking—tracks or ruts in the snow. Explaining these "ruts" or habits by paying attention to what is happening in the brain is not putting old wine in new bottles. It is not a case of simply "rebranding" learning theory and renaming "bad habits" as "bad plasticity." It was always a mystery in conventional learning theory as to why it is so much harder to *un*learn something than to learn it. Unlearning is hard, because once circuits are established in the brain, they are notoriously difficult to change because plasticity is competitive.

For instance, I gave examples in *The Brain That Changes Itself* of many brain-based problems wherein a person tries to change a habit and actually makes it worse. A simple example would be a person who has an accent, who can't hear a word clearly, tries to say a word in a new way, but simply deepens the accent in the process. Because

neurons that fire together wire together, a violinist who has moved two fingers almost simultaneously many hundreds of thousands of times while playing could develop a single brain motor map for those two fingers. Now, if he tries to move one finger the other moves—a condition called focal dystonia. Such a person is caught in a "brain trap." The more he tries to separate the movements, the worse the problem becomes. One reason habits are so hard to break is that established circuits fire stronger, faster, clearer signals than less-established ones. They have an advantage that contributes to the difficulty of learning "a new way" despite the person's best attempts.

I became intensely interested in hypnosis after I began to understand neuroplasticity. First, Pascual-Leone's demonstration that pianists who do only mental practice have the same profound brain changes as those who do physical practice, made clear that visualization techniques often used by hypnotists could change the brain. Second, it seemed that able hypnotists frequently found ways to "get around" what I called the plastic paradox and rigid behaviors. Hypnosis seemed (among other things) to be a technique that might allow a person caught in a brain trap of one kind or another to get out of their rut and not dig themselves in deeper trying. Erickson, Rossi, and Rossi (1976) emphasized helping the patient use existing resources, to "reorganize" themselves, and find new ways of doing things.

Despite my initial reservations about "heightened plasticity," some new discoveries made me wonder whether or not hypnosis might be one way of putting the brain into a state of heightened plasticity or at least facilitating plastic change that did seem to occur spontaneously. Since my first book, it became clear to me that there are heightened and lessened states of neuroplasticity. For instance, cardiovascular exercise increases the production of neurotrophic growth factors that allow the brain to make new connections and learn more effectively. Alzheimer's disease, toxic states, and sleep deprivation, all compromise the brain's ability, at a cellular level, to make new connections. The Alzheimer's brain is a very unplastic brain.

So, now I ask of these individual articles, are there any persuasive suggestions as to how hypnosis might shift the brain into a more neuroplastic state and, at the same time, help a person get out of various highly routinized brain traps and rigid behaviors? I think so.

Hope and Sugarman make the most explicit reference to plasticity, stating first that "hypnosis is a skill set that helps change minds," (this issue, p. 214) which is well known of course, but then assert their hypothesis that, "Trance is defined as the process of developing plasticity within the organic system that is us" (this issue, p. 214). While they don't prove this to be the case, they make some intriguing observations that are extremely helpful in understanding how change is possible in hypnosis, in brain terms. Most importantly, they link the hypnotic trance to the orienting response of organisms to novel stimuli and remind us that when we reorient ourselves, we are preparing to register *something that will involve novel learning*. This is our "hard-wired"—to use their term—adaptation to novelty that "necessarily requires a shift to render the mind

more malleable or plastic" (this issue, p. 218). I believe here they mean not just the "mind" but the brain. The plastic process of trance as they describe it has attributes of the openness to learning that goes with childhood plasticity. In effect, they are arguing that the same brain state that allows curious, fascinated children to learn so effortlessly seems to be triggered in hypnosis. This linking of trance with the orienting response goes well with Rossi's observations of how Erickson (and he) used wonder and the numinous experience to evoke and deepen trance and explains why it is often so effective to arouse curiosity, playfulness, excitement, or even confuse a patient to trigger hypnosis. The orienting response occurs very rapidly in the brain. Because it has been so well studied, researchers may now have a whole new way of investigating and understanding hypnosis and the brain. And as I have shown in *The Brain That Changes Itself* (Doidge, 2007), novelty strongly triggers the chemistry of neuroplastic change.

Lynn, Laurence, and Kirsch's article on hypnosis, suggestion, and suggestibility again emphasizes how hypnosis can trigger changes in the mind that are not normally possible. Quoting Erickson et al. (1976, p. 20) approvingly, they argue that therapeutic hypnosis occurs when "the limitations of one's usual conscious sets and belief systems are temporarily altered so that one can be receptive to an experience of patterns of association and modes of mental functioning . . . that are usually experienced as involuntary by the patient." Lynn, Laurence, and Kirsch argue that for this kind of shift to occur, "a certain degree of mental flexibility is required" (this issue, p. 322). They then make an important link: "Research suggests that activation of the anterior cingulate cortex (ACC) plays a role in state-like change in response to a hypnotic induction" (this issue, p. 322), and cite the 2009 work of Barabasz and Barabasz, among others.

Indeed, the ACC comes up a number of times in this collection of articles. We are very hampered in our ability to say what an individual brain area does, because, in truth, most brain areas are parts of extensive circuits—in fact, parts of *many* extensive circuits, both affective and cognitive. We know that the ACC is over-activated in severe obsessive–compulsive disorder (OCD) and locks people into rigid thought patterns. (Scans show that the person with OCD, who can't get the idea that they made an error out of their head, has an over-firing ACC and related brain areas.) They can't switch out of a thought pattern—"turn the page" and go on to the next thought—and are rigid. By repeating the same thought or action neuroplastically, they reinforce their fears and behavioral rigidities. It is thus interesting to think that being receptive to suggestion might be in some way the opposite of being trapped in an obsessive rumination.

Wark's article on anteriorization also sees a role for the ACC in hypnosis. He cites evidence for a number of activities in hypnotizable subjects that all involve increased frontal cortex activity and behavioral inhibition. Highly hypnotizable subjects are better than others at the following activities: shifting their attention, inhibiting, or "disattending" to unwanted distractions, inhibiting pain, changing sorting rules, breaking promises (in studies, at least), and generally reducing routine, familiar responses, including some destructive impulses. (To use my language, they don't get caught in highly routinized

behavioral activities or ruts.) As Wark puts it, the data has shown that, "When the inferior frontal cortex is activated and signals are sent to other parts of the brain, a routine, familiar, or highly likely response can be reduced or suppressed entirely" (this issue, p. 258). And which parts of the brain are involved in this "anteriorization" in which the centroid electroencephalography (EEG) signals appear to shift forward? Brain signals from the posterior parts of the brain decrease, and there is an increase in firing from the right ACC and the left inferior frontal gyrus. In combination, those two areas help make up the pre-frontal cortex.

The article by Frisaldi, Piedimonte, and Benedetti on placebos and nocebos is intriguing and thorough in its own right. The authors point out that in Alzheimer's (which, we have said, leads to poor brain connectivity and decreased plasticity), the normal placebo response is lost, apparently because of a reduced connectivity between the prefrontal lobes and the rest of the brain. Presumably that connectivity would be essential for hypnosis to be effective as well; it would be interesting to see if patients with Alzheimer's are less subject to the effect of hypnosis. Again we are reminded that the prefrontal cortex is necessary for human neuroplasticity.

The article by Jensen, Adachi, and Hakimian raises interesting questions about brain waves and hypnosis. Earlier researchers anticipated that hypnosis would correlate with alpha states thought to be linked to creative states that occur when the eyes are closed—but the person is awake. Instead, the most common correlation is with the slightly slower theta waves. These waves are increased as people respond to both hypnotic inductions and suggestions. Theta is perhaps best known to be associated with states of drowsiness, but interestingly, it is also associated in the literature with states of orienting (calling to mind the observations of Hope and Sugarman about the orienting response) as well as attention, decision making, emotional arousal, and perhaps equally important for hypnosis in enhancing memory coding and retrieval. (This may account for why patients in hypnosis may have a privileged access, with the help of suggestion, to very vivid sensations or memories, and [according to work such as that done by Watkins and Watkins], warded off ego states, memories, and age regression.) As the authors point out, many hypnotic subjects are encouraged to "let go" and not consciously evaluate or control their responses. This may facilitate novel experiences and thoughts and some "looser associations," such as the kind that occur in dreams and like states before and after sleep. As Freud observed, conscious control of thought is relatively loosened as we approach dream-like hypnagogic and hypnopompic states and begin to make use of primary process associations. Such states may also allow us to be free of the habituated thought patterns that "box us in."

In his article on hypnosis, the self, and empathy, Wickramasekera II shows the high correlation of ability to empathize with others and hypnotizability. He also reminds us of the groundbreaking work of Steven Porges, who has shown that the parasympathetic system (so often triggered in hypnosis) is not only involved in turning off the sympathetic system and calming us, but it also turns on the social engagement system (including all systems involved in communication with others, such as the muscles of the

speech system, facial expression, and those muscles of the middle ear involved in hearing speech) and helps to regulate cardiac vagal tone. Cardiac vagal tone turns out to be a good indicator of unconscious engagement and psychophysiological synchrony with others. It turns out to be a good predictor of the state of hypnosis a person is in and their hypnotic skill. The ability to attune to others and be influenced by others may be one way a person who is trapped in their own rigidities can break out of them. One of Porges's most important insights is how other people can help us with our own autonomic regulation. People can calm us. And all else being equal, this is perhaps why people in successful relationships have better health. The emphasis on empathy and the nervous system reminds us how a master like Erickson could apparently hypnotize people offering only a few words, simply by pacing his breathing, and non-verbal expressions. With an empathic patient, this would be enough for mutual attunement and trance induction. Interestingly, Wickramasekera II also reminds us that the ACC is active during many different types of hypnotic phenomena.

So too does the extremely comprehensive article by Landry and Raz, which points out that neuroimaging studies of hypnosis "reveal a primary role of top-down modulations indexed by the PFC and ACC activity. Dissociation theories argue that most hypnotic phenomena stem from a decoupling of control and monitoring processes ... According to this model, PFC activity reflects the selection and implementation of hypnotic responses, while modulations of the ACC index changes in monitoring. This decoupling between control and monitoring procedures seems to enable suggestions to bypass evaluative procedures and directly act upon control processes" (this issue, p. 304). Thus they link the ACC with monitoring and, if I understand them correctly, evaluative activities that appear to get in the way of allowing new suggestions to take hold.

These important articles are too diverse to be brought under one theme. We have seen, once again, that hypnosis is composed of complex, nuanced phenomena, and that suggestions are so varied that it is obvious that many different brain areas will be involved, even if using standard inductions. All in all, it is wonderful to have an up-to-date assembly of the research on hypnosis and the brain. And yet I cannot help but think that the ACC sticks out like a lilac in January in this collection, and this stirs me to wonder whether it has a special role to play in helping to switch the brain into a state in which it can use its innate neuroplasticity in the service of behavioral flexibility as opposed to behavioral rigidity.

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