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Brain Oscillations, Hypnosis, and Hypnotizability

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Abstract

In this article, we summarize the state-of-science knowledge regarding the associations between hypnosis and brain oscillations. Brain oscillations represent the combined electrical activity of neuronal assemblies, and are usually measured as specific frequencies representing slower (delta, theta, alpha) and faster (beta, gamma) oscillations. Hypnosis has been most closely linked to power in the theta band and changes in gamma activity. These oscillations are thought to play a critical role in both the recording and recall of declarative memory and emotional limbic circuits. Here we propose that it is this role that may be the mechanistic link between theta (and perhaps gamma) oscillations and hypnosis; specifically that theta oscillations may facilitate, and that changes in gamma activity observed with hypnosis may underlie, some hypnotic responses. If these hypotheses are supported, they have important implications for both understanding the effects of hypnosis, and for enhancing response to hypnotic treatments.

Keywords

Brain mechanisms; hypnosis; hypnotizability; theta

Although hypnosis has been used for at least as long as recorded history (Pintar & Lynn, 2008), we are only now beginning to get a glimpse of its neurophysiological underpinnings. During the past decade, in particular, researchers have taken advantage of imaging technology to identify areas of the brain and brain activity patterns associated with both (1) differences between those who score high (so called “highs”) versus those who score low (so called “lows”) on measures of trait hypnotizability and (2) how the human brain responds to hypnotic inductions and suggestions. The knowledge gained from this research has had a tremendous impact on the field, both by helping to validate the effects of hypnosis as being “real” (i.e., as having reliable effects on objective measures of brain activity and function) and by increasing our understanding of the biological mechanisms of hypnosis.

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The main goals of this review are to (1) summarize the current state-of-science knowledge regarding the associations between hypnosis and brain oscillation patterns as measured by electroencephalogram (EEG) and (2) discuss the implications of this knowledge for developing a model of hypnosis based on brain oscillations. In the first section, we briefly describe the physiologic underpinning of EEG signals. The second section provides a review of what is known regarding the role that two brain oscillation bandwidths – theta and gamma oscillations – play in critical cognitive processes; in particular in memory functions. In the third section, we review research on the EEG correlates of hypnotizability and hypnotic responding. In the fourth and final section, we propose a model of how theta oscillations may facilitate response to hypnotic procedures, and provide some tentative speculations regarding the role that gamma oscillations may play in this process. We then discuss the implications of this model for (1) understanding what hypnosis is (and is not) and (2) potentially enhancing the efficacy of hypnotic treatment.

Measuring Brain Activity with Electroencephalogram

Some brain imaging modalities used by researchers, including magnetic resonance imaging (MRI) or computed tomography, provide a structural or anatomical view of the brain. Others, such as positron emission tomography (PET) and its ligand studies, provide a visual display of certain receptors. There are fewer strategies that can be used for measuring brain *function*, and each one that is available has its own limitations. Functional MRI (fMRI) compares changes in blood oxygenation levels that occur as a result of increases in blood flow. These changes likely reflect changes in neuronal activity. fMRI technology is most useful when the goal is to identify neuronal activity changes that occur at a time scale of a few seconds, and can be used to assess relatively rapid (in the seconds to minute range) changes in brain activity in response to specific tasks or specific stimuli; for example, for assessing the immediate response to a hypnotic induction or hypnotic suggestions (e.g., Abrahamsen et al., 2010; Derbyshire, Whalley, & Oakley, 2009). However, fMRI is generally not useful for assessing brain state changes that occur on a longer time scale of minutes or hours. Fludeoxyglucose PET, on the other hand, can examine brain glucose metabolism changes in the brain (associated with neuronal activity) at these slower time scales. Unfortunately, PET involves administration of radioactive materials and is also not ideal for assessing general brain state changes.

Electroencephalogram studies (EEG), on the other hand, are commonly used to measure brain activity (specifically, brain oscillations) associated with brain states such as wakefulness, sleep, and attentiveness. Thus, EEG may be particularly useful for deciphering activity associated with procedures – such as hypnosis – that are thought to produce and be operative via changes in brain *states*. Such brain states can be difficult to decipher using other imaging modalities such as MRI, fMRI, and PET.

The brain is composed of many cells, including neurons, its most functional units. Although there are a variety of different types, sizes, and shapes of neurons, they do share a number of key functional properties. First, each neuron generally has three functional parts: dendrites, the soma (the neuron cell body) and the axon. The dendrites (often organized in structures called dendritic trees) make up the receiving (or input) portion of the neuron. They receive

communication mostly in the form of neurotransmitters released from other neurons, although they can also receive communication from other cells in the brain and via direct physical contact or chemical sensors.

The signals that are received by the dendrites are then combined to provide a time-varying signal to the soma, although exactly how this process occurs is only partially understood. The soma then accumulates the dendritic inputs to produce a binary signal of its own, called an action potential, which becomes the output of that neuron. This output follows an all-or-none principle: once a neuron reaches a certain output threshold it generates a signal to the downstream structures, and then resets itself. This “firing” of an action-potential (the name given to the volley of electrical activity that occurs during the action potential) as the neuron's output signal then travels (“downstream”) to receiving structures via the axon, the output structure of the neuron. Once an action potential reaches the ends of the axon, it triggers the release of signaling chemicals called neurotransmitters. The neurotransmitters allow the signal to pass from the axon to downstream receivers, which are usually the dendrites of other neurons.

An important principle is that different types of neurons are distinct in a number of ways. Most neurons produce, and quite reliably release, a small repertoire of specific neurotransmitters at their axon terminus. The type of output neurotransmitters released by a neuron's axon designate the type of the neuron. Neurons that release neurotransmitters that *stimulate* downstream neurons towards generating an action potential (i.e., make the downstream neuron more likely to fire) are called excitatory neurons. Neurons that release neurotransmitters that make the downstream neurons less likely to fire are called inhibitory neurons. Most neurons in the brain can be classified as either excitatory or inhibitory. Roughly 90% of neurons release excitatory neurotransmitters (with glutamine being the most common excitatory neurotransmitter), and roughly 10% of the brain's neurons release inhibitory neurotransmitters (with GABA being the most common inhibitory neurotransmitter).

Whereas neurons tend to release a very small subset of neurotransmitters, and tend therefore to be classified as either excitatory or inhibitory neurons, their dendrites, by contrast, have receptors for a great variety of neurotransmitters. The excitatory and inhibitory input signals that are combined by the dendrites have different functional properties including evidencing activity at different frequencies. Thus, and depending on the type of neurotransmitters that are present and acting on a neuron, the receiving neuron integrates signal arriving at different time scales some of which are very rapid (e.g., for ionic currents associated with cell signaling that occurs via AMPA, NMDA, and GABA-A receptors) and some that are slow (e.g., for cell signaling that occurs via GABA-B or mGluRs receptors). Moreover, the same neurotransmitter can even have different excitatory or inhibitory effects, depending on the type of receptor that responds to the neurotransmitter.

There are roughly 100 *billion* neurons in the human brain (about as many as there are stars in the Milky Way galaxy), and about 20 billion of these are in the cerebral cortex. Pyramidal neurons, which make up the majority of the cortical excitatory neurons, have roughly 30,000

excitatory and 1700 inhibitory inputs each. It is the dendritic electrical activity of the large numbers of (mostly cortical pyramidal) neurons that form the raw data assessed by EEG.

However, the contribution of different frequencies of electrical activity to the raw EEG is not equal. First, only ionic currents (including ionotropic currents) directly contribute to brain's electrical activity; metabotropic effects are much more difficult to assess. Further, because of the anatomical distribution of different neurons and geometry and orientation of the cells, the post-synaptic dendritic activity from pyramidal cells contributes the most to the EEG signal. Finally, EEG signal reflects the *input* to the pyramidal cells, not necessarily their *output* (although the two clearly correlate to some degree).

The EEG is recorded from the scalp signal via (anywhere from 19 to 256 or more) electrodes which are some distance away from the signal sources. Thus, the raw EEG signal assessed by any one electrode represents the sum of the tens of billions post-synaptic currents in the dendrites of cortical cells, which are further “blurred” by the skull. As a result, the signal at each EEG electrode has a poor spatial sampling of just a few centimeters. Moreover, the synaptic currents of individual neurons are too weak and too fast to be detected by the limited spatial sampling of scalp EEG. Scalp EEG therefore records the common din of the neuronal processes, much the same as microphones placed around and outside the thick wall of an amphitheater might “hear” the response of a 100 or so billion people inside the stadium, who themselves may be responding to the music and rhythms of different bands (or perhaps even the simple “hum” of the equipment) playing on multiple stages. Moreover, the microphones that are placed outside of the stadium walls can only reliably hear the shouts and claps of the 16 or so billion people who are at the edges of the stadium.

Two additional network constraints of the brain give EEG its oscillatory activity. One is that recurrent excitatory and inhibitory loops in groups of neurons (neuron “assemblies” or “ensembles”) form among the ongoing activity of different cells. The brain's network architecture, the amount of neuronal activity, the temporal properties of synaptic signals, and the distance of the neurons from each other within the network, all contribute to the oscillations or frequency patterns that emerge in the background activity of the brain. These are called cortico-cortical sources of EEG.

Second, the common weak inputs to large population of neurons may have a large influence on EEG, because the same activity projects to multiple neurons simultaneously and therefore allows the signals to summate at the scalp. Deep subcortical structures, such as thalamus (and probably basal ganglia) may have this effect. These common inputs, further, have their own neuronal bursting and the temporal characteristics, leading to creation of large coherent oscillations in the postsynaptic dendrites of wider networks. These effects from a few deeper structures are like the effects of the drummer in a band playing close to the middle of the stadium (“deep” structure); a drummer who might influence the rhythmic clapping or singing of a large number of people at the edge of the stadium, but whose activity can only be indirectly assessed by its effects on these individuals who are distant from the source of the rhythms. The most significant of these signals that are generated from relatively deep brain structures are the thalamo-cortical EEG sources. Note that both the cortico-cortical and

the thalamo-cortical influences may be weak at the individual neuron level (i.e., make a relatively small contribution to the function of individual neurons) but summate easily on the EEG due to their commonality among large numbers of neurons.

The oscillatory nature of EEG signals have long been recognized, since the first EEGs were recorded in the 1920s (Berger, 1929). The amplitude of the coherent signal at different frequencies can be expressed in cycles per second (Hz) and can be measured and quantified as a proxy measure of network level activity of large neuronal ensembles. These activities are historically classified as bandwidths of oscillations that occur between specific frequency cutoffs, each of which has a label: delta (0.5 – 4.0 Hz), theta (4.0 – 8.0 Hz), alpha (8.0 – 13.0 Hz), beta (13.0 Hz – 30.0 Hz), gamma (30.0 - 60 Hz), and high gamma (60 -200 Hz). Activities in each band may be assessed using broad or narrow (limited to a narrow frequency range) ranges (and EEG appears to have both narrow-band and broad band activities). Some EEG researchers focus on specific narrow bandwidth ranges (e.g., “high” and “low” alpha; or “38.0 – 42.0 Hz gamma”).

EEG assesses brain activity in these bandwidths using electrodes that are placed on the scalp, using highly conductive paste. However, it is important to remember that given the high level of interconnectivity of the brain, activity assessed from any one electrode (say, an electrode placed near or over the prefrontal cortex), can reflect not only activity in the neurons that lie just below the electrode, but also activity in nearby cortical areas. Moreover, as discussed previously, the primary *source* of the oscillation pattern measured might or might not be the cortical area under the electrode – the initiating source of the oscillation could be in a different area of the cortex or even a deeper structure. For this reason, EEG is much less useful than fMRI or PET for locating the specific source(s) of the brain activity being assessed; EEG indirectly measures *what* happens throughout the brain more than *where* things happen in the brain.

Although there are significant challenges in using EEG to confirm the location or source of a particular bandwidth activity, given the complexities of the many factors that contribute to the signal assessed by scalp electrodes, we do know some things about the sources of different EEG oscillations. For example, slower oscillations (e.g., delta, theta, and alpha) tend to be the common signals among large groups of neurons spread out over larger areas of the brain (likely cortico-thalamic networks), whereas faster oscillations (e.g., beta and gamma) tend to reflect regional activity (mostly cortico-cortical), primarily due to the limitations associated with the conduction delays of axons (Buzsáki, 2006).

Two additional factors make these distinctions between high and low frequency signal characteristics more complex. First, cortico-cortical activities and cortico-thalamic activities occur together, not in isolation, and they can influence each another. For example, it has been observed that theta and gamma oscillations often co-vary and may be phase-locked in different areas of cortex (Buzsáki, 2006; Klimesch, Freunberger, & Sauseng, 2010). We will return to this point when we speculate about the possible role that gamma may play in hypnotic responding.

A second factor that makes the distinctions between different oscillation patterns complex is that anatomically distant structures (such as motor-cortices on the left and right side of the brain, or frontal cortex and limbic system structures) are linked to one another via a large number of reciprocating fast axonal connections. It has been observed for example, that fast wave oscillations in neuron assemblies in one part of the brain sometimes co-vary with fast wave oscillations more distant parts of the brain (Burgess & Ali, 2002; Castro et al., 2014; Zhang, Gan, & Wang, 2014). The physiological and functional implications of these observations and their significance are not yet clear.

Several authors have speculated about the underpinning reasons for co-occurrence of fast wave oscillations occurring in different parts of the brain which may be facilitated or influenced by the generation of slower wave oscillations (mostly theta, but also perhaps alpha; cf. Klimesch, 2012; Klimesch, et al., 2010; Sauseng, Griesmayr, Freunberger, & Klimesch, 2010). This “binding theory” of how information across the brain is organized by underlying rhythms is a motivating concept for our proposed model here (discussed in the fourth section of the paper). However, we are fully aware that the binding theory is as yet unproven. Fortunately, our proposed model is mainly based on empiric observation of the covariance and phase coupling of rhythmic activities, which is compatible with the binding theory but does not rely on it per se.

As we proceed to describe the results of hypnosis EEG research (summarized in section two) and animal and human memory EEG research (summarized in section three) that lay the foundation for our hypotheses regarding the role that theta (and perhaps gamma) oscillations play in hypnotic responding, it is important to emphasize some of the difficulties in interpreting the extant research findings, and which cause us to maintain our ideas as a hypotheses to be tested in future research, rather than hypotheses with existing strong support. First, as will be seen, some of the evidence that contributed to the development of these hypotheses has come from EEG research using intracranial electrodes that directly measure local (mostly gamma) field potentials in the brain. Translating focal gamma changes observed in intracranial studies to measurable scalp EEG signals has significant perils. Because higher frequency activities are more reflective of regional activities, they summate less well at the scalp level. As a result, they lack spatiotemporal coherence. This limitation means that EEG data has a so-called “brown spectrum”: the higher frequency oscillations assessed by EEG, such as gamma, have amplitude orders of magnitude smaller than slower theta or alpha activities. Moreover, faster gamma and beta activities measured from the scalp are more prone to noise contamination from extracranial sources such as myogenic (muscle) activity and electrical noise (e.g., from light bulbs). The scalp EEG measures of faster wave frequencies are therefore less reliable than measures of slower wave frequencies; this would tend to result in less consistent findings of correlates of beta and gamma activity in EEG research, making tests of hypotheses related to these frequencies challenging. In fact, because of these problems, gamma activities faster than 60 Hz probably cannot be reliably assessed by scalp EEG.

In sum, EEG signal power in specific frequency bands may be used as a way of gaining insight into brain states. EEG measures likely reflect (mostly) cortical function at a network level, but may also (to a somewhat limited extent) focal brain activities. The correlates of

brain oscillations at different bandwidths (e.g., sleep states, Ferrara & De Gennaro, 2011; Poe, Walsh, & Bjorness, 2010; memory function, Ferrara & De Gennaro, 2011; Klimesch, 1999, 2012; mood, Davidson, 1992; Fumoto et al., 2010; Yu et al., 2011) are easier to demonstrate for lower frequency (theta and alpha) oscillations, but have been reported for a variety of frequency bands.

Studies on physiologic roles of gamma and theta brain oscillations outside of the hypnotic context

In this section, we summarize the findings that provide an empirical basis of our hypotheses related to the possible associations between gamma, theta, and theta-gamma to hypnotic responding. This research supports the idea that theta oscillations in particular, and perhaps also theta and gamma working together ("theta-gamma coupling"), play an important role in declarative memory encoding and retrieval. As will be discussed in the fourth section of this paper, we hypothesize that the role that brain oscillations play in declarative memory processes may be central to the response to many hypnotic suggestions, and may therefore explain the links found between brain oscillations and hypnosis.

Declarative memory includes memories of facts and events that are recalled and can be "declared." They include both episodic memories (memories of previous events, such as the physical, visual, and auditory sensations associated with on a beach) and sematic memories (memories of facts that can be expressed as words). The link between theta activity and structures in the temporal lobe – in particular the hippocampus – in the formation declarative memories comes from a number of lines of evidence. First, the central role that the hippocampus plays in memory came to light with clinical case studies of people who had sustained damage to this structure, and who were then found to be unable to form new declarative memories or recall events that occurred just before or after the damage (e.g., Scoville & Milner, 1957). Second, the hippocampus has connections with and regularly communicates with virtually all areas of the neocortex (where memories are stored) (Battaglia, Benchenane, Sirota, Pennartz, & Wiener, 2011), which makes it an ideal structure for facilitating the encoding and recall of memories. Third, the dominant oscillation of the hippocampal neurons in mammals is theta (Stella & Treves, 2011) suggesting a role of this oscillation in function of hippocampus and declarative memory.

Additional evidence linking theta to key declarative memory processes in humans comes from studies (for reviews, see Buzsáki, 2006; Lisman & Jensen, 2013) demonstrating that (1) the amount of theta power present at the time individuals are asked to recall a stimulus is associated with the recall of that stimulus (Fell et al., 2011; Guderian, Schott, Richardson-Klavehn, & Duzel, 2009; Lega, Jacobs, & Kahana, 2012; Watrous et al., 2013) and (2) theta activity during a delay period (the period of time between the presentation and a stimulus and the subsequent recollection of that stimulus) is associated with recall accuracy (Gevins, Smith, McEvoy, & Yu, 1997; O. Jensen & Tesche, 2002; Raghavachari et al., 2001; Scheeringa et al., 2009). In short, the evidence is strong that the more theta that is present, the better the "record" and "playback" functions of declarative memory. These findings suggest the possibility that when theta is enhanced, so might declarative memory encoding

and retrieval functions. Further, theta activities in hippocampus and limbic circuits of the brain may be distantly projected to neocortical areas as well (Sirota et al., 2008).

Gamma activities are another brain rhythm which have generated much interest in our understanding of brain function and in hypnosis research. Task specific increased rhythmic gamma activities are observed in related regions across several studies in humans and animals suggesting a functional role of gamma oscillatons (Hermes, Miller, Wandell, & Winawer, 2014). However, the functional role of gamma oscillations (and even the ideal frequency range that is labeled “gamma”) is controversial because of regional differences across the brain, across tasks, and even among type of gamma activity recorded (Crone, Korzeniewska, & Franaszczuk, 2011). Nevertheless, gamma activity has been shown to correlate with brain activation and fMRI both in primary sensory motor cortices and in association cortex (see recent review by Ojemann and cooleague; Ojemann, Ojemann, & Ramsey, 2013). Two types of gamma oscillations appear to be present: a narrow band rhythmic gamma activity and a broad band gamma (see Hermes, et al., 2014). The broad band gamma effects seem to correlate with neuronal firing across different regions and different scales of neuronal activation. They are postulated to reflect of increased pre-synaptic neuronal activation in general. Their correlation with neuronal function is not disputed. Narrow band low gamma activity, particularly in the 40 - 80 Hz range, has been observed in the primary sensory areas (particularly vision) in multiple human and animal studies. Gamma activity also sometimes appears over congruent areas leading to their hypothesized role in information binding across different cortical areas. These are not universally present, however, and appear to strongly influenced by the specific conditions of the task (Hermes, et al., 2014). They are also primarily seen over the early sensory parts of the brain, rather than in the higher cortical areas and association cortex (where they would have been expected to play a role in information binding). Thus, there is not yet consensus on the role that gamma activity plays in cerebral functioning (see Shadlen & Movshon (1999), for example, for a counter argument to the gamma hypothesis).

Moreover, at small scales of local field potentials and small populations of neurons, the distinction between narrow and broad band gamma activities, as well as high and low gamma activities (i.e., < 60 vs. > 60 Hz) is difficult to make. Many studies, in particular fail to make a distinction between narrow and broad band gamma increases. This becomes important in discussions of the gamma-theta association, where the distinction between broad band and narrow band gamma activities if often not made.

Gamma activities have most reliably been observed in intracranial recordings including a number of electrocorticography studies, local field potential studies and other studies done in humans and other primates. Reliable *scalp* measures of focal cortical gamma activities have been more challenging, but have been reported particularly with magnetoecephalography (a technic related to EEG). Whether, when, and where focal gamma activity is seen in association with theta (so called theta-gamma coupling) is therefore not yet entirely clear. Even the frequency of the gamma activity coupled with theta activity is not entirely established.

Linking gamma and theta together are observations of their phase coupling and co-modulation. Gamma frequency activities are sometimes seen in certain phases of theta oscillations in different parts of neocortex. However, there are inconsistencies in the frequency bands involved (such as narrow band “40 Hz” gamma [as assessed, for example, using a 38 – 42 Hz band] or broad band > 60 Hz gamma). Thus, while some research suggest that theta-gamma coupling might occur and be related to some functions (such as memory functions), it is not entirely clear if theta-gamma coupling always happens and whether both the phase and amplitude of gamma co-vary with the phase and amplitude of theta.

It has been postulated that when theta is present, it could inhibit recipient downstream neuron assemblies so that only (or mostly) phase-locked gamma frequencies occur. In this way, slower frequencies (perhaps hippocampal theta but potentially theta from other structures such as basal ganglia) might “control” the firing of faster (neocortical) gamma frequencies. Because declarative memories are thought to be stored in clusters of cortical neuron assemblies that fire at faster frequencies, memories might therefore be more likely to encoded and retrieved in the presence of theta oscillations from the hippocampus (Buzsáki, 2006). Some hypothesize further that theta oscillations help to “bind” neuron assemblies that fire at gamma frequencies in different parts of the brain into a coherent whole of an recallable experience (e.g., the sights, smells, sounds, feelings, and thoughts associated with being on a beach; Buzsáki, 2006).

The amygdala, another part of the limbic system, is closely associated with and lies adjacent to the hippocampus. Also, like the hippocampus, the amygdala receives inputs from and projects to virtually all of the cortical structures. Interestingly, during the waking state, theta oscillations are prominent in the amygdala during periods of intense emotional arousal (Paré & Collins, 2000). Moreover, declarative memory function is enhanced during emotional arousal (Christianson, 1992; Paré, 2003), and the amygdala has been shown to play an important mediating role in this enhancing effect (Cahill & McGaugh, 1998). This observation is consistent with the finding that memory enhancement (perhaps via increases in theta power and/or increases in theta-gamma coupling) occurs following events that trigger both positive or negative emotions (Paré, 2003).

In sum, the primary oscillation pattern of two key components of the limbic system -- the hippocampus at all times and the amygdala during states of emotional arousal -- is theta. A great deal of evidence also supports the conclusion that the hippocampus is involved in declarative memory encoding and retrieval, and that the amygdala is involved in the retention and recollection of emotional memories (e.g., fear conditioning; Buzsáki, 2006; Paré, Collins, & Pelletier, 2002; Sauseng, et al., 2010). The hippocampus is in almost constant dialogue with multiple cortical areas, and this communication has been hypothesized to be mediated primarily via theta oscillations (Buzsáki, 2006). Declarative memories (thought to be brought into awareness when the neuronal assemblies that represent those memories are activated in local neuronal assemblies firing in gamma frequencies) are stored throughout the cortex – tactile memories in the somato-sensory cortices, visual memories in the visual cortex, etc. Based on these ideas, it has been speculated that the hippocampus and amygdala, via theta oscillations, communicate with

neuron assemblies throughout the brain via their influence on phase-locked fast oscillations, which themselves represent specific components of memory. However, we should emphasize that this latter "binding" effect of theta via theta-gamma phase linking, remains a hypothesis that has yet to be adequately tested and established as fact, although preliminary evidence from a number of laboratories – mostly using data from animal memory studies – has provided preliminary support for this model (Buzsáki, 2006) for declarative memory. If evidence is found that supports this theta-gamma mechanism as playing a role in declarative memory formation and retrieval in humans, it may also help explain the findings regarding the links between theta and gamma power and hypnotic responding, as discussed in the next section.

Brain oscillation patterns and hypnosis

Because response to hypnosis is thought to be facilitated by the changes that hypnotic inductions produce in brain states (i.e., "hypnotic trance"), and given the fact that scalp EEG measures brain activity that has been shown to reflect brain states, it is perhaps not surprising that researchers have been examining the associations between EEG-assessed brain oscillations and hypnosis and hypnotic responding for decades. Early on, researchers hypothesized that hypnosis would be associated with alpha rhythms given both (1) initial findings linking meditation practices to alpha oscillations and (2) the perceived similarities between hypnotic and meditative states. Moreover, some early (and even some recent) findings show greater alpha activity among highs relative to lows, as well as increases with alpha following hypnotic procedures (De Pascalis & Palumbo, 1986; Graffin, Ray, & Lundy, 1995; Macleod-Morgan, 1979; Morgan, Macdonald, & Hilgard, 1974). However, even though when significant effects of hypnosis on alpha have been found the direction of the effects are consistent (i.e., hypnosis is not linked to decreases in alpha power), a number of studies have not found an increase in alpha activity with hypnosis (Kihlstrom, 2013; Ray, 1997; Sabourin, Cutcomb, Crawford, & Pribram, 1990).

Findings linking hypnosis to theta oscillations, however, are more common. The evidence shows that highs tend to evidence more baseline theta activity than lows (Freeman, Barabasz, Barabasz, & Warner, 2000; Galbraith, London, Leibovitz, Cooper, & Hart, 1970; Kirenskaya, Novototsky-Vlasov, & Zvonikov, 2011; Montgomery, Dwyer, & Kelly, 2000; Sabourin, et al., 1990; Tebecis, Provins, Farnbach, & Pentony, 1975). There is also a tendency for all individuals – especially highs – to respond to hypnotic inductions and suggestions with increases in theta activity (Jensen, Sherlin, et al., 2013; Sabourin, et al., 1990; Williams & Gruzelier, 2001; but see De Pascalis & Perrone (1996) indicating that this finding is not 100% consistent across all samples and all hypnotic procedures).

The possible involvement of gamma oscillations in hypnosis is intriguing because of the properties of gamma discussed above, such as a high association between gamma activity and measures of focal cortical activation. In the context of difficulties in measuring gamma, disagreements about frequencies involved, the focal nature of gamma activities, and their amplitude fluctuation related to other factors such as theta oscillations, it is not surprising that the research results regarding the associations between gamma activity and hypnosis is complex and at times contradictory (De Pascalis, 2007; Jensen et al., in press). Some studies

have found higher regional areas of baseline gamma activity in highs relative to lows (Akpinar, Ulett, & Itil, 1971; De Pascalis, 1993; Schnyer & Allen, 1995), and also an increase in gamma activity in response to hypnosis (De Pascalis, 1993). Other studies have found *lower* levels of gamma power in highs, relative to lows (De Pascalis, Marucci, Penna, & Pessa, 1987) and decreases in gamma with hypnotic analgesia suggestions among highs (De Pascalis, Cacace, & Massicolle, 2004). Thus, although gamma activity has been shown to be influenced by hypnosis in a number of studies, the direction of that influence is not consistent.

The inconsistency of findings with respect to gamma activity (as measured by scalp EEG) and hypnosis could have multiple explanations. For example, they may be due to the (relative) unreliability of faster oscillations EEG measures alluded to in the first section of this paper; unreliable measures will give unreliable results. Second, there may be regional variations in gamma activity in the cortex which may result in different results from different recording protocols. Fourth, a clear distinction between rhythmic and broad band gamma activities in these studies of gamma-theta oscillation has not been clearly made. A fifth possibility is that the phase locking of theta and gamma may not affect the average amount of gamma present, but its timing. As alluded to earlier, rhythmic gamma activities in primary visual cortices have also been very task dependent. A similar process may be occurring with hypnosis.

Given the findings regarding the consistent associations between theta and hypnotic responding and less consistent associations between gamma oscillations and hypnosis, and if future research continues to support (1) a positive association between hypnosis and theta power and (2) a tendency for activity in the gamma band to respond inconsistently (i.e., sometimes increase, sometimes decrease, and sometime evidence no change) to hypnosis, an important next question is, “What functional role, if any, might theta and gamma oscillations play in facilitating response to hypnosis and hypnotic suggestions?” Addressing this question is the goal of the next section.

A preliminary theta/gamma oscillation model of hypnosis

The research findings demonstrating a link between theta power and hypnotic responding do not prove that theta activity is a “biomarker” of hypnosis, or even that theta oscillations *necessarily* play a causal role in facilitating response to hypnosis. They are, however, consistent with the hypothesis that theta activity *may* play one or both of these roles (Jensen, et al., in press), given that correlation is a necessary but not sufficient condition for causality. In this section, we speculate that slow wave oscillations, and in particular theta, facilitate response to hypnotic suggestions. In addition, given the preliminary evidence that slow wave oscillations may exert some control over fast wave oscillations via phase-linked mechanisms, it is possible that the effects of hypnosis on gamma activity observed in hypnosis and EEG research may be related to these phase-linked mechanisms. While the evidence supporting these ideas – in particular the latter one – is limited at this point, we view these hypotheses as promising and worthy of testing in future research. Moreover, if supported, they could lead to important new understanding of the mechanisms and enhancing the effect of hypnosis.

The potential role of slow wave (primarily theta) oscillations

If research continues to support slow wave (in particular, theta) oscillations as facilitating responses to hypnotic suggestions, how might this occur? While acknowledging that theta is associated with a large number of cognitive activities and states (including, among others, attention, orienting, decision making, feelings of drowsiness, and emotional arousal; Buzsáki, 2005; Paré, et al., 2002), the most commonly identified role for theta, as discussed earlier in this paper, is for enhancing declarative memory coding and retrieval (Bastiaansen & Hagoort, 2003; Buzsáki, 2006; Klimesch, 1999; Klimesch, et al., 2010; Paré, et al., 2002).

We propose here that hypnosis and responses to many hypnotic suggestions involves processes that require – or at least could benefit from – access to activation of the limbic circuits, which are facilitated by theta oscillations. These could include, for example, memories of specific sensations, such as what it feels like for a body part to feel "light," as might be needed for an arm levitation suggestion, or memories of details from places associated with relaxation and pleasure, as might be required to respond to suggestions to re-experience a pleasant activity or to experience oneself as being in a favorite and relaxing place (e.g., a beach, on vacation). Theta may therefore facilitate access to information needed to be able to respond to suggestions for changes in one's experience; that is, *recall* functions necessary for hypnotic responding.

In addition, theta activity may also reflect new connections and new learning. For example, an increased ability to respond to post-hypnotic suggestions or to suggestions for new ideas and perspectives on a problem; that is, suggestions that use the *record* function of the brain (e.g., ".... And the next time it would be of benefit to you, your mind can automatically create these sensations of comfort, contentment, and confidence..."). Thus – and this is one of the key hypothesis that emerges from our proposed model – our model proposes that procedures that increase slow wave oscillations (and in particular theta) will facilitate hypnotic responding.

The hypothesis that slow wave oscillations facilitate response to suggestions, if supported, may help to explain the known variability in hypnotic responding between individuals (Hilgard & Hilgard, 1975; Jensen & Patterson, 2014). We know, for example, that some individuals do not require hypnotic inductions to respond to challenging or difficult suggestions (e.g., suggestions for hallucinations, complete amnesia for the hypnotic session, or elimination of severe pain; Barabasz & Watkins, 2005). On the other hand, we also have evidence that there are many individuals who are not able to easily respond to hypnotic suggestions without an induction, and increase their responsivity to suggestions following hypnotic inductions (e.g., Derbyshire, et al., 2009; Derbyshire, Whalley, Stenger, & Oakley, 2004). Finally, there are some individuals who find response to even the easiest of suggestions challenging, even after hypnotic inductions. If slow wave activity facilitates hypnotic responding, as we hypothesize here, then we would predict that not only would baseline slow wave activity predict subsequent response to hypnotic suggestions, we would also predict that: (1) those individuals who do not require a hypnotic induction to respond to challenging hypnotic suggestions would have higher than average *baseline* levels of theta wave power; (2) individuals who demonstrate significant increases in response to

suggestions following a hypnotic induction would evidence an *increase* in theta following the induction; while (3) individuals who evidence an inability to respond to hypnotic suggestions even after a hypnotic induction would not evidence elevations in slow wave power following the induction. These hypotheses are easily testable.

Some preliminary support for these hypotheses has been reported in the research literature, previously discussed, showing significantly higher level of baseline theta activity in highs relative to lows (Freeman, et al., 2000; Galbraith, et al., 1970; Kirenskaya, et al., 2011; Montgomery, et al., 2000; Sabourin, et al., 1990; Tebecis, et al., 1975) and a tendency for hypnotic inductions to result in increases in theta activity, especially among highs (Jensen, Sherlin, et al., 2013; Sabourin, et al., 1990; Williams & Gruzelier, 2001). Further support for the slow wave hypothesis would come from research demonstrating that the observed increases in responding to suggestions following a hypnotic induction are mediated by increases in slow wave power.

The role of faster (primarily gamma) oscillations

As we have alluded to above, theta-gamma coupling has been described for both hippocampal neurons and neocortex. Some researchers argue that phased-locked coupling of higher frequency oscillations (such as gamma) to the lower frequency ones is a mechanism that creates a mental representation (during declarative memory encoding or recall, for example) by linking neuron assemblies that fire together at the same frequency (Klimesch, et al., 2010). This has been hypothesized as a mechanism information binding (Buzsáki, 2006). A competing hypothesis, however, could be that gamma-theta oscillation is a reflection of co-activation of limbic and neocortical circuits that have their own inherent frequencies, but that this coupling does not serve another purpose beyond reflecting the co-activation. Either way, there is ample evidence that gamma activities reflect increase in neuronal firing and their phase locking to lower frequencies may reflect coactivation of another process.

Thus, in our model, an induction that invites or requires a subject to “let go” and not monitor, evaluate, or consciously control responses would be hypothesized to require a reduction of activity in the neuron assemblies associated with these tasks – perhaps assemblies in the anterior cortex (Dienes & Hutton, 2013; Gruzelier, 1998). Successful response to suggestions for analgesia would be hypothesized to be associated with reductions in the activity or connections of neuron assemblies associated with the processing of pain (i.e., a reduction in gamma activity in assemblies associated with pain in the sensory cortex), while suggestions for increased comfort would be hypothesized to be associated with increases the activity of or connections between neuron assemblies associated with memories of comfortable sensations (i.e., an increase in gamma activity in assemblies associated with comfort in the sensory cortex). Note that the activation of the emotional content (comfort, pain, etc.) could potentially lead to activation of limbic circuits as well. Thus, depending on the suggestions, once the suggestion is made, and assuming of course an adequate amount of motivation is present and hypnotic talent of the subject (Jensen et al., in press), the subject should pull together the resources to respond to the suggestion by eliciting or inhibiting neuron assemblies to fire in gamma frequencies.

Thus, we propose that response to hypnosis and hypnotic suggestions should be associated with (1) an *increase* in slow wave oscillations (primarily theta,) and (2) a *change* (either an increase or decrease, depending on the suggestion offered and the neuron assemblies involved in the experience being suggested) in fast wave oscillations (primarily gamma). Further, since theta-gamma coupling may be at play, an absolute increase or decrease in gamma may not be necessary or even easily measured. Gamma may be higher certain phases of theta oscillations and lower in other phases, and this may vary across brain regions as well.

The evidence emerging from EEG research on hypnosis and other mental processes appears to be consistent with this model, but many aspects of the model have yet to be tested and many questions remain. Primary among these questions is whether slow wave activity theta (1) merely enhances, (2) is necessary for, or (3) is both necessary and sufficient for hypnotic responding. If slow wave oscillations merely *enhance* hypnotic responding, then procedures that tend to increase slow wave oscillations should increase the efficacy of hypnotic treatments, but perhaps more so in some individuals (i.e., those who respond to the procedures by showing an increase in slow wave activity) than others. If slow wave oscillations are *necessary* for hypnotic responding, then it is likely that a certain minimal level of slow oscillation power is needed in order to respond to hypnotic suggestions.

Clinical implications of a brain oscillation model of hypnotic responding

Support for the slow oscillation hypothesis support the need for research that would examine factors that could enhance slow oscillations, as a method for enhancing the beneficial responses to hypnotic interventions. This could include not only an examination of different types of hypnotic inductions, but also therapist factors (i.e., therapists “being hypnotic”; Yapko, 2003) such as ability to develop and enhance rapport, the timing and phrasing of language, and environmental factors (music in the waiting area, calming versus chaotic clinic environment) that could influence oscillation patterns, and therefore clinical responsivity.

The model presented here suggests the possibility that hypnotic procedures that result in increases in theta (or suggestions presented to individuals who already have a relative preponderance of theta oscillations) may allow for the facilitation of activation of limbic circuits and other neo-cortical circuits in a controlled way that allows for rapid changes in learned associations that have formed in the brain between these areas. For example, to produce dissociations between memories and emotions (i.e., reduce anxiety and fear responses to stimuli that were previously closely linked), or to strengthen associations between stimuli and images or emotions that were previously only weakly associated (i.e., enhance positive responses to specific cues). The model may thus explain the common observation that adding hypnosis to other treatments can enhance their efficacy (Kirsch, Montgomery, & Sapirstein, 1995).

If additional research supports a role for slow wave oscillations for enhancing response to hypnosis, the clinical implications would be significant. In this case, clinicians could potentially increase response to hypnotic suggestions among “lows” – that is, individuals

who do not generally respond well to classic hypnotic inductions – by using one or more of a growing number of strategies that have been shown to increase theta activity, such as music or monochrome sounds (Lee, Bhattacharya, Sohn, & Verres, 2012), some (but not necessarily all) meditation training practices (Lutz et al., 2009), and neurofeedback (Batty, Bonnington, Tang, Hawken, & Gruzelier, 2006; Jensen, Gertz, et al., 2013). Depending on the results of research examining the effects of additional clinical practices on enhancing slow wave oscillations, briefly alluded to above, these practices could be systematically added to clinician repertoires.

Implications of the theta/gamma model for understanding what hypnosis is and is not

If, as the model presented here hypothesizes, slow wave oscillations are found to at least facilitate if not be necessary to respond to hypnotic suggestions, then this could provide what might be a novel broadened view of what hypnosis is and is not. In our model, "hypnosis" could be viewed as a use of suggestions for creating changes in thoughts, feelings, or behaviors when the clinician views the client as having enough theta power to be able to respond to those suggestions.

With this view, "hypnotic" strategies, then, could include (1) *any* strategy that enhances slow oscillations (using traditional hypnotic inductions, but also any technique that has been or is ultimately shown to increase slow oscillations) and/or being aware of behavioral signs indicating an increase in or adequate level of theta, coupled with (2) suggestions that enhance existing connections among neuron assemblies (e.g., those consistent with the subject experiencing of himself or herself with useful images or having a positive view of the future) or that create new ones.

What is and is not "hypnosis"?

A number of interventions that are not necessarily viewed as "hypnosis" would fit into a broadened definition of hypnosis that includes any strategy that increases slow oscillations coupled with suggestions. These include classic relaxation training and autogenic training procedures, where the clinician suggests experiences of relaxation that would require engagement of theta activity in our model (i.e., asking the subject to turn inward and pull from declarative memory experiences of relaxation, or "warm and heavy arms"). Because relaxation training and autogenic training also include at least one explicit suggestion – for the subject to feel relaxed – these procedures combine a strategy that potentially results in both an increase in slow oscillations plus a suggestion; that is, "hypnosis" by our definition. In addition to explicit suggestions for relaxation, these treatments also often include additional implicit suggestions. For example, relaxation training in the context of headache treatment has the implicit suggestion that the intervention will reduce headache frequency and severity. We would hypothesize from our model that individuals with more pre-treatment theta power and individuals with headache who evidence an increase in theta power with "relaxation training" would therefore be more likely to be treatment responders by evidencing reductions in headache frequency and severity.

A number of other treatments might also be viewed as hypnosis using our model; or at least very close cousins. For example, some, but not all, meditation practices result in increases in theta power (Lutz, et al., 2009). More often than not, people engage in meditation training for a specific reason or because they believe that meditation will result in specific benefits. That is, they have received suggestions (perhaps through reading, perhaps through discussions with the individual providing the training) regarding the potential benefits of meditation. In the presence of such suggestions – here in the form of self-suggestions also known as outcome expectancies – our model would predict that those participants with higher baseline theta or who evidence increases in theta with meditation training and practice would be more likely to experience the expected benefits of the meditation training.

Here we can identify a clear overlap between the model of hypnosis presented here and some social-cognitive views of hypnosis. Specifically, many social-cognitive models place an emphasis on subject beliefs as playing an important (if not critical or central) role in hypnotic responding (Kirsch, 1991; Lynn, Kirsch, & Hallquist, 2008). In the model presented here, we would view such beliefs as (self) suggestions, which are then more likely to be responded to when and if the subject engages in procedures (such as a hypnotic induction, relaxation training, certain meditation practices) that increase slow wave activity. Moreover, the model would predict, as do some social-cognitive models, that a classic hypnotic induction is neither necessary nor sufficient to respond to suggestions, because there will likely always be at least some individuals who have an adequate level of baseline theta activity to be able to respond to suggestions, with or without an induction. Our model *would* predict, however, that outcome expectancies play a larger role in treatment outcome in hypnosis interventions among individuals with more baseline theta power than among individuals with less baseline theta power. Social-cognitive models of hypnosis would not make this specific hypothesis, because they focus on social and psychological factors, and not biological ones.

On the other hand, there are also clearly interventions – many of which are known to be effective – which would fall outside of our definition of “hypnosis”. We have already mentioned two of these: meditation procedures that do not result in increases in theta (and there are some of these, e.g., Chiesa & Serretti, 2010; Fell, Axmacher, & Haupt, 2010; Travis & Shear, 2010), as well as meditation procedures that have a clear lack of therapeutic goal; i.e., a lack of even an implicit suggestion – meditation for meditation's sake alone. In addition, we expect that cognitive therapy as practiced by some clinicians would not fall into our definition of hypnosis. For example, cognitive therapy can be provided with a focus on logic and Socratic discussion, which may involve less focus on one's felt experience, and a greater focus on what the clinician is saying and an ongoing client-clinician discussion; activities that may be more likely associated with beta oscillations than theta oscillations.

On the other hand, we also expect – indeed we have observed – cognitive therapists who are, as Michael Yapko puts it, able to “be hypnotic” when providing cognitive therapy. These are therapists who seem to naturally engage in behaviors and interactions that result in increased rapport. They may speak rhythmically and slowly, and invite moments of quiet reflection in their clients. In the presence of such a clinician, we speculate many clients would evidence an increase in slow oscillation patterns, and then be more open to suggestions (perhaps

presented in the form of “rational thinking”). Related to these ideas, we have developed and are now in the process of testing via a randomized clinical trial a form of hypnotic cognitive therapy based in large part on the work of Michael Yapko (Yapko, 2001), Assen Alladin (Alladin, 2008), Moshe Torem (Torem, 2006), and others, which seeks to first increase responsivity to new ideas and cognitive content via a hypnotic induction, followed by the presentation of these ideas and cognitive content (Jensen et al., 2011).

What role for “trance”?

A great deal of energy has been spent in our field discussing and debating the relative merits of the “trance” concept in explaining the effects of hypnosis. On the positive side, this debate has led to some important advances in our knowledge about hypnosis and the predictors of hypnotic responding (Jensen, et al., in press). Our current thinking on this issue is that the existing evidence can be (and has been, by different scientists) interpreted to be consistent *both* with the notion that “trance” plays an important role in hypnotic responding *and* that “trance” is a concept that is not needed to explain hypnotic responding (Jensen, et al., in press).

Our model does not help to resolve this debate. The key hypothesis is that slow wave oscillations, and in particular theta oscillations, facilitate hypnotic responding. Therefore, according to this model, hypnotic inductions, when they enhance hypnotic responses, do so in part because they increase theta. Because theta power is associated with certain states (including high levels of focused attention), it would be possible to view the “hypnotic trance” (as experienced by the subject or as observed via behavioral correlates by the operator) as reflecting a high level of theta activity. In this case, then our model would hypothesize that such a “trance” would facilitate response to hypnotic suggestions; indeed, “trance” as defined as reflecting a requisite level of slow oscillations, might ultimately be found to be necessary for hypnotic responding. From this viewpoint, and with high levels of theta representing a “state” of readiness to respond to suggestions, our model can be viewed as a state model of hypnosis.

On the other hand, there is at least some measurable slow wave activity in all living mammals at all times. Slow oscillations are not unique to hypnosis or even unique to humans. Moreover, theta oscillations vary in power naturally throughout the course of a day, while an individual is awake and asleep. Like the socio-cognitive (i.e., non-state) models of hypnosis, our model hypothesizes a role for subject beliefs and expectations (viewed as self-suggestions in our model), as playing a role in hypnotic responding. Thus, one could easily say that our model does not propose a “hypnotic” state that is qualitatively different from an individual's “usual” state; merely a continuous biological factor (theta power) that varies naturally throughout the day – although one that can also be influenced by environmental factors, including formal hypnotic inductions – that can enhance an individual's ability to respond to suggestions.

Our model can therefore be viewed as something “in between” the state and non-state approaches. Perhaps, because the same evidence can be interpreted as either supporting or not supporting trance (state) and non-trance (non-state) models, we wonder if the questions,

"Is hypnosis a state or is it not a state?" and "Is or is not a trance required for hypnotic responding?" may have outlived their usefulness.

Summary and Conclusions

A growing body of research has shown that the magnitudes of different brain oscillation patterns are associated with response to hypnotic inductions and suggestions. Specifically, hypnosis has been shown to be associated with more theta oscillations, and hypnotic responding has been shown to be associated with changes in patterns of gamma oscillations (with potentially increases, decreases, or changes in timing of gamma oscillations), depending on many factors including the suggestions given. Memory research supports the importance of theta oscillations in particular and perhaps also theta-gamma phase-locked oscillations in the recording and recall of declarative memory. Declarative memories (memories of events and sensations that can be "declared") appear to be important to virtually all hypnotic responses. We therefore hypothesize that theta oscillations facilitate hypnotic responding, and speculate that theta-gamma phase-locked oscillations may provide a physiological explanation for hypnosis by suggesting linking of limbic and neocortical circuits. If supported by future research, these hypotheses have important implications for understanding (and predicting) hypnotic responding, as well as for enhancing response to hypnosis treatments.

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