Intl. Journal of Clinical and Experimental Hypnosis, 56(1): 99–116, 2008

ISSN: 0020-7144 print / 1744-5183 online
DOI: 10.1080/00207140701506482

Routledge
Taylor & Francis Group

GENETICS AND NEUROIMAGING OF ATTENTION AND HYPNOTIZABILITY MAY ELUCIDATE PLACEBO

AMIR RAZ[1,2,3]

Columbia University College of Physicians & Surgeons and New York State Psychiatric Institute, New York, New York, USA

Abstract: Attention binds psychology to the techniques of neuroscience and exemplifies the links between brain and behavior. Associated with attentional networks, at least 3 brain modules govern control processes by drawing on disparate functional neuroanatomy, neuromodulators, and psychological substrates. Guided by data-driven brain theories, researchers have related specific genetic polymorphisms to well-defined phenotypes, including those associated with different attentional efficiencies and hypnosis. Because attention can modulate both cognitive and affective processes, genetic assays together with neuroimaging data have begun to elucidate individual differences. Findings from genetic assays of both attention and hypnotizability pave the way to answering questions such as how high hypnotizable individuals may differ from less-hypnotizable persons. These exploratory findings may extend to the identification of placebo responders.

Genetic findings from attentional and hypnotic assays can serve as a vehicle for elucidating individual differences (Raz, 2006b; Raz & Buhle, 2006). Moreover, genetic data may permit not only discerning high hypnotizable persons from less-hypnotizable individuals but also unraveling the biological characterizations of good placebo responders (Raz, 2006a). Following a "paradigm shift" to extend the results from the genetics and brain imaging of attention and hypnotizability to placebo (Raz, 2006b; Raz & Buhle, 2006; Raz, Fan, & Posner, 2005; Raz, Fan, & Posner, 2006), the present article outlines the rationale as well as mechanics for such a transition and the relative merits such an approach may entail.

Manuscript submitted December 15, 2004; final revision accepted March 8, 2005.

[1]I thank Irving Kirsch and George Aaron for their support and encouragement throughout various stages of this project.

[2]Dr. Raz is now at McGill University, Montreal, Quebec, Canada.

[3]Address correspondence to Amir Raz, PhD, ABPH, Institute of Community & Family Psychiatry, 4333 Cote Ste Catherine Rd., Montreal, Quebec H3T 1E4. E-mail: amir.raz@mcgill.ca

Relying on the relationship between attention and hypnosis (Raz et al., 2005), the efficiency of disparate attentional typologies relates to specific genetic polymorphisms (Raz, 2004; Raz & Buhle, 2006). Ample findings propose that attention influences both cognition and affect (Bush, Luu, & Posner, 2000); more recent findings posit that genetic assays in concert with brain-imaging data pave the way to a more scientific basis of individual differences in general and of hypnotizability in particular (Fan, Fossella, Sommer, Wu, & Posner, 2003; Raz, 2006b; Raz et al., 2005). Finally, drawing on such factors as self-regulation, effortful control, hypnotic suggestion, and expectation—accounts of these constructs appear in a recent review (Raz & Buhle, 2006)—the present article leads to the notion of placebo and spotlights those individuals who may favorably respond to placebo-like interventions.

TYPOLOGIES OF ATTENTIONAL NETWORKS

According to Michael I. Posner, attention can be construed in terms of three networks: 1) obtaining and maintaining the alert state; 2) orienting to sensory information; and 3) the executive functions involved in the resolution of conflict between competing areas of the brain that might be simultaneously active (Posner, 2004). The alerting network relies heavily on thalamic areas, locus coeruleus, and cortical areas. The orienting network relies heavily upon parietal systems, including the superior parietal lobe and the temporal parietal junction. It is involved in both orienting to visual information and stimuli in other modalities. The executive attention network relies heavily on the anterior cingulate as well as lateral areas of the prefrontal cortex (Figure 1). Detailed accounts of the typology and anatomy of these attentional networks have been described elsewhere (Raz, 2004; Raz & Buhle, 2006).

This model of attention has been influential in the field, not only because other researchers have independently proposed similar accounts (Parasuraman, Warm, & See, 1998) but because neuroimaging assays (Corbetta, Kincade, Ollinger, McAvoy, & Shulman, 2000; Hopfinger, Buonocore, & Mangun, 2000) and rigorous analyses of more ecological data supported Posner's putative typology in both adults and children (Manly et al., 2001; Robertson, Ward, Ridgeway, & Nimmo-Smith, 1996). Furthermore, Posner and his colleagues have developed methods for measuring each of these attentional networks and many researchers have since been widely using their paradigm (Fan, McCandliss, Sommer, Raz, & Posner, 2002; Raz, 2004; Raz & Buhle, 2006).

In a set of important experiments into the neurochemical bases of attention, Richard Marrocco and his collaborators were among the first to associate these brain networks with different neuromodulators

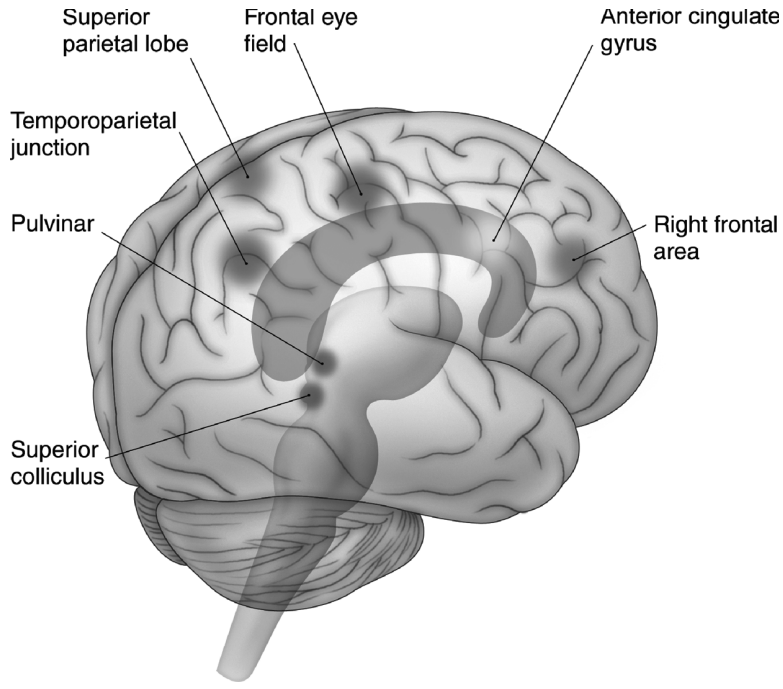

Figure 1. **A sketch of the functional anatomy of the attentional networks. The pulvinar, superior colliculus, superior parietal lobe, and frontal eye fields are often activated in studies of the orienting network. The temporoparietal junction is active when a target occurs at a novel location. The anterior cingulate gyrus is an important part of the executive network. Right frontal and parietal areas are active when people maintain the alert state. From Raz & Shapiro (2002). Copyright 2002 by JAMA. Adapted by permission.**

(Marrocco & Davidson, 1998). Marrocco's study of the neuropharmacology of attention in alert monkeys shows that norepinepherine largely modulates the alerting network, acetylcholine wields a strong influence on the orienting network, and dopamine is the main neurotransmitter of the executive network.

Drugs such as clonidine and guanfacine act to block norepinepherine and reduce or eliminate the normal effect of warning signals on reaction time, but they have no influence on orienting to the target location. Injections of scopolamine—which acts by interfering with the transmission of nerve impulses by acetylcholine—directly into the lateral intraparietal area of monkeys, a brain area containing cells that are influenced by cues about spatial location, have been shown to have a large effect on the ability to shift attention to a target. In addition,

cholinergic drugs do not affect the ability of a warning signal to improve performance. Thus, a double dissociation relates norepinepherine to the alerting network and acetylcholine to the orienting network. The executive network involves the anterior cingulate and lateral frontal cortex modulated by the dopamine system. Together with Figure 1, Table 1 depicts the structures involved as the sources of the "Posnerian Trinity of Attention," the sites on which these structures operate, and the neuromodulators they use (Raz, 2004).

Although the sources of attentional effects are limited to networks, attention can influence any part of the brain, including the primary sensory areas and emotional areas of the brain. This was demonstrated in a summary of many studies looking at the role of the anterior cingulate cortex (ACC) in the monitoring and resolution of conflict (Bush et al., 2000). Although there are many current disputes about the exact mental operations the ACC performs (Bush, 2004), it is useful to think about this area of the brain as involved in self-regulation, where subjects are required to "damp down" or "ward off" negative thoughts (Ochsner, Bunge, Gross, & Gabrieli, 2002; Ochsner et al., 2001) or even pleasant thoughts (Beauregard, Levesque, & Bourgouin, 2001).

It is possible to distinguish between the dorsal portion of the ACC, which is involved in cognitive tasks, and the ACC's ventral region, which is more involved in emotional tasks (Bush, 2004; Bush et al., 2000). Thus, it is reasonable to consider the ACC an important node in

Table 1
Attentional Networks – Brain Regions and Neuromodulators

Attentional Network	Gross Neuroanatomy	Primary Neuromodulator
Alert	Locus Coeruleus	Norepinepherine
(arousal, vigilant attention)	Right frontal and parietal cortex	
Orient	Superioparietal	Norepinepherine
	Temporoparietal	
	Frontal eye fields	
	Superior colliculus	
	Acetylcholine	
Select	Anterior cingulate cortex (ACC)	Dopamine
(Executive, conflict, supervisory, focal, metacognitive attention)	Lateral ventral prefrontal cortex	
	Basal ganglia	

the monitoring and resolution of conflict that is involved in emotional and cognitive regulation.

Cognitive Regulation and Effortful Control in the Context of Hypnosis

Attention, suggestion, and expectation may effortlessly alter information processing in the human brain (Fan, McCandliss, et al., 2002; Raz, 2004; Raz & Buhle, 2006; Raz et al., 2005; Wager et al., 2004). The ability to use higher brain functions to influence downstream processing draws on the neural substrates that are often explored in the study of volitional agency, effortful control, and consciousness (Baumeister & Vohs, 2004; Churchland, 2002; Posner, 2004; Raz & Buhle, 2006; Raz et al., 2005; Raz, Kirsch, Pollard, & Nitkin-Kaner, 2006; Venter et al., 2001; Wegner, 2002). For example, by creating a situation in which subjects could look directly at a five-letter word without attending to it (i.e., they had to respond to a superimposed stream of pictures shown in different orientations), an fMRI study reported failure to perceive words even for decidedly familiar and meaningful stimuli placed at the center of gaze (Rees, Russell, Frith, & Driver, 1999). Additionally, positron emission tomography (PET) data showed that highly hypnotizable individuals who were hypnotized neither perceived color nor activated extrastriate areas related to color after they had been given the hypnotic suggestion to see a color pattern in gray-scale (Kosslyn, Thompson, Costantini-Ferrando, Alpert, & Spiegel, 2000). Finally, PET assays of pain showed that specific modulatory hypnotic suggestions could affect activation of different brain structures: whereas suggesting a drop in pain unpleasantness (i.e., pacifying conflict) reduced specific activity in the ACC (Rainville, Duncan, Price, Carrier, & Bushnell, 1997), hypnotically suggesting decreased pain intensity produced activity reduction in somatosensory cortex (Hofbauer, Rainville, Duncan, & Bushnell, 2001). These accounts underline the influence attention and hypnotic suggestion can impart to conflict situations and top-down cognitive control (Fan, McCandliss, et al., 2002; Posner & Rothbart, 1998; Rainville, 2002; Rainville, Hofbauer, Bushnell, Duncan, & Price, 2002). Recent findings from my work with colleagues also support this view (Fan, McCandliss, et al., 2002; Raz, 2004; Raz, 2006b; Raz & Buhle, 2006; Raz et al., 2005; Raz et al., 2006; Raz et al., 2003; Raz, Moreno-Íñiguez, Martin, & Zhu, 2007).

Cognitive regulation relates to top-down processing, the preferential processing of sensory stimuli according to preexisting schemas or expectations. Cognitive regulation (e.g., not scratching an itchy mosquito bite) is associated with effortful control. Mary Rothbart initially coined the term *effortful control* to describe a level of control that emerges in children's development in the context of temperament. The

term has since evolved to entail "the ability to inhibit a dominant response to perform a subdominant response" (Rothbart & Bates, 1998, p. 137) or the efficiency of executive attention, including the ability to inhibit a dominant response or to activate a subdominant response, to plan, and to detect errors (Rothbart & Bates). In fact, effortful control now pertains to the ability to willfully or voluntarily inhibit, activate, or modulate attention (Rueda, Posner, & Rothbart, 2004). It is not surprising, therefore, that measures of effortful control often include indices of attentional regulation (e.g., the ability to voluntarily focus or shift attention as needed, also called attentional control) or behavioral regulation (e.g., the ability to inhibit behavior as appropriate, called inhibitory control).

NEUROIMAGING AND GENETIC ASSAYS PROBE INDIVIDUAL DIFFERENCES

Two major events permitted a breakthrough in science's ability to illuminate individual differences. First, imaging of the living brain provided science with a glimpse inside the human brain as people think (Posner & Raichle, 1996). When combined with electrical or magnetic recordings, it is possible to observe the neural circuits that are involved in computing aspects of various cognitive tasks. Researchers can now create local images of human brain activity through changes in cerebral blood flow.

Being able to see things always had a dramatic impact on science (e.g., the microscope). While it is possible to use imaging data to argue for separate networks underlying disparate cognitive abilities, it would be impossible to argue from imaging data alone that these are the only separable domains. Indeed, it is more likely that different tasks, clearly within one domain, can still be distinguished by their functional anatomy.

The second major event at the end of the 20th century was sequencing the entire human genome (Venter et al., 2001). Consequently, it was possible not only to study the functional anatomy of brain networks but also to examine how genetic differences might lead to individual variations in the potential to use these networks in order to acquire and to perform skills. However, the route from genetic endowment to performance is neither simple nor separate from an understanding of the brain networks themselves.

In the domain of attention, researchers have begun to develop methods for examining the efficiency of attentional networks in individuals to study how genes and specific experience change them in the course of human development. Cognitive neuroscientists are beginning to explore the possibility of testing some of the genetic effects on attentional networks. For example, since a twin study of attention

suggested that the executive attention network had high heritability (Fan, Wu, Fossella, & Posner, 2001), genes related to the dopamine system were examined (Fossella et al., 2002). Four genes in this system were found to be significantly related to the executive network. When the alleles yielding relatively good executive performance were compared with those yielding relatively bad performance in an fMRI study, the major difference between the subjects was in the ACC, a part of the executive network associated with dopamine (Fan et al., 2003). These results suggest that it is possible to examine individual efficiency in specific neural networks by combining the methods of brain imaging with modern genetic studies.

The Genetics of Attention Inform the Genetics of Hypnotizability

The DRD4 gene had been shown to relate to some of the behaviors that occur in attention-deficit hyperactivity disorder (ADHD; Ding et al., 2002; D. L. Grady et al., 2003; LaHoste et al., 1996; Swanson, Oosterlaan, et al., 2000; Swanson et al., 1998, 2001; Swanson, Flodman, et al., 2000; Wang et al., 2004). For example, previous clinical studies show that a subset of children who have a particular allele of the DRD4 gene (7 repeat) have normal performance in attentional tests, while children with the same diagnosis but without this allele show abnormal performance. Testing a large sample of children diagnosed with ADHD both with and without the 7-repeat allele as well as healthy children with and without the allele should test and extend this result. These data would show if there are common genes in families that contribute to the occurrence of ADHD and if children respond differently to psychopharmacological treatment (e.g., methylphenidate) based on their genetic makeup. In addition, the candidate gene approach allows insights into the genetics of normal attention. We have identified at least 40 genetic polymorphisms that affect transmitter systems related to known attentional networks. These candidate gene polymorphisms may relate to the efficiency of the three attentional networks. Accordingly, genotyping 200 healthy individuals who responded to ads in the New York City area, my colleagues recently looked at different alleles, or polymorphisms, in the DRD4 genes (i.e., the number of times a particular 48 base pair was repeated in the population: 2, 4, or 7 times). We found that whereas those alleles significantly correlated with performance on the conflict network, they correlated with neither latency nor other attentional networks. Based on these findings, we reported on two genes whose alleles impart different levels of efficiency in resolving conflict, the DRD4 and the MAOA (Fossella et al., 2002). Examining small subsets (i.e., 6–8 individuals per group) of this 200-person cohort, we looked at attention network test (ANT) performance in the

scanner and found significant differences between people with the two alleles in the ACC (i.e., the central node in this conflict and self-regulatory network; Fan et al., 2003). Similar to these findings with dopamine, alleles of cholinergic genes have been shown to relate to the orienting network, that is to the ability to carry out visual search tasks, which involve a high level of orienting (Parasuraman, Greenwood, & Sunderland, 2002). Thus, this approach permits relating the behavioral differences to the actual underlying networks. In fact, another gene, encoding for catechol-O-methyltransferase (COMT) and involved in the catecholamine metabolism, has been recently shown to relate to this network (Diamond, Briand, Fossella, & Gehlbach, 2004).

With a few exceptions (de Chaldee et al., 2001; Norton et al., 2002), COMT has also been shown to relate to the abnormalities of schizophrenia (Bilder et al., 2002; Bray et al., 2003; C. Chen et al., 1996; X. Chen, Wang, O'Neill, Walsh, & Kendler, 2004; Egan et al., 2001; Fan et al., 2002; Kotler et al., 1999; Li et al., 1996; C. Matsumoto, Shinkai, Hori, Ohmori, & Nakamura, 2004; M. Matsumoto et al., 2003; Nolan et al., 2000; Palmatier et al., 2004; Shifman et al., 2002, 2004; Strous et al., 2003; Tunbridge, Burnet, Sodhi, & Harrison, 2004; Wei & Hemmings, 1999; Wonodi, Stine, Mitchell, Buchanan, & Thaker, 2003; Zammit et al., 2004) and has been generally implicated in attentional and executive functions. Importantly, interpretation of data from exploratory genetic assays suggests that functional polymorphisms of the COMT gene relate to hypnotizability or susceptibility to hypnotic suggestion (Benjamin et al., 2000; Raz, 2004). Specifically, individuals homozygous for the valine allele (i.e., producing more COMT) tend to show lower hypnotizability scores (Raz, 2004).

COMT

COMT codes the substitution of valine (val) by methionine (met) at codon 158 (val158met). This substitution is associated with a difference in thermostability leading to a three- to four-fold reduction in the activity of the COMT enzyme (Lotta et al., 1995). The alleles are codominant so that individuals with the val/val genotype have the highest activity of COMT, those with the met/met genotype have the lowest activity of COMT, and heterozygous individuals are intermediate. The val158met genotypes have been linked to a number of behavioral diseases of complex etiology (Egan et al., 2001; Enoch, Xu, Ferro, Harris, & Goldman, 2003; Tiihonen et al., 1999).

As one of the enzymes that metabolizes catecholamines, COMT acts as a key modulator of dopaminergic but also adrenergic/noradrenergic neurotransmission (Cumming, Brown, Damsma, & Fibiger, 1992). Different levels of COMT activity conferred by val158met genotypes may then have important influences on functions regulated by these

neurotransmitters, including μ-opioid system responses associated with pain (Zubieta et al., 2003). Toward this end, effective hypnotic techniques for pain management have been known since the early 1800s, and it is therefore plausible that COMT might be implicated in hypnotizability. Furthermore, there is evidence that the effects of COMT appear to be mediated by attentional mechanisms and do not correlate with measures of role playing or social compliance (Bachner-Melman & Ebstein, 2002; Benjamin et al., 2000; Lichtenberg, Bachner-Melman, Ebstein, & Crawford, 2004).

Is There a Biological Marker for Hypnosis?

Herbert Spiegel likely was one of the first practitioners to suggest that hypnosis may have an innate component. Reporting empirical data, David Spiegel garnered at least nascent support for his father's theory by reporting a link between homovanilic acid and hypnotizability (Spiegel & King, 1992). More recently, morphometric data have attempted to delineate structural differences between the brains of high and low hypnotizable individuals (Horton, Crawford, Harrington, & Downs, 2004). Whereas researchers have gained some insight into the genetics of hypnotizability (Bauman & Bul', 1981; Rawlings, 1978), more recent efforts to establish viable relations between phenotype and genotype led Richard Ebstein, a reputable geneticist based in Israel, to examine a number of such correlations, including an association between COMT high/low enzyme activity polymorphism and hypnotizability. Using the Stanford Hypnotic Susceptibility Scale: Form C (SHSS:C; Weitzenhoffer & Hilgard, 1962), these data revealed a significant difference in hypnotizability between subjects who carried the val/met and val/val COMT genotypes (Benjamin et al., 2000; Ebstein, Bachner-Melman, & Lichtenberg, 1999). In their report, Ebstein and his colleagues encourage researchers to replicate their findings and one recent confirmation and extension surfaced unexpectedly following an independent research effort, spearheaded by Michael Posner, into the genetic bases of attention (Raz, 2004).

Whereas Ebstein et al. used the SHSS:C, they tested a group of 107 Israeli subjects using either English or, more often, Hebrew versions of the scale. Posner et al. obtained comparable results using the standard English version. For example, subject demographics indicate that whereas Ebstein et al. reported data from a wide range of moderately educated individuals (15.6 ± 2.4 years of education), Posner et al. provided a similar account from an age-uniform international community of highly educated adults (25.2 ± 2.1 years of education). Although Posner and his colleagues conducted their assay independently and learned about the findings of Ebstein et al. only in retrospect, it is rather remarkable that two autonomous studies, administered in

different contexts, yielded comparable results. Nonetheless, the larger question remains: based on these data, is there a genetic "fingerprint" for hypnotizability?

Correlation is not synonymous with causation; that certain genetic traits correlate with hypnotizability does not warrant the assumption that those polymorphisms cause hypnotizability. Indeed, the cause of hypnotizability may refer to more than its antecedent conditions. Reminiscent of the explosive growth in molecular biology, the fields of neuroscience and genetics also may need to be much further along in order to explicitly address these issues. However, an interim question can be carefully crafted to constructively dodge the subtle difference between *identity* and a *correlation*: what are the neuronal/genetic configurations that either correlate or are identical with the phenomenon of hypnosis? Answers to this question are likely to clarify the "explanatory fit" of a correlate versus identity. Understanding a theory does not cause the phenomena (e.g., understanding gravity does not cause us to fall) and understanding the neural/genetic correlates of hypnosis would at best constitute but one small step in understanding hypnosis (Churchland, 2002). Further, bottom-up explanations that usually apply to higher level systems often overzealously advocate for reduction (e.g., ontological reduction).

However, whereas science typically seeks a causal explanation to then have an ontological reduction, in the case of hypnosis as in the case of consciousness, a causal explanation is not likely to yield such an ontological reduction (Searle, 2000). As a case in point, the history of hypnosis research features the futile "state" versus "no-state" battle, which has long outlived its usefulness. The gist of this seminal conundrum, relevant to the context of this piece, can be summarized in a few short statements. The sociocognitive school of hypnosis purports that hypnosis does not exist as a unique "state" of consciousness and instead views it as learned behavior largely shaped by expectation and social pressure. Proponents of this school often reject the notion of hypnotizability as a stable trait and oppose the view regarding either hypnosis as involving specific neurophysiological correlates or hypnotizability as an innate and robust characteristic.

Data from the domain of attention suggest that training exercises can influence the developmental rate of attentional networks (Berger, Jones, Rothbart, & Posner, 2000; Klingberg et al., 2005; Klingberg, Forssberg, & Westerberg, 2002; Olesen, Westerberg, & Klingberg, 2004; Rueda, Rothbart, McCandliss, Saccomanno, & Posner, 2005). The use of these exercises on children with different genetic and attentional backgrounds gives the opportunity for specific studies of genetic-environmental interaction and individual differences (Fan et al., 2003; Posner, 2004; Sommer, Fossella, Fan, & Posner, 2003). Attentional training seems to improve the underlying neural networks involved in

conflict resolution and may even generalize to different tasks (Posner, 2004; Raz et al., 2005). Related findings also suggest that working memory training produced a significant improvement in motor performance and IQ on the nonverbal complex reasoning task of the Raven's progressive matrices (Klingberg et al., 2002, 2005; Olesen et al., 2004).

As neuroimaging is beginning to unravel the effects of practice and learning on brain substrates (Garavan, Kelley, Rosen, Rao, & Stein, 2004; Landau, Schumacher, Garavan, Druzgal, & D'Esposito, 2004), cumulative data suggest that modifications can be made to these attentional networks. However, there is no disagreement between these apparently disparate "nature versus nurture" findings: uninterrupted hypnotizability may indeed be stable (Piccione, Hilgard, & Zimbardo, 1989).

Importantly, whether the COMT gene identifies an individual's susceptibility to hypnosis or is simply a correlate of this trait is irrelevant. There appears to be a robust and significant relationship between the COMT gene and hypnotizability, such that identifying specific COMT polymorphisms may index individual hypnotizability.

CONCLUSION

In some cases, it has been possible to connect attention to underlying molecular events. This approach holds the promise of linking networks of attention to the genes and environmental events of early development but also to such traits as hypnotizability and potentially even placebo response. Work on the role of genes in attention and other cognitive networks underlying performance of humans is still in its infancy. These effects tend to be relatively small and probably no single gene is going to turn out to be the most important gene to determine individual differences in these networks. In fact, there are probably going to be a number of genes and possibly also a number of complex interactions. However, this work opens up an opportunity to examine not only individual differences but also how genes actually build the physical basis of the neural networks that we study. For example, the DRD4 gene, which is important for the conflict or executive network, has been recently knocked out in mice (Avale et al., 2004; Falzone et al., 2002; D. K. Grady & Kruzich, 2004). Mice missing the DRD4 gene produce predictable hyperactivity-related changes in their behavior and perform less exploration of their environment. More precise tests of attention are planned for these animals and it seems possible as we develop animal models of some of these networks that we will be able to tell how genes carry out the task of building the networks that are common among individuals as well as what alleles might account for individual differences.

As important as the genetic basis of individual difference is, however, it is not at all clear that most individual differences are due to variations in genetic alleles. In fact, we generally expect that differences between cultures may well depend upon socialization or culture-specific learning processes and not just upon genetic differences. Indeed, the nature-nurture debate applies equally well here, with the added insight that in recent time we have grown to appreciate more fully the strong effect genes impart.

Attention and hypnosis are anything but disjoint sets (Raz, 2004). Indeed, in this piece I have shown how the genetics of attention inform the genetics of hypnosis and can potentially characterize placebo response. Recent neuroscience findings drawing on the neural basis of attention and individual differences may further unravel this putative relationship.

References

Avale, M. E., Falzone, T. L., Gelman, D. M., Low, M. J., Grandy, D. K., & Rubinstein, M. (2004). The dopamine D4 receptor is essential for hyperactivity and impaired behavioral inhibition in a mouse model of attention deficit/hyperactivity disorder. *Molecular Psychiatry*, *9*, 718–726.

Bachner-Melman, R., & Ebstein, R. (2002). No link between hypnotizability and the Self-Monitoring Scale. *American Journal of Clinical Hypnosis*, *45*, 21–30.

Bauman, D. E., & Bul', P. I. (1981). Human inheritability of hypnotizability. *Genetika*, *17*, 352–356.

Baumeister, R. F., & Vohs, K. D. (2004). *Handbook of self regulation*. New York: Guilford.

Beauregard, M., Levesque, J., & Bourgouin, P. (2001). Neural correlates of conscious self-regulation of emotion. *Journal of Neuroscience*, *21*(18), RC165.

Benjamin, J., Osher, Y., Lichtenberg, P., Bachner-Melman, R., Gritsenko, I., Kotler, M., et al. (2000). An interaction between the catechol O-methyltransferase and serotonin transporter promoter region polymorphisms contributes to tridimensional personality questionnaire persistence scores in normal subjects. *Neuropsychobiology*, *41*, 48–53.

Berger, A., Jones, L., Rothbart, M. K., & Posner, M. I. (2000). Computerized games to study the development of attention in childhood. *Behavior Research Methods, Instruments, & Computers*, *32*, 297–303.

Bilder, R. M., Volavka, J., Czobor, P., Malhotra, A. K., Kennedy, J. L., Ni, X., et al. (2002). Neurocognitive correlates of the COMT Val(158)Met polymorphism in chronic schizophrenia. *Biology of Psychiatry*, *52*, 701–707.

Bray, N. J., Buckland, P. R., Williams, N. M., Williams, H. J., Norton, N., Owen, M. J., et al. (2003). A haplotype implicated in schizophrenia susceptibility is associated with reduced COMT expression in human brain. *American Journal of Human Genetics*, *73*, 152–161.

Bush, G. (2004). Multimodal studies of cingulate cortex. In M. I. Posner (Ed.), *Cognitive Neuroscience of Attention* (pp. 207–218). New York: Guilford.

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Science*, *4*, 215–222.

Chen, C. H., Lee, Y. R., Liu, M. Y., Wei, F. C., Koong, F. J., Hwu, H. G., et al. (1996). Identification of a BglI polymorphism of catechol-O-methyltransferase (COMT) gene, and association study with schizophrenia. *American Journal of Medical Genetics*, *67*, 556–559.

Chen, X., Wang, X., O'Neill, A. F., Walsh, D., & Kendler, K. S. (2004). Variants in the catechol-o-methyltransferase (COMT) gene are associated with schizophrenia in Irish high-density families. *Molecular Psychiatry*, *9*, 962–967.

Churchland, P. S. (2002). *Brain-wise: Studies in neurophilosophy*. Cambridge, MA: MIT Press.

Corbetta, M., Kincade, J. M., Ollinger, J. M., McAvoy, M. P., & Shulman, G. L. (2000). Voluntary orienting is dissociated from target detection in human posterior parietal cortex. *Natural Neuroscience*, *3*, 292–297.

Cumming, P., Brown, E., Damsma, G., & Fibiger, H. (1992). Formation and clearance of interstitial metabolites of dopamine and serotonin in the rat striatum: An in vivo microdialysis study. *Journal of Neurochemistry*, *59*, 1905–1914.

de Chaldee, M., Corbex, M., Campion, D., Jay, M., Samolyk, D., Petit, M., et al. (2001). No evidence for linkage between COMT and schizophrenia in a French population. *Psychiatry Research*, *102*, 87–90.

Diamond, A., Briand, L., Fossella, J., & Gehlbach, L. (2004). Genetic and neurochemical modulation of prefrontal cognitive functions in children. *American Journal of Psychiatry*, *161*, 125–132.

Ding, Y. C., Chi, H. C., Grady, D. L., Morishima, A., Kidd, J. R., Kidd, K. K., et al. (2002). Evidence of positive selection acting at the human dopamine receptor D4 gene locus. *Proceedings of the National Academy of Science USA*, *99*, 309–314.

Ebstein, R. P., Bachner-Melman, R., & Lichtenberg, P. (1999). Genetic and cognitive factors in hypnotizability: Association between the low enzyme activity catechol O- methyl transferase (COMT) MET allele and high hypnotizability. *Molecular Psychiatry*, *4*(240), Suppl.

Egan, M. F., Goldberg, T. E., Kolachana, B. S., Callicott, J. H., Mazzanti, C. M., Straub, R. E., et al. (2001). Effect of COMT Val108/158 Met genotype on frontal lobe function and risk for schizophrenia. *Proceedings of the National Academy of Science USA*, *98*, 6917–6922.

Enoch, M. A., Xu, K., Ferro, E., Harris, C. R., & Goldman, D. (2003). Genetic origins of anxiety in women: A role for a functional catechol-O-methyltransferase polymorphism. *Psychiatric Genetics*, *13*, 33–41.

Falzone, T. L., Gelman, D. M., Young, J. I., Grandy, D. K., Low, M. J., & Rubinstein, M. (2002). Absence of dopamine D4 receptors results in enhanced reactivity to unconditioned, but not conditioned, fear. *European Journal of Neuroscience*, *15*, 158–164.

Fan, J., Fossella, J., Sommer, T., Wu, Y., & Posner, M. I. (2003). Mapping the genetic variation of executive attention onto brain activity. *Proceedings of the National Academy of Science USA*, *100*, 7406–7411.

Fan, J., McCandliss, B. D., Sommer, T., Raz, A., & Posner, M. I. (2002). Testing the efficiency and independence of attentional networks. *Journal of Cognitive Neuroscience*, *14*, 340–347.

Fan, J., Wu, Y., Fossella, J. A., & Posner, M. I. (2001). Assessing the heritability of attentional networks. *BMC Neuroscience*, *2*, 14.

Fan, J. B., Chen, W. Y., Tang, J. X., Li, S., Gu, N. F., Feng, G. Y., et al. (2002). Family-based association studies of COMT gene polymorphisms and schizophrenia in the Chinese population. *Molecular Psychiatry*, *7*, 446–447.

Fossella, J., Sommer, T., Fan, J., Wu, Y., Swanson, J. M., Pfaff, D. W., et al. (2002). Assessing the molecular genetics of attention networks. *BMC Neuroscience*, *3*, 14.

Garavan, H., Kelley, D., Rosen, A., Rao, S. M., & Stein, E. A. (2004). Practice-related functional activation changes in a working memory task. *Microscopy Research and Technique*, *51*, 54–63.

Grady, D. K., & Kruzich, P. J. (2004). A molecular genetic approach to the neurobiology of attention utilizing dopamine receptor-deficient mice. In M. I. Posner (Ed.), *Cognitive neuroscience of attention* (pp. 260–268). New York: Guilford.

Grady, D. L., Chi, H. C., Ding, Y. C., Smith, M., Wang, E., Schuck, S., et al. (2003). High prevalence of rare dopamine receptor D4 alleles in children diagnosed with attention-deficit hyperactivity disorder. *Molecular Psychiatry*, *8*, 536–545.

Hofbauer, R. K., Rainville, P., Duncan, G. H., & Bushnell, M. C. (2001). Cortical representation of the sensory dimension of pain. *Journal of Neurophysiology*, *86*, 402–411.

Hopfinger, J. B., Buonocore, M. H., & Mangun, G. R. (2000). The neural mechanisms of top-down attentional control. *Natural Neuroscience*, *3*, 284–291.

Horton, J. E., Crawford, H. J., Harrington, G., & Downs, J. H., 3rd (2004). Increased anterior corpus callosum size associated positively with hypnotizability and the ability to control pain. *Brain*, *127*(Pt 8), 1741–1747.

Klingberg, T., Fernell, E., Olesen, P. J., Johnson, M., Gustafsson, P., Dahlstrom, K., et al. (2005). Computerized training of working memory in children with ADHD—A randomized, controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, *44*, 177–186.

Klingberg, T., Forssberg, H., & Westerberg, H. (2002). Training of working memory in children with ADHD. *Journal of Clinical and Experimental Neuropsychology*, *24*, 781–791.

Kosslyn, S. M., Thompson, W. L., Costantini-Ferrando, M. F., Alpert, N. M., & Spiegel, D. (2000). Hypnotic visual illusion alters color processing in the brain. *American Journal of Psychiatry*, *157*, 1279–1284.

Kotler, M., Barak, P., Cohen, H., Averbuch, I. E., Grinshpoon, A., Gritsenko, I., et al. (1999). Homicidal behavior in schizophrenia associated with a genetic polymorphism determining low catechol O-methyltransferase (COMT) activity. *American Journal of Medical Genetics*, *88*, 628–633.

LaHoste, G. J., Swanson, J. M., Wigal, S. B., Glabe, C., Wigal, T., King, N., et al. (1996). Dopamine D4 receptor gene polymorphism is associated with attention deficit hyperactivity disorder. *Molecular Psychiatry*, *1*, 121–124.

Landau, S. M., Schumacher, E. H., Garavan, H., Druzgal, T. J., & D'Esposito, M. (2004). A functional MRI study of the influence of practice on component processes of working memory. *Neuroimage*, *22*, 211–221.

Li, T., Sham, P. C., Vallada, H., Xie, T., Tang, X., Murray, R. M., et al. (1996). Preferential transmission of the high activity allele of COMT in schizophrenia. *Psychiatric Genetics*, *6*, 131–133.

Lichtenberg, P., Bachner-Melman, R., Ebstein, R. P., & Crawford, H. J. (2004). Hypnotic susceptibility: Multidimensional relationships with Cloninger's Tridimensional Personality Questionnaire, COMT polymorphisms, absorption, and attentional characteristics. *International Journal of Clinical and Experimental Hypnosis*, *52*, 47–72.

Lotta, T., Vidgren, J., Tilgmann, C., Ulmanen, I., Melen, K., Julkunen, I., et al. (1995). Kinetics of human soluble and membrane-bound catechol O-methyltransferase: A revised mechanism and description of the thermolabile variant of the enzyme. *Biochemistry*, *34*, 4202–4210.

Manly, T., Anderson, V., Nimmo-Smith, I., Turner, A., Watson, P., & Robertson, I. H. (2001). The differential assessment of children's attention: The Test of Everyday Attention for Children (TEA-Ch), normative sample and ADHD performance. *Journal of Child Psychology and Psychiatry*, *42*, 1065–1081.

Marrocco, R. T., & Davidson, M. C. (1998). Neurochemistry of attention. In R. Parasuraman (Ed.), *The Attentional Brain* (pp. 35–50). Cambridge, MA: MIT Press.

Matsumoto, C., Shinkai, T., Hori, H., Ohmori, O., & Nakamura, J. (2004). Polymorphisms of dopamine degradation enzyme (COMT and MAO) genes and tardive dyskinesia in patients with schizophrenia. *Psychiatry Research*, *127*, 1–7.

Matsumoto, M., Weickert, C. S., Beltaifa, S., Kolachana, B., Chen, J., Hyde, T. M., et al. (2003). Catechol O-methyltransferase (COMT) mRNA expression in the dorsolateral prefrontal cortex of patients with schizophrenia. *Neuropsychopharmacology*, *28*, 1521–1530.

Nolan, K. A., Volavka, J., Czobor, P., Cseh, A., Lachman, H., Saito, T., et al. (2000). Suicidal behavior in patients with schizophrenia is related to COMT polymorphism. *Psychiatric Genetics*, *10*, 117–124.

Norton, N., Kirov, G., Zammit, S., Jones, G., Jones, S., Owen, R., et al. (2002). Schizophrenia and functional polymorphisms in the MAOA and COMT genes: No evidence for association or epistasis. *American Journal of Medical Genetics*, *114*, 491–496.

Ochsner, K. N., Bunge, S. A., Gross, J. J., & Gabrieli, J. D. (2002). Rethinking feelings: An fMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience*, *14*, 1215–1229.

Ochsner, K. N., Kosslyn, S. M., Cosgrove, G. R., Cassem, E. H., Price, B. H., Nierenberg, A. A., et al. (2001). Deficits in visual cognition and attention following bilateral anterior cingulotomy. *Neuropsychologia*, *39*, 219–230.

Olesen, P. J., Westerberg, H., & Klingberg, T. (2004). Increased prefrontal and parietal activity after training of working memory. *Natural Neuroscience*, *7*, 75–79.

Palmatier, M. A., Pakstis, A. J., Speed, W., Paschou, P., Goldman, D., Odunsi, A., et al. (2004). COMT haplotypes suggest P2 promoter region relevance for schizophrenia. *Molecular Psychiatry*, *9*, 859–870.

Parasuraman, R., Greenwood, P. M., & Sunderland, T. (2002). The apolipoprotein E gene, attention, and brain function. *Neuropsychology*, *16*, 254–274.

Parasuraman, R., Warm, J., & See, J. (1998). Brain systems of vigilance. In R. Parasuraman (Ed.), *Varieties of attention* (pp. 221–256). Cambridge, MA: MIT Press.

Piccione, C., Hilgard, E. R., & Zimbardo, P. G. (1989). On the degree of stability of measured hypnotizability over a 25-year period. *Journal of Personality and Social Psychology*, *56*, 289–295.

Posner, M. I. (2004). *Cognitive neuroscience of attention*. New York: Guilford.

Posner, M. I., & Raichle, M. E. (1996). *Images of mind (revised)*. Washington, DC: Scientific American Books.

Posner, M. I., & Rothbart, M. K. (1998). Attention, self-regulation and consciousness. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *353*, 1915–1927.

Rainville, P. (2002). Brain mechanisms of pain affect and pain modulation. *Current Opinions in Neurobiology*, *12*, 195–204.

Rainville, P., Duncan, G. H., Price, D. D., Carrier, B., & Bushnell, M. C. (1997). Pain affect encoded in human anterior cingulate but not somatosensory cortex. *Science*, *277*, 968–971.

Rainville, P., Hofbauer, R. K., Bushnell, M. C., Duncan, G. H., & Price, D. D. (2002). Hypnosis modulates activity in brain structures involved in the regulation of consciousness. *Journal of Cognitive Neuroscience*, *14*, 887–901.

Rawlings, R. M. (1978). *The genetics of hypnotisability*. Dissertation, University of New South Wales, Australia.

Raz, A. (2004). Atypical attention: Hypnosis and conflict reduction. In M. I. Posner (Ed.), *Cognitive neuroscience of attention* (pp. 420–429). New York: Guilford.

Raz, A. (2006a). Perspectives on the efficacy of antidepressants for child and adolescent depression. *PLoS Medicine*, *3*, e9.

Raz, A. (2006b). Individual differences and attentional varieties. *Europa Medicophysica*, *42*, 53–58.

Raz, A., & Buhle, J. (2006). Typologies of attentional networks. *Nature Reviews Neuroscience*, *7*, 367–379.

Raz, A., Fan, J., & Posner, M. I. (2005). Hypnotic suggestion reduces conflict in the human brain. *Proceedings of the National Academy of Science USA*, *102*, 9978–9983.

Raz, A., Fan, J., & Posner, M. I. (2006). Neuroimaging and genetic associations of attentional and hypnotic processes. *Journal of Physiology - Paris*, *99*, 483–491.

Raz, A., Kirsch, I., Pollard, J., & Nitkin-Kaner, Y. (2006). Suggestion reduces the Stroop effect. *Psychological Science*, *17*, 91–95.

Raz, A., Landzberg, K. S., Schweizer, H. R., Zephrani, Z. R., Shapiro, T., Fan, J., et al. (2003). Posthypnotic suggestion and the modulation of Stroop interference under cycloplegia. *Consciousness and Cognition*, *12*, 332–346.

Raz, A., Moreno-Íñiguez, M., Martin, L., & Zhu, H. (2007). Suggestion overrides the Stroop effect in highly hypnotizable individuals. *Consciousness and Cognition*, *16*, 331–338.

Rees, G., Russell, C., Frith, C. D., & Driver, J. (1999). Inattentional blindness versus inattentional amnesia for fixated but ignored words. *Science*, *286*, 2504–2507.

Robertson, I. H., Ward, T., Ridgeway, V., & Nimmo-Smith, I. (1996). The structure of normal human attention: The Test of Everyday Attention. *Journal of the International Neuropsychological Society*, *2*, 525–534.

Rothbart, M. K., & Bates, J. E. (1998). Temperament. In N. Eisenberg (Ed.), *Handbook of Child Psychology* (Vol. 3 Social, emotional, personality development, pp. 105–176). New York: Wiley.

Rueda, M. R., Posner, M. I., & Rothbart, M. K. (2004). Attentional control and self regulation. In R. F. Baumeister & K. D. Vohs (Eds.), *Handbook of self-regulation* (pp. 283–300). New York: Guilford.

Rueda, M. R., Rothbart, M. K., McCandliss, B. D., Saccomanno, L., & Posner, M. I. (2005). Training, maturation, and genetic influences on the development of executive attention. *Proceedings of the National Academy of Science USA*, *102*, 14931–14936.

Searle, J. R. (2000). Consciousness. *Annual Review of Neuroscience*, *23*, 557–578.

Shifman, S., Bronstein, M., Sternfeld, M., Pisante, A., Weizman, A., Reznik, I., et al. (2004). COMT: A common susceptibility gene in bipolar disorder and schizophrenia. *American Journal Medical Genetics*, *128B*, 61–64.

Shifman, S., Bronstein, M., Sternfeld, M., Pisante-Shalom, A., Lev-Lehman, E., Weizman, A., et al. (2002). A highly significant association between a COMT haplotype and schizophrenia. *American Journal of Human Genetics*, *71*, 1296–1302.

Sommer, T., Fossella, J. A., Fan, J., & Posner, M. I. (2003). Inhibitory control: Cognitive subfunctions, individual differences and variation in dopaminergic genes. In I. Reinvang, M. W. Greenlee, & M. Herrmann (Eds.), *The cognitive neuroscience of individual differences — New perspectives* (pp. 27–44). Oldenburg, Germany: Bibliotheks- and Informations system der Universitat Oldenburg.

Spiegel, D., & King, R. (1992). Hypnotizability and CSF HVA levels among psychiatric patients. *Biological Psychiatry*, *31*, 95–98.

Strous, R. D., Nolan, K. A., Lapidus, R., Diaz, L., Saito, T., & Lachman, H. M. (2003). Aggressive behavior in schizophrenia is associated with the low enzyme activity COMT polymorphism: A replication study. *American Journal of Medical Genetics*, *120B*, 29–34.

Swanson, J., Oosterlaan, J., Murias, M., Schuck, S., Flodman, P., Spence, M.A., et al. (2000). Attention deficit/hyperactivity disorder children with a 7-repeat allele of the dopamine receptor D4 gene have extreme behavior but normal performance on critical neuropsychological tests of attention. *Proceedings of the National Academy of Science USA*, *97*, 4754–4759.

Swanson, J., Posner, M., Fusella, J., Wasdell, M., Sommer, T., & Fan, J. (2001). Genes and attention deficit hyperactivity disorder. *Current Psychiatry Reports*, *3*, 92–100.

Swanson, J. M., Flodman, P., Kennedy, J., Spence, M. A., Moyzis, R., Schuck, S., et al. (2000). Dopamine genes and ADHD. *Neuroscience & Biobehavioral Reviews*, *24*, 21–25.

Swanson, J. M., Sunohara, G. A., Kennedy, J. L., Regino, R., Fineberg, E., Wigal, T., et al. (1998). Association of the dopamine receptor D4 (DRD4) gene with a refined phenotype of attention deficit hyperactivity disorder (ADHD): A family-based approach. *Molecular Psychiatry*, *3*, 38–41.

Tiihonen, J., Hallikainen, T., Lachman, H., Saito, T., Volavka, J., Kauhanen, J., et al. (1999). Association between the functional variant of the catechol-O-methyltransferase (COMT) gene and type 1 alcoholism. *Molecular Psychiatry*, *4*, 286–289.

Tunbridge, E., Burnet, P. W., Sodhi, M. S., & Harrison, P. J. (2004). Catechol-o-methyltransferase (COMT) and proline dehydrogenase (PRODH) mRNAs in the dorsolateral prefrontal cortex in schizophrenia, bipolar disorder, and major depression. *Synapse*, *51*, 112–118.

Venter, J. C., Adams, M. D., Myers, E. W., Li, P. W., Mural, R. J., Sutton, G. G., et al. (2001). The sequence of the human genome. *Science*, *291*, 1304–1351.

Wager, T. D., Rilling, J. K., Smith, E. E., Sokolik, A., Casey, K. L., Davidson, R. J., et al. (2004). Placebo-induced changes in fMRI in the anticipation and experience of pain. *Science*, *303*, 1162–1167.

Wang, E., Ding, Y. C., Flodman, P., Kidd, J. R., Kidd, K. K., Grady, D. L., et al. (2004). The genetic architecture of selection at the human dopamine receptor D4 (DRD4) gene locus. *American Journal of Human Genetics*, *74*, 931–944.

Wegner, D. M. (2002). *The illusion of conscious will*. New York: Bradford Books/MIT Press.

Wei, J., & Hemmings, G. P. (1999). Lack of evidence for association between the COMT locus and schizophrenia. *Psychiatric Genetics*, *9*, 183–186.

Weitzenhoffer, A. M., & Hilgard, E. R. (1962). S*tanford Hypnotic Susceptibility Scale, Form C*. Palo Alto, CA: Consulting Psychological Press.

Wonodi, I., Stine, O. C., Mitchell, B. D., Buchanan, R. W., & Thaker, G. K. (2003). Association between Val108/158 Met polymorphism of the COMT gene and schizophrenia. *American Journal of Medical Genetics*, *120B*, 47–50.

Zammit, S., Jones, G., Jones, S. J., Norton, N., Sanders, R. D., Milham, C., et al. (2004). Polymorphisms in the MAOA, MAOB, and COMT genes and aggressive behavior in schizophrenia. *American Journal of Medical Genetics*, *128B*, 19–20.

Zubieta, J. K., Heitzeg, M. M., Smith, Y. R., Bueller, J. A., Xu, K., Xu, Y., et al. (2003). COMT val158met genotype affects mu-opioid neurotransmitter responses to a pain stressor. *Science*, *299*, 1240–1243.

Genetik und funktionelle Bildgebung von Aufmerksamkeit und Hypnotisierbarkeit ten den Plazeboeffekt erklären

Amir Raz

Zusammenfassung: Die Erforschung von Aufmerksamkeitsprozessen schlägt eine Brücke zwischen Psychologie und Neurowissenschaft und verdeutlicht die Zusammenhänge zwischen Gehirn und Verhalten. Wenigstens drei neuronale Module regeln Kontrollprozesse unter Verwendung verschiedener neuronanatomischer Substrate, Neuromodulatoren und psychologischer Prozesse. Geleitet von datenbasierten Gehirntheorien haben Forscher spezifische genetische Polymorphismen zu klar definierten Phänotypen in Bezug gesetzt, einschließlich solcher, die verschiedene attentionale Prozesse und Hypnose betreffen. Da Aufmerksamkeit kognitive und affektive Prozesse modulieren kann, wurde in letzter zeit damit begonnen, unter Verwendung von genetischen und bildgebenden Methoden individuelle Unterschiede zu untersuchen. Befunde genetischer Untersuchungen von Aufmerksamkeit und Hypnotisierbarkeit ermöglichen nun die Beantwortung von Fragen wie etwa nach den Unterschieden zwischen hoch- und gering hypnotisierbaren Individuen. Diese explorativen Befunde könnten ausgeweitet werden auf die Identifikation von plazeboempfänglichen Personen.

RALF SCHMAELZLE
University of Konstanz, Konstanz, Germany

On peut trouver une explication possible de l'effet placébo dans la génétique et la neuro-imagerie de l'attention et de l'hypnotisabilité

Amir Raz

Résumé: L'effort d'attention lie la psychologie et les techniques de la neuroscience, et démontre les relations qui existent entre le cerveau et le

comportement. En association avec les réseaux attentionnels, au moins trois modules cérébraux gouvernent les processus de contrôle, en faisant appel à la neuro-anatomie fonctionnelle asymétrique, à des neuromodulateurs et à des substrats psychologiques. Guidés par des théories relatives au cerveau fondées sur des données, des chercheurs ont établi un lien entre des polymorphismes génétiques spécifiques et des phénotypes bien définis, y compris ceux associés à différentes efficacités attentionnelles et à l'hypnose. Sachant que l'attention peut modifier les processus cognitifs et affectifs, des test génétiques, associés à des données de neuro-imagerie, ont permis de commencer à élucider la cause de différences individuelles. Les résultats de tests génétiques d'attention et d'hypnotisabilité permettent de répondre à des questions portant sur ce qui différencie des personnes hautement hypnotisables d'autres personnes moins facilement hypnotisables. Ces recherches pourraient déboucher sur le repérage de placébos réacteurs.

JOHANNE REYNAULT
C. Tr. (STIBC)

La genética e imagines neuronales de la atención y la hipnotizabilidad pueden dilucidar al placebo

Amir Raz

Resumen: La atención vincula a la psicología con las técnicas de la neurociencia y ejemplifica los nexos entre el cerebro y el comportamiento. Asociados con redes, atencionales, por lo menos 3 módulos cerebrales rigen los procesos de control utilizando distintos substratos neuroanatómicos, neuromoduladores, y psicológicos. Guiados por teorías cerebrales basadas en la investigación, los investigadores han relacionado poliformismos genéticos específicos a fenotipos bien-definidos, incluyendo los asociados con diferentes eficiencias atencionales y la hipnosis. Ya que la atención puede modular tanto procesos afectivos como cognitivos, las pruebas genéticas y los datos de las imagines cerebrales han comenzado a dilucidar diferencias individuales. Los hallazgos de las pruebas genéticas sobre la atención y la hipnotizabilidad preparan el terreno para contestar preguntas tales como cuánto difieren las personas con alta o baja hipnotizabilidad. Estos hallazgos exploratorios tal vez se puedan extender a la identificación de personas que responden al placebo.

ETZEL CARDEÑA
Lund University, Lund, Sweden