BETTER LONG-TERM OUTCOME FOR HYPNOTHERAPY THAN FOR CBT IN ADULTS WITH ADHD: RESULTS OF A SIX-MONTH FOLLOW-UP

SEPPO HILTUNEN[1], MAARIT VIRTA[1], ANITA SALAKARI[1], MERVI ANTILA[2], ESA CHYDENIUS[3], MARKUS KASKI[3], RISTO VATAJA[4], MATTI IIVANAINEN[3,5], MARKKU PARTINEN[6,7]

[1]Institute of Behavioural Sciences, University of Helsinki, Helsinki, Finland, [2]National Institute for Health and Welfare (THL), Finland, [3]Rinnekoti Research Centre, Finland, [4]Kellokoski Hospital, Finland, [5]Department of Child Neurology, University of Helsinki, Helsinki, Finland, [6]Helsinki Sleep Clinic, Vitalmed Research Centre, Helsinki, Finland, [7]Department of Clinical Neurosciences, University of Helsinki, Helsinki, Finland

ABSTRACT

This is the first controlled, randomized follow-up study investigating the effectiveness of hypnotherapy in treating adults with attention deficit hyperactivity disorder (ADHD). The aim was to compare the follow-up outcome of short cognitive behavioural therapy (CBT) and hypnotherapy. Six-month follow-up data were analyzed from 10 weekly individual treatments with cognitive hypnotherapy (n = 8) and CBT (n = 9). The treatment benefits were measured by self-report ADHD symptom scales (BADDS, SCL-16), psychiatric symptom scales (SCL-90, BDI-II), quality of life scale (Q-LES-Q) and independent evaluation (CGI). The treatment benefits remained with both treatment groups when measured with self-report ADHD symptom scales. However, the hypnotherapy and CBT groups differed statistically significantly from each other in general psychological well-being (SCL-90), anxiety (SCL-90) and depression (BDI-II), and almost significantly in ADHD symptoms (SCL-16) indicating better long-term outcome for hypnotherapy. Independent evaluators' ratings qualitatively supported the results. Hypnotherapy seems to be a usable method for treating ADHD in adults. The possible causes discussed for better long-term outcome for hypnotherapy than for CBT include stronger alliance in hypnotherapy, impact of deep relaxation to core symptoms and greater influence of hypnotherapy to emotional regulation. The generalization of the results requires further studies.

Keywords: ADHD, attention deficit hyperactivity disorder, hypnotherapy, cognitive behavioural therapy (CBT)

INTRODUCTION

Attention deficit hyperactivity disorder (ADHD) is a developmental neurobiological disability that emerges in childhood. In adults, the prevalence of ADHD has been estimated to be around 4% (Kessler et al., 2006). The major symptoms according to DSM-IV are inattention and/or impulsivity and hyperactivity (American Psychiatric Association, 1994). Problems in executive functioning, such as organizing and prioritizing, are typical. The core neurobiological impairments

can lead to long-lasting behavioural and emotional symptoms. Life-long experiences of failure or underachievement reinforce problems via negative cognitions and beliefs (Safren et al., 2004; Safren, 2006) and may often result in problems of self-esteem (Newark and Stieglitz, 2010). There is growing evidence that emotional dysregulation is also a core feature in adult ADHD (e.g. Mitchell et al., 2012; Retz et al., 2012). Psychiatric comorbidities such as anxiety, depression, bipolar disorder, personality disorders, and substance abuse are common (Biederman, 2004; Jacob et al., 2007; McGough et al., 2005; Sobanski et al., 2007; Sprafkin et al., 2007). Adults with ADHD often have considerable difficulties in managing finances, work, and/or social relationships (Goodman, 2007).

The management of ADHD in adulthood is based on pharmacological and psychological treatments. Both individual cognitive behavioural therapy (CBT) (Rostain and Ramsay, 2006; Safren et al., 2005; Safren et al., 2010; Wilens et al., 1999) and various group interventions (Emilsson et al., 2011; Hesslinger et al., 2002; Hirvikoski et al., 2011; Philipsen et al., 2007; Solanto et al., 2008; Solanto et al., 2010; Stevenson et al., 2002; Virta et al., 2008; Zylowska et al., 2008) have yielded promising results in treating ADHD adults. There is some evidence that relaxation training or hypnotherapy (Calhoun and Bolton, 1986; Copeland, 1980; Denkowski and Denkowski, 1984; Dunn and Howell, 1982; Raymer and Poppen, 1985) may be effective in children with attention deficit disorder or hyperactivity. Alladin (2008; 2009) has described an approach of cognitive hypnotherapy. It is theoretically based on CBT but also utilizes hypnotherapy techniques. Cognitive hypnotherapy has been effective in treatment of depression (Alladin and Alibhai, 2007), which is a common comorbidity in ADHD. To the best of our knowledge, only one previous controlled experiment has been published on either hypnotherapy alone, or on hypnotherapy combined with CBT in adults with ADHD (Virta et al., 2010a) with promising results.

There have been at least three group interventions where the ADHD participants have been followed after treatment. In the study by Stevenson et al. (2002), the improvement found in ADHD symptomatology and organizational skills after the intervention was still preserved after one year. In a study by our group (Salakari et al., 2010), treatment responders maintained most of the benefit during a six-month follow-up. Emilsson et al. (2011) found that the treatment effect still increased during a three-month follow-up. Of individual interventions, there has been only one follow-up study where Safren et al. (2010) found that responders and partial responders of 12 sessions CBT maintained their treatment gain during a 12-month follow-up.

In our previous papers (Virta et al., 2010a; Virta et al., 2010b), we investigated the utility and efficacy of novel short-treatment procedures, individual CBT and hypnotherapy, specifically tailored for treating adult ADHD. It was found that both treatments resulted in reduced self-reported ADHD symptoms. There was no difference in symptom reduction between CBT and hypnotherapy groups (Virta et al., 2010a). Here, our aim was to further evaluate the outcome of these therapy modalities by determining (a) whether the improvement was still maintained during the six-month follow-up and (b) whether there was a difference in efficacy between the two treatments in follow-up.

METHOD

PARTICIPANTS

Participants were recruited by announcements in an ADHD magazine, in an adult ADHD internet discussion forum, and by informing local physicians and clinics specializing in treating ADHD in adults. The inclusion criteria were as follows: (1) 18–49 years of age, (2) ADHD diagnosis made by a physician, (3) no diagnosis of psychosis, severe depression, or paranoia, (4) deficits of attention, executive functions, or working memory found by neuropsychological evaluation, (5) no current alcohol dependency or drug use, (6) not retired, (7) no participation in our previous group rehabilitation study, (8) currently not undergoing any other psychological rehabilitation, and (9) no medication or medication that has been stable for at least three months. In total, 71 interested candidates contacted the researchers, 46 were accepted and 39 participated. For more details about included and excluded participants and recruiting in general see Virta et al. (2010a; 2010b).

The participants were originally randomized to three treatment groups (hypnotherapy, CBT, cognitive training) and to a control group. Participants in the control group didn't take part in the follow-up period of the study. The follow-up period was six months and the participants were evaluated after three months (T3) and after six months (T4) from the end of the treatment (T2). Because the participants of our previous studies (see Virta et al., 2010a; 2010b) gained benefit only from hypnotherapy and CBT treatments, these two interventions were selected for the follow-up. Originally, there were nine participants in hypnotherapy treatment and ten participants in CBT treatment, but two participants (one in each treatment group) didn't participate in the follow-up. Thus, there were eight follow-up participants in the hypnotherapy group, and nine follow-up participants in the CBT group. The demographic data of the groups at the beginning of the intervention are presented in Table 1.

In hypnotherapy group, six of the eight follow-up participants received medication for ADHD: five of them took methylphenidate and one took dextroamphetamine at the beginning of the treatment. There were no medication changes during the treatment. In follow-up, medication of one participant was changed twice: the dose of dextroamphetamine was decreased during the first three months and then increased back to the original level during the last three months. In the CBT group, five of the nine follow-up participants were receiving medication for ADHD at the beginning of the treatment, and all of them took methylphenidate. One participant ceased taking her medication during the treatment, and one added short-acting methylphenidate to the previous long-acting methylphenidate. During the follow-up, medication of four participants was changed twice: one participant's short-acting methylphenidate was first reduced during the first three months and then removed in the second three-month period, two participants ceased taking long-acting methylphenidate in the first three-month period and then returned back to the original level in the second three-month period, and one participant without medication started dextroamphetamine during the first three months but it was removed in the last three months. In addition, one participant's medication was changed only during the last three months of the follow-up: long-acting methylphenidate was removed.

The groups of hypnotherapy and CBT follow-up participants did not differ at the beginning of the treatment, as analyzed by an analysis of variance or chi-squared test (Fisher's exact test), in age, gender, education, work status, Wender Utah Rating Scale (WURS, see Ward

Table 1. Characteristics of the follow-up study participants at the beginning of the treatment.

	Hypnotherapy	CBT
Participants (*n*)	8	9
Age: mean (range)	32.1 (21–42)	39.0 (25–49)
Gender: male/female	3/5	3/6
Education: compulsory/additional [a]	2/6	1/8
Working or studying: yes /no[b]	6/2	4/5
ADHD medication (*n*)	6	5
Antidepressant medication (*n*)	1	2
Psychiatric comorbidity (*n*)	4	7
depression (*n*)	2[c]	6
anxiety (*n*)	3[c]	0
personality disorder (*n*)	1	1
WURS score: mean (SD)	50.9 (18.5)	53.1 (13.0)
Severity of ADHD (CGI): mean (SD)	3.6 (0.7)	3.8 (0.8)

a Compulsory = the participant had completed only lower secondary education (i.e. Finnish compulsory education)
b Working/studying yes = the participant was working (at least a half-time job) or studying
c Two participants had both depression and anxiety diagnosis

et al., 1993) score, severity of ADHD (measured by Clinical Global Impressions, CGI, at the baseline), number of participants having psychiatric comorbidity or number of participants having certain psychiatric diagnosis (all *p*s > 0.05).

The study was approved by the Ethics Committee of Helsinki University Central Hospital, Finland and performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki. All participants gave their written informed consent prior to participating in the study.

HYPNOTHERAPY

The hypnotherapy treatment, designed and implemented by Virta et al. (2010a), was theoretically grounded in cognitive hypnotherapy. It consisted of a set of hypnotherapeutic interventions.The themes of the treatment sessions were selected to cover the main ADHD symptoms set out in the DSM-IV diagnostic criteria (American Psychiatric Association, 1994), and by Brown (2000, 2005) when suitable for the hypnotherapy.

Before the hypnotherapy, participants were evaluated by a psychiatrist (R.V.) to ensure that there were no contraindications for hypnosis. None of the participants was excluded. Hypnotic susceptibility was also evaluated using the Finnish version (Kallio, 1996; Kallio and Ihamuotila, 1999) of the Harvard Group Scale of Hypnotic Susceptibility, Form A (HGSHS:A) (Shor and Orne, 1962). The mean susceptibility score was 5.9 (range from 2 to 10).

The hypnotherapy consisted of 10 weekly sessions led by a psychologist experienced in hypnosis and ADHD (M.V.). The themes and content of the sessions were: stillness/calming, motivation to change, attention, initiation of activities, memory, self-esteem, three individually chosen topics (e.g. fear of social situations, reducing impulsivity, anger management, second treatment of previous themes) and continuation of the process. See Virta et al. (2010a) for more details about the content of the sessions. Each session followed the same procedure: discussion of the preceding hypnotherapy session, discussion of the current theme, induction, hypnotherapy, and discussion. The duration of a session was 40 to 60 minutes.

CBT

CBT treatment was designed and implemented by Virta et al. (2010b). The themes of the treatment sessions were selected to cover the main ADHD symptoms set out in the DSM-IV diagnostic criteria (American Psychiatric Association, 1994), and by Brown (2000; 2005). The CBT consisted of 10 weekly sessions led by a psychologist experienced in ADHD and with training in CBT (A.S.). The themes and contents of the sessions were: treatment goals and symptoms of ADHD, attention, motivation and initiation of activities, organization and planning, stress management and relaxation, self-esteem, three individually chosen topics (e.g. memory techniques, managing impulsivity, anger management, second treatment of previous themes) and continuation of the process. The therapeutic procedures are discussed in more detailed in Virta et al. (2010b).

The psychologist followed a written manual (Virta et al., 2009) and used a whiteboard and written material to illustrate the most important points and tasks at hand in the therapy. Individually tailored homework was given at each semi-structured session. Each session followed the same procedure: discussion of the previous homework and theme, introduction and discussion of the new theme, and assignment of the new homework and distribution of the written material. The duration of a session was approximately 60 minutes.

OUTCOME MEASURES

Self-report questionnaires and independent evaluations were used as outcome measures. Data were collected before the treatment (T1), immediately after the treatment (T2), three months after the end of the treatment (T3), and six months after the end of the treatment (T4). The mean time elapsed between T2 and T3 (i.e. the first three-month follow-up period) self-report questionnaires was 94 days (range 77–105) for the hypnotherapy group and 89 days (range 77–99) for the CBT group. The mean time elapsed between T3 and T4 (i.e. the second three-month follow-up period) self-report questionnaires was 84 days (range 64–111) for the hypnotherapy group and 81 days (range 67–100) for the CBT group. T3 and T4 independent evaluations were collected within nine days of the collection of self-report questionnaires. The independent evaluator was a clinical psychologist experienced in adult ADHD (M.A.) who was blind to the study group of the participants.

For this follow-up study, we chose the ADHD symptom measures that had proved most sensitive in our previous studies (see Virta et al., 2010a; 2010b). The outcome measures for ADHD symptoms, psychiatric symptoms, quality of life and independent evaluation were:

Brown Attention Deficit Disorder Scale – Adult Version (BADDS) (Brown, 1996)

BADDS is a 40-item inventory from which we used the self-report version. From the BADDS, a total score and scores of the five sub-domains of activation, attention, effort, affect, and memory were derived. Higher scores indicate a greater impairment.

Symptom Check List (SCL-90) (Derogatis et al., 1973)

SCL-90 is a 90-item self-report scale for the measurement of psychiatric symptoms. Several subscales can be calculated, e.g. for anxiety and depression. Total scores and subscale scores were used in the analyses of the follow-up period. Moreover, a 16-item sum score (SCL-16) reflecting the characteristics prominent in ADHD (Hesslinger et al., 2002) was calculated from the SCL-90. The higher the scores the greater were the symptoms.

Beck Depression Inventory – Second Edition (BDI-II) (Beck et al., 1996)

BDI-II is a 21-item scale that evaluates current self-reported symptoms of depression. Higher scores reflect greater problems.

Quality of Life Enjoyment and Satisfaction Questionnaire (Q-LES-Q) (Endicott et al., 1993)

Q-LES-Q is a 93-item self-report scale, from which 91 items can be grouped into eight subscales that indicate: satisfaction with physical health, subjective feelings, work, household duties, school, leisure activities, social relationships, and general activities. We combined the work and school subscales into a work/study subscale, as was done earlier (Virta et al. 2010a; 2010b). If the participant gave both scores, the more important score was used (i.e. if the participant was working full-time and also taking some educational courses, the work score was used). Higher scores indicate greater enjoyment or satisfaction. The scores are reported as a percentage of the maximum score.

Clinical Global Impressions (CGI) (Guy, 1976)

CGI was completed by the independent evaluator. At T1, severity of ADHD was evaluated according to the CGI, which is a single seven-point rating scale of functioning varying from 1 = normal, not at all ill, to 7 = among the most extremely ill patients. At T2, T3 and T4, global improvement was assessed using a seven-point scale varying from 1 = very much improved, to 7 = very much worse (4 = no change). Each assessment was performed in comparison to the participant's preceding evaluation.

STATISTICAL ANALYSIS

Missing values on the questionnaires were substituted with that particular respondent's mean score. However, no replacements were made in Q-LES-Q as the scores were calculated as a percentage of the maximum score. If the participant's outcome scores were missing completely for T3 or T4, the participant was excluded from the analysis. Distribution properties of the variables were inspected visually and with Shapiro-Wilk tests. Parametric tests were chosen for the statistical analyses. The repeated measures ANOVA was performed to find time x group

interactions for outcome variables at follow-up. Where Mauchly's test indicated violation of the sphericity assumption, Greenhouse–Geisser corrected values were used. The effect sizes were quantified by partial eta squared (η_ρ^2). Paired samples *t*-tests were performed for both groups separately for comparing T2 versus T4 outcomes. Changes in CGI were analyzed using the chi-squared test (Fisher's exact test, χ^2).

RESULTS

Mean scores of the self-report measures for the hypnotherapy and the CBT groups are presented in Table 2.

To compare the hypnotherapy group with the CBT group during the follow-up, repeated measures ANOVA was performed (see Table 2). There was a significant Time × Group interaction in SCL-90 total score of overall psychiatric symptoms [$F(2,30) = 4.10$, $p = .03$, $\eta_\rho^2 = .215$], a significant interaction in BDI-II score of the depression symptoms [$F(2,30) = 3.34$, $p < .05$, $\eta_\rho^2 = .182$] and an almost significant in SCL-16 score of ADHD symptoms [$F(2,30) = 3.24$, $p = .053$, $\eta_\rho^2 = .178$]. There was no significant Time × Group interaction found in BADDS total scale or any of the BADDS subscales. However, there was a trend in BADDS Affect subscale [$F(2,30) = 1.96$, $p = .16$, $\eta_\rho^2 = .116$]. As seen from Table 2, Figure 1 and Figure 2, there was decreasing trend of the symptoms in the hypnotherapy group during the six-month follow-up. In CBT group, there was more variation in the treatment outcome during the follow-up, but qualitatively the symptoms seemed to increase during the second three-month period of the follow-up as compared to the preceding evaluation (see Table 2, Figure 1 and Figure 2). When inspecting the treatment gains of the whole treatment and follow-up period in BADDS subscales (i.e. (T4–T1)/T1), it was found that the groups differed most in the BADDS Affect subscale: CBT group had only 2.4% decrease of symptoms from T1 to T4 whereas hypnotherapy group had 14.1% decrease of symptoms.

To evaluate how the improvement gained from the treatment remained during follow-up, ADHD symptoms were compared between T2 and T4 for both groups separately. In the hypnotherapy group, there was a decrease of ADHD symptoms in SCL-16 [$t(7) = 3.01$, $p < .05$] and no change in BADDS total [$t(7) = 0.44$, $p > .05$]. Thus, ADHD symptoms decreased or did not increase during follow-up in hypnotherapy group (see Figures 1 and 2). In the CBT group, ADHD symptoms of SCL-16 appeared to increase between T2 and T4 qualitatively (see Figure 1), but the increase was not statistically significant [$t(8) = –1.41$, $p > .05$]. Also, there was not a statistically significant change in BADDS total scale between T2 and T4 in the CBT group [$t(8) = –0.11$, $p > .05$]. In BADDS total scale, there was no statistically significant change between T2 and T4 in the CBT group [$t(8) = –0.11$, $p > .05$]. Mean value of the BADDS total scale at T4 was about the same level as at T2, despite of the trend of symptom increase from T3 (see Figure 2). As a summary, treatment outcome as measured with ADHD symptoms appears more stable in the hypnotherapy group during follow-up.

In SCL-90 subscales, there was a significant Time × Group interaction in repeated measures ANOVA for the follow-up period (T2–T3–T4) in anxiety symptoms [$F(2,30) = 5.73$, $p = .01$, $\eta_\rho^2 = .28$], and almost significant interaction in depression [$F(2,30) = 3.23$, $p = .05$, $\eta_\rho^2 = .18$], hostility [$F(2,30) = 3.09$, $p = .06$, $\eta_\rho^2 = .17$] and psychotic symptoms [$F(2,30) = 3.45$, $p = .07$, $\eta_\rho^2 = .19$].

Mean scores of the SCL-90 depression subscale and BDI-II for the groups changed in quite similar way during the follow-up: in the CBT group, mean scores seemed to increase towards the end of the follow-up, whereas in the hypnotherapy group mean scores typically remained in the decreased level or even decreased more. For instance, when comparing BDI-II scores between T2 and T4 for both groups separately with a paired *t*-test, there was an almost statistically significant decrease in BDI-II scores in the hypnotherapy group [$t(7) = 2.16, p = .07$]. Between T1 and T4, the decrease was statistically significant [$t(7) = 2.55, p = .04$]. In the CBT group, on the contrary, BDI-II scores seemed to return to the pretreatment level (Figure 3). However, increase of BDI-II scores from T2 to T4 was not statistically significant [$t(8) = -1.89, p > .05$].

According to the independent evaluators' CGI ratings, three of the eight participants in the hypnotherapy group improved from T2 to T4 and none declined. In the CBT group, one individual of the nine participants improved from T2 to T4, and two declined. However, statistically the group differences were not significant (Fisher's exact test: $\chi^2 = 2.61, p > .05$).

DISCUSSION

At follow-up, ADHD symptoms of the hypnotherapy group either remained at the decreased post-treatment level (measured by BADDS total) or even continued to decrease from the post-treatment level (as measured by SCL-16). In the CBT group, no difference was found between the end-of-treatment ADHD symptoms and symptoms at six months follow-up, indicating persistence of treatment benefits as well. However, in CBT group, there was more individual variation and a general tendency towards symptom increase during the last three months of the follow-up. Thus, long-term treatment outcome of hypnotherapy was generally stronger and more stable than the outcome of CBT. In our previous studies (Virta et al. 2010a; 2010b), both CBT and hypnotherapy were effective in treating adults with ADHD. The CBT and the hypnotherapy groups did not differ in any of the outcome measures during the treatment period (Virta et al. 2010a).

The finding that ADHD symptoms might start to return during the follow-up is in line with the previous results of CBT interventions. To the best of our knowledge, only one individual CBT intervention study with a long follow-up period has been previously published. In this Safren et al. (2010) study, participants showing at least partial response to CBT, maintained their improvement at the follow-up assessments, up to nine months post-treatment. However, CBT participants had increasing scores of small magnitude in the self-report measure of ADHD symptoms during follow-up. The similar tendency, i.e. increase of ADHD symptoms during follow-up, has been found in CBT group intervention studies as well. In our previous study (Salakari et al. 2010), the improved participants had some increase of ADHD symptoms three months after the treatment. They, however, maintained improvement when compared to the pre-treatment level. In Stevenson et al. (2002) study with a one year follow-up, there was a slight decrease of ADHD symptoms at two months post-treatment and thereafter an increase of ADHD symptoms towards the post-treatment level when looking at the mean scores of ADHD symptoms. At the end of follow-up, the mean score of ADHD symptoms in the group still remained under the post-treatment level. In Emilsson et al. (2011) study, self-reported ADHD symptoms did not start to increase during three-month follow-up period, which matches well with the results of our current study at three-month posttreatment. Slight qualitative differences between the results

Table 2. Mean (standard deviation) scores for participants' self-ratings at T1 (before treatment), T2 (immediately after treatment), T3 (three months after treatment), and T4 (six months after treatment) with the results of Time x Group interaction in repeated measures ANOVA for the follow-up period (T2-T3-T4).

	Hypnotherapy (n = 8)				CBT (n = 9)				$F(2,30)$	p-value
	T1	T2	T3	T4	T1	T2	T3	T4		
BADDS										
Activation	17.8 (5.3)	14.4 (6.5)	15.3 (6.8)	14.9 (6.0)	20.2 (2.2)	17.4 (4.7)	15.3 (3.9)	17.2 (3.5)	0.94	ns
Attention	18.9 (4.9)	16.6 (5.7)	14.6 (7.6)	16.0 (6.5)	21.8 (3.1)	18.1 (5.3)	15.7 (5.2)	18.1 (5.5)	0.08	ns
Effort	14.8 (5.5)	13.5 (4.9)	11.5 (6.0)	11.6 (6.2)	18.2 (4.5)	15.9 (4.6)	13.7 (6.0)	14.4 (4.7)	0.05	ns
Affect	9.3 (4.0)	7.5 (3.8)	7.9 (4.4)	7.4 (2.9)	11.4 (2.2)	9.8 (2.9)	8.9 (4.0)	11.3 (4.4)	1.96	ns
Memory	12.9 (4.1)	10.6 (2.5)	11.3 (2.9)	9.9 (3.7)	11.8 (4.4)	10.1 (5.1)	9.4 (3.6)	10.8 (4.5)	1.85	ns
Total	73.5 (17.0)	62.6 (17.7)	60.5 (21.3)	59.8 (19.9)	83.4 (12.8)	71.3 (19.7)	63.0 (18.7)	71.9 (18.4)	0.69	ns
SCL-16	28.1 (12.4)	20.4 (10.0)	18.5 (9.7)	16.3 (8.8)	30.3 (7.5)	25.1 (5.6)	24.9 (11.6)	29.6 (12.6)	3.24	.05
SCL-90	87.6 (46.5)	62.0 (40.8)	56.6 (33.0)	51.6 (36.2)	92.3 (19.9)	78.7 (27.3)	80.9 (53.7)	106.4 (54.5)	4.10	.03
BDI-II	12.0 (10.8)	9.6 (10.9)	6.4 (4.6)	6.5 (7.4)	13.3 (5.8)	9.0 (8.8)	12.0 (10.6)	13.6 (6.9)	3.34	.05
Q-LES-Q										
General	57.0 (17.4)	65.1 (13.8)	67.8 (12.4)	70.6 (16.3)	56.4 (8.8)	63.0 (14.1)	60.0 (14.2)	57.3 (9.6)	1.37	ns
Work/study[a]	72.0 (20.5)	83.3 (17.8)	90.0 (9.7)	88.0 (13.0)	45.8b (23.1)[b]	75.2 (17.6)	78.3 (16.6)	71.5 (14.6)	0.77	ns

a Included the participants, which have the value in every three measures (T2, T3 and T4), n = 4 in hypnotherapy and n = 6 in CBT group. All the participants have Q-LES-Q Work scale here, since those who had Q-LES-Q study as their main score had missing values in some of the T2, T3 or T4 measures.
b n = 4

Figure 1. SCL-16 score for ADHD symptoms at T1 (before treatment), T2 (immediately after treatment), T3 (three months after treatment) and T4 (six months after treatment) for CBT (n=9) and hypnotherapy (n=8) groups

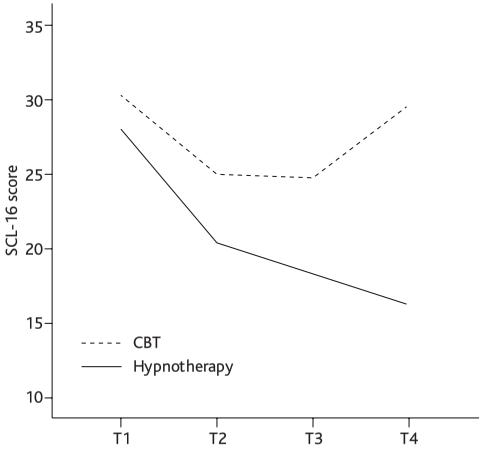


Figure 2. BADDS total score for ADHD symptoms at T1 (before treatment), T2 (immediately after treatment), T3 (three months after treatment) and T4 (six months after treatment) for CBT (n=9) and hypnotherapy (n=8) groups

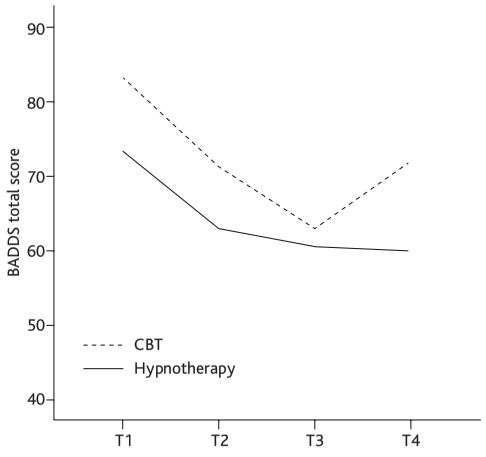
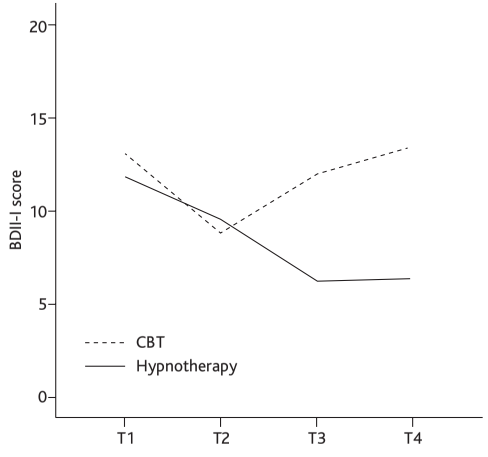


Figure 3. BDI-II score for depression symptoms at T1 (before treatment), T2 (immediately after treatment), T3 (three months after treatment) and T4 (six months after treatment) for CBT (n=9) and hypnotherapy (n=8) groups



in the CBT studies may be due to different ADHD symptom scales and due to different contents of the therapy.

Since there are no other controlled studies about hypnotherapy for treating adults with ADHD, the long-term outcome results are not available for comparison. In Kirsch et al. (1995) meta-analysis, CBT was compared with the same therapy supplemented by hypnosis. There was follow-up data available only for the treatment of obesity. When hypnosis was used, the biggest weight loss actually happened during six months after the treatment and the weight remained at reduced level at two year follow-up. Kirsch et al. (1995) speculated that advantages of adding the hypnosis to CBT treatment may increase over time regardless of the target disorder. Our study suggests that the treatment outcome in adult ADHD has become more stable with the hypnotherapy than CBT during the follow-up.

During six-month follow-up, the outcome of the treatments differed from each other to the favour of the hypnotherapy group in overall psychological well-being (SCL-90 total), depression (BDI-II), anxiety (SCL-90 anxiety) and almost statistically significantly in some of the ADHD symptoms (in SCL-16, not in BADDS). In the second three-month period of the follow-up, self-reported ADHD symptoms and self-reported psychiatric symptoms tended to increase in the CBT group, whereas in the hypnotherapy group the symptoms remained at decreased post-treatment level or even continued to decrease from that. This is in line with Alladin and Alibhai (2007) study where cognitive hypnotherapy and CBT were compared in treating depression. At one year follow-up, hypnotherapy was found to be more efficient than CBT. However, improvement in depression symptoms also remained in the CBT group during the whole one year follow-up. Our CBT was designed for treating ADHD, not for treating anxiety or depression, so this may have had an influence on the returning of psychiatric symptoms. The outcome of the treatments did not differ in quality of life at follow-up. As discussed in Virta et al. (2010a), it may be that Q-LES-Q scale is not sensitive enough for assessment of the adult ADHD population.

There was no statistically significant change in independent evaluator's ratings (CGI) during follow-up or between the groups in follow-up. However, there was a tendency that the improvement of the hypnotherapy participants remained better than in the CBT group. The limitations of the independent evaluation procedure are more widely discussed in Virta et al. (2010a; 2010b): the impairments may not be clearly apparent in a short discussion, participants may want to give a better impression, and blindness for the therapy method cannot always be secured in the assessment situation.

It is not clear from the literature why hypnotherapy yields longer-lasting improvement than CBT, but several possible reasons and mechanisms can be suggested. Since the outcome of the two treatments did not differ at the end of the actual treatment period, difference in efficiency emerged during the follow-up. Baker and Jensen (2011) have suggested that the use of hypnosis can lead to a more powerful and rapid alliance than with any other therapy method. The alliance, on the other hand, is known to correlate well with the outcome of therapy (Horvath and Symonds, 1991; Horvath and Bedi, 2002; Martin et al., 2000). Huynh et al. (2008) have discussed that in hypnotherapy one main goal is often to teach the attitude of hope within the context of mastery. Expectations of the patient have been recognized as one of the influencing factors in psychological treatments (Greenberg et al., 2006; Mondloch et al., 2001). Can expectations be higher with hypnotherapy than with CBT? At least, after the treatment our hypnotherapy participants had more belief than the CBT participants that

things in their life are going to get better in the future. They estimated this question with a 0–4 scale at each evaluation point from T1 to T4 (the group main effect [$F(1,15) = 10.73$, $p < .01$, $\eta_p^2 = .41$] in favour of the hypnotherapy group at the follow-up period (T2–T3–T4), whereas there was no difference in the scores before the treatment [$t(15) = .58$, $p > .05$]). Interestingly, if there is stronger effect in expectations in hypnotherapy than in CBT, it seems to last at least six months.

Hypnotherapy and CBT of our study were mainly designed for teaching new skills, new coping strategies and addressing secondary problems of ADHD, such as negative thoughts and beliefs. Hypnotherapy, with focused attention involved, may strengthen the new positive thoughts and beliefs more than CBT. In addition, the hypnotherapy may also have a direct impact on ADHD core symptoms, which is not similarly possible with CBT. We suggest here three possible factors that hypnotherapy may bring to the treatment of ADHD core deficits: First, the deep relaxation experience produced by hypnosis may influence directly in the hyperactivity and impulsivity symptoms. Such an experience, happening for the first time in the participant's life, may be an unforgettable and empowering experience (Alladin and Alibhai, 2007). Creating direct and compelling evidence for participants, that their subjective experience can be altered, might give rise to a new sense of hope (Alladin, 2012b). However, it is known from the previous research that relaxation alone without hypnosis and with psychoeducational support is not as effective as CBT for treating adults with ADHD (Safren et al., 2010).

Second, hypnosis itself may exercise the brain's attentional and executive functions. Hypnotic induction and suggestions contain focusing or narrowing the attention. We suggest that this may serve directly as a rehabilitation exercise for the brain areas involved in attentional functions, which are impaired in ADHD. In addition, hypnosis has a capability of reducing the activation of the anterior cingulate cortex (Kihlstrom, 2013). Anterior cingulate cortex is crucial for executive functioning, inhibitory control monitoring, target detection and error processing (Schneider et al., 2006), in which functions the ADHD adults typically have problems. One main difference between the CBT and hypnotherapy is that the hypnotherapy has traditionally been concerned with insight and unconscious reframing, while CBT concentrates on cognitive restructuring (Alladin, 2012a). Therefore, hypnotherapy may have a broader influence in different brain functions and regions than is possible with CBT.

Third, hypnosis might influence emotional regulation. Interestingly, the difference in the long-term outcome of hypnotherapy and CBT was most evident in the area of emotional regulation, as measured with BADDS Affect subscale. Lately, it has been discussed that there is enough evidence for the emotional dysregulation to be included to the core deficits in adult ADHD (Retz et al., 2012). The emotions play a role in all executive functions: they can influence attention, memory, decision making, planning and even muscle tension. In general, when the emotional regulation is improved, it may have a positive influence on all other executive functions as well. From the follow-up point of view, when the improvement in emotional regulation remains, it may support to maintain improvement gained in other executive functions as well. Surely, emotional dysregulation have a role on depression and anxiety symptoms, which are common comorbidities of ADHD.

In our current study, the CBT participants had several medication changes during follow-up. When inspecting the follow-up ADHD symptoms and depression symptoms of the five CBT participants with medication changes and the symptoms of the rest of the CBT participants, it

seemed that both subgroups had a quite similar increasing trend of symptoms during the last three months of the follow-up. Thus, medication changes did not seem to explain the slight increase of the symptoms during the last three months. Treatment groups in our study did not differ statistically from each other at pre-treatment, but participants in the CBT group had slightly more ADHD symptoms in general. Also, qualitative differences in comorbidity between the participants in the treatment groups may have influenced the long-term outcome, especially when short interventions such as ours are used.

Our study has limitations that should be considered when interpreting the results. First, the sample consisted only eight participants in the hypnotherapy group and nine in the CBT group, which are small numbers. Thus, the results of the follow-up must be considered with caution. Second, there was no control group during the follow-up period. The third limitation is the severity of the ADHD symptoms of the participants. In CGI, participants were rated mildly to markedly ill, thus leaving the most extreme cases missing. Also, the recruitment of the participants may have caused some bias to the more motivated and less severely disabled ADHD adults to participate in the study. Thus, the results cannot be generalized to the whole ADHD population.

Despite these limitations, our study has many strengths. All diagnoses were made by a specialist who is familiar with neuropsychiatric disorders, and they were duly verified. The outcome measures were wide-ranging (self-report questionnaires, independent evaluations), and participants were randomly allocated to different treatment groups. Both treatments are originally designed to take account the characteristics of ADHD.

It may be a sum of all the factors discussed about that leads to the longer-lasting improvement in the hypnotherapy group. According to Rostain and Ramsay (2006), the pharmacotherapy is a 'bottom up' treatment for the core symptoms of ADHD and the CBT provides a 'top-down' psychosocial approach for addressing functional problems, modifying negative thought patterns and developing new coping strategies. In a way, hypnotherapy may contain elements of both 'bottom up' and 'top-down' approaches.

Since both interventions had long-lasting treatment effects, our study supports the utility of psychological interventions in treatment of adult ADHD. The findings of this study suggest that hypnotherapy yields longer-term benefits than CBT in treating adults with ADHD. Since this is the first follow-up study on hypnotherapy for treating ADHD in adults, further studies with larger sample sizes, longer treatments and longer follow-ups are needed to generalize the results.

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Correspondence to Maarit Virta, Institute of Behavioural Sciences, Division of Cognitive and Neuropsychology, P.O.Box 9, 00014 University of Helsinki, Finland
Email: maarit.virta@helsinki.fi
Phone: +358 2941 29535

INHIBITION AND DISINHIBITION IN HYPNOSIS

PETER L. N. NAISH

The Sackler Centre for Consciousness Science, University of Sussex

ABSTRACT

This small-scale, quickly-administered study was designed as an initial exploration of the extent to which general inhibition plays a part in hypnosis. Some models of hypnosis emphasize the strategic use of prefrontal disinhibition, while others suggest that frontal regions become less involved overall, so that disinhibition is general rather than focused. A stop signal reaction time task was used to assess the level of inhibition available in high and low susceptible participants, both waking and hypnotized. Results implied that hypnosis increases inhibition for the highly susceptible. From this it is concluded that any disinhibition must be localized and strategic.

Keywords: consciousness, altered states, hypnotic susceptibility, inhibition, stop signal reaction time

INTRODUCTION

The advent of brain scanning has been of assistance in showing that hypnosis is more than just a simple blend of compliance to suggestions and 'imagining along' with them (e.g. Naish, 2013). As a result, although there may still be some dissenting voices (see Kirsch, et al., 2011) it has become reasonable to consider hypnosis to be an altered state of consciousness. However, the various forms of brain scanning and monitoring available to us are, as yet, unable to provide full explanations of consciousness itself, so to describe hypnosis as some kind of modification to consciousness is not entirely helpful.

What we do know, of both hypnosis and consciousness more generally, is that the prefrontal cortex (PFC) plays a key role. The work of van Gaal and Lamme (2012) has helped to draw together and explain several significant observations. First, it is known that a briefly displayed word, shown too quickly for us consciously to know what we saw, can nevertheless influence our behaviour; clearly, analysis has taken place outside conscious awareness. Brain scanning in this situation shows that activity spreads forward through the brain, starting at the back at the onset of the stimulus, in the visual cortex, then moving through the processing sequence, getting closer to the front of the brain. Crucially, when the stimulus is very brief, the activity never reaches as far forward as the PFC. Interestingly, a slightly longer-lasting stimulus that does result in PFC activity still does not necessarily result in conscious awareness. That seems to occur only after the PFC initiates backward-acting signals. These appear to result in the phase-locking of activity pertinent to the experience (Doesburg et al., 2008).

Phase-locking is analogous to having different sections of an orchestra (but not all of it) following the conductor. Because the brain is highly modular the various aspects of an experience

will be represented by activity in different regions; for example the round shape of an orange, its colour and its smell will each be recognized in different brain areas. Nevertheless, the conscious experience is of a unified whole and scanning reveals that oscillations in those different regions remain locked in step. The synchronization is achieved by long-range feedback via neural tracts originating in the PFC; without this there seems to be no conscious awareness.

Imagining and remembering appear to utilize the same feedback pathways from the PFC. In this case, rather than controlling activity caused by external stimuli, the PFC is actually generating activity. One theory of why humans developed consciousness is based upon this ability to re-activate regions of brain that were no longer receiving external information. Baumeister & Masicampo (2010) proposed that this was very valuable to a social animal, since it would permit the repeated 're-running' of a social interaction, so enabling the ruminator to fine-tune future encounters. Scanning someone who is remembering or visualizing reveals activity that is very similar to that observed when the stimuli are actually present. Significantly, the similarity is much enhanced when visualization occurs in hypnosis (Szechtmanet et al., 1998; Kosslyn, et al., 2000). Comparison with the experiences of schizophrenia can help to explain why hypnosis may enhance the sense of reality.

Schizophrenia patients exhibit lower than normal levels of phase locking (Haig et al., 2000) and this appears to be the result of poor connectivity with the PFC (Lawrie et al., 2002). Perhaps an even more significant deficit is that, along with the excitatory links used for phase-locking or visualizing, the patients lose inhibitory connections too (Shergill et al., 2005). These are used to reduce activity in areas of the brain that are not contributing to conscious awareness (Buehlmann & Deco, 2008) especially those resulting from the person's own actions. Thus, when a healthy person moves their arm, although the receptors in the joints will be stimulated, little corresponding activity is seen in the brain. This low response is due to the arm movements being self-initiated, so making it possible to predict the potential neural responses and inhibit them. In contrast, if a person has their arm moved by someone else, then prediction is not possible and a clear, uninhibited signal is observed in the brain. Schizophrenia patients fail to inhibit self-generated signals so, presumably as a consequence, have the feeling that their actions are being controlled by external forces. Brain scanning hypnotic 'highs' reveals that, as with the patients, they exhibit reduced phase-locking (Fingelkurts et al., 2007). Moreover, when experiencing things 'happening by themselves', such as in the arm levitation test, they fail to inhibit the signals, just as if outside forces were moving the arm (Blakemoreet al. 2003).

The foregoing is a clear indication of the importance of the PFC in the generation of both normal and atypical conscious experiences. Although a more detailed understanding of the processes involved would permit a more precise description of hypnotic mechanisms, nevertheless it seems reasonable to claim that changes in the excitatory and inhibitory behaviour of the PFC underpin the experiences of hypnosis. Since people exhibit a range of hypnotic abilities it may be supposed that the extent of the changes in PFC activity varies between individuals. Plausibly, the type of change could differ too, with some people perhaps very good at reducing inhibition, so making actions feel involuntary, while others may be better at manipulating excitatory signals in order to generate hallucinations. These conjectures imply considerable mental versatility, with PFC activity being finely tuned to achieve the desired outcome. As Spanos frequently emphasized (e.g. Spanos, 1986) people in hypnosis do what they believe people in hypnosis are meant to do. Thus, the PFC control is strategic and must involve a good deal of the executive, planning function associated with that region.

In contrast to the above, a number of researchers have associated hypnotic effects with a reduction in executive function, characterizing this as a state of hypofrontality. Semmens-Wheeler, Dienes and Duka (2013) present a good summary of the evidence suggesting a reduction of PFC involvement, and contribute their own finding that alcohol increases hypnotic susceptibility. Participants in their study, who had consumed a quantity of alcohol equivalent to approximately 500 ml of wine, rated their hypnotic experiences as more intense than those who had consumed placebo. Other tests confirmed that the alcohol had, as expected, impaired frontal lobe activity, so the authors concluded that hypnosis is associated with a reduction in frontal involvement. However, it is difficult to generalize from this finding, for there are a number of caveats, most of which the authors acknowledge. Importantly, it is not entirely clear what exactly is being facilitated or disrupted by the alcohol. It is obvious that increasing levels of intoxication would eventually impair hypnotic responding, so there is presumably some optimum level which permits the best hypnotic response. Whether that level impairs excitatory and inhibitory processes equally is unclear. It is generally assumed that alcohol disinhibits, and one of the tests used by Semmens-Wheeler et al. (2013) did confirm that participants in the alcohol condition had a reduced ability to inhibit. On the basis of the account given earlier, this might be expected to permit inappropriate activity in the brain, leading to the erroneous experience that the hypnotic effects were happening without volition. However, a generalized reduction in inhibition would tend to raise the overall activity across the brain; that is not what brain scanning in hypnosis shows, at least not for hypnotic 'highs'. McGeown et al. (2009) showed that the so-called default mode activity (the background activity when there is no specific task to be performed) was reduced in 'highs' when hypnotized. If these participants sometimes used disinhibition, as the results of Blakemore et al. (2003) would predict, then they must have been able to deploy the strategy very selectively. This is not what would be expected when alcohol was used as the disinhibiting agent. It should be noted that the McGeown et al. participants were 'highs', whereas those in the Semmens-Wheeler et al. study were deliberately selected to be only moderately susceptible, to allow for either enhanced or impaired susceptibility to be registered. It is not known whether people who are moderately susceptible simply do less of whatever 'highs' do, or whether the latter have a different way of achieving their responsiveness. The experiment to be reported here attempted to determine what 'highs' were doing – specifically looking for evidence of general disinhibition.

The test used was similar to the one employed by Semmens-Wheeler et al. (2013) when checking that alcohol had reduced the ability to inhibit behaviour; it is called the Stop Signal Reaction Time (SSRT) task. A participant has to respond as quickly as possible to a signal, but has to withhold the response if a second signal indicates *Stop!* That stop signal is delivered a little later than the first trigger signal, and if it is too much later the participant is unable to prevent him- or herself from responding. Those who have a reduced ability to inhibit behaviour, for example through intoxication or having a condition such as obsessive compulsive disorder, require the stop signal to be presented with relatively little delay. In this experiment participants were to be tested in and out of hypnosis, to determine whether their levels of inhibition changed between the two states.

METHOD

PARTICIPANTS

Fourteen students attending an Open University Summer School volunteered to take part, having been selected following testing with the Harvard Group Scale of Hypnotic Susceptibility (Shor & Orne, 1962). There were seven 'highs' and seven 'lows', each group comprising six female and one male participant. All were fully informed about the nature of the study, although not its precise purpose; they were merely told that it was 'looking to see in what ways the brain performed differently in hypnosis'. Since the testing diverted students from their primary objective at Summer School the test procedures were designed to be as brief as possible.

STIMULUS PRESENTATION

Stimuli comprised large block arrows, pointing either left or right, presented on a laptop computer. Participants responded by pressing a left- or right-hand key on the computer keyboard. Presentation began with only the shaft of the arrow appearing in the centre of the screen, comprising a broad, black, horizontal line. After a random delay of between 1 and 2 seconds a black arrow head was added to one end or the other, with equal probability. At this point reaction timing began. Participants were instructed to respond to the direction as quickly as possible, but on 30% of presentations the black arrow turned red. This was the stop signal, and if it occurred participants were not to make a response.

Initially, the delay between arrowhead presentation and the stop signal was 200 ms, but this delay tracked the success or otherwise of the participant in withholding the response. After two consecutive successes the delay was lengthened by 64 ms, but after two consecutive inhibition failures the change was reversed and the delay reduced. At each reversal the step size was halved: 32, 16 ms and so on. In this way the delay was adjusted towards a value at which the participant had a 50% chance of correctly withholding the response.

If no response was made the screen was cleared after 3 s from stimulus onset; alternatively, if there had been a response the screen went blank 750 ms after the key was pressed. After a further 750 ms the sequence was repeated, until a total of 72 stimuli had been presented. At the end, the grand mean of all reaction times (RTs) to black arrows was calculated. The stop signal delay (SSD) was taken to be the mean of the most recent delays resulting in a) success and b) failure. Thus, if a participant successfully withheld a response with a delay of 320 ms, but responded when the delay was 336 ms, then the SSD was assumed to be 328 ms. The SSRT was calculated as mean RT – SSD. A larger value of SSRT implies less inhibition.

EXPERIMENTAL PROCEDURE

All participants were first given 24 practice stimuli, to familiarize them with the task. Following this, four from each group went on to be tested in the waking condition first, while three were hypnotized before continuing. The induction consisted of progressive relaxation and guided visual imagery, following which participants were instructed that it would be easy for them to open their eyes and perform the task they had seen before. After testing they were told to close their eyes, then were given formal waking instructions. After the first sequence, participants repeated the procedures in the other condition.

Table 1 Mean and (SD) SSRT values (ms) for the two groups, in the waking and hypnosis conditions

	Waking	Hypnosis
Low hypnotizable	522 (152)	583 (184)
High hypnotizable	579 (118)	509 (102)

RESULTS

Table 1 shows the mean stop signal reaction times for the 'highs' and 'lows' in the two conditions. Overall, 'highs' maintained shorter SSRTs, but the difference was not statistically significant. A two-way analysis of variance (state × hypnotizability) revealed only the interaction to be significant, $F_{1,12} = 11.8$, $p = 0.005$. This shows that attempting hypnosis by people who are of low susceptibility results in an increase in SSRT, whereas in those who are highly hypnotizable hypnosis lowers SSRT (i.e. increases inhibition) with respect to the waking value.

DISCUSSION

This small-scale study suggests strongly that people who are adept at hypnosis do not achieve this by engaging in a generalized reduction in inhibition. In fact the reverse appears to be true; they become better able to inhibit. This can be interpreted as being in line with the results of McGeown et al. (2009) who showed with fMRI that hypnotized 'highs' had reduced default mode activity. Of course, the relative inactivity of their participants' brains may have been attributable to lack of excitation, rather than copious inhibition, but it is implausible that the level of inhibition had been reduced.

Another parallel may be found in the results of Gruzelier, Gray and Horn (2002) who recorded evoked potentials in an odd-ball experiment. In this kind of test brain activity is monitored via scalp electrodes, while a series of identical stimuli (simple 'beeps') is presented. Each stimulus produces a neural response, the evoked potential, which is detected via electroencephalography. The unchanging stimuli produce only a small response, but when an 'odd-ball' is introduced (a beep of different pitch or duration) there is a very strong, characteristic electrical response. Gruzelier et al. found that in hypnotized 'highs' this response was dramatically reduced, whereas when 'lows' were hypnotized an enhanced response was produced.

The diminished response of the 'highs' in the Gruzelier et al. study is presumably attributable to increased inhibition, while the 'lows' appear to have reduced it. Both these effects precisely mirror those of the current experiment and appear to be part of a general tendency in 'lows' not simply to fail to produce effects, but actually to show reverse effects (Naish, 2014).

A clinical observation may be of relevance to the role of inhibition in hypnosis. It is that obsessive-compulsive patients are widely held to be very difficult to hypnotize; if formally tested they would score as 'lows'. It is also the case that these people are poor at inhibiting, so perform relatively badly on the SSRT task (Lipszyc & Schachar, 2010). If disinhibition were an aid to hypnosis, then obsessive-compulsive disorder should be associated with high susceptibility. It is not, but another condition is: post-traumatic stress disorder (PTSD). Those suffering from PTSD are more than averagely hypnotizable (Yard, et al., 2008) and appear to have good inhibition, except for trauma-related material (Naish, 2012). The flashbacks of PTSD are remarkably like hypnotic hallucinations, and for patients the focus on the trauma material combined with the

ignoring of disconfirming information can lead to the flashback being interpreted as reality, even months after the precipitating event. As with hypnotic 'highs', these effects are explicable in terms of a well-tuned deployment of inhibition and disinhibition.

If, as is being argued, strategic inhibition is an important part of achieving hypnotic effects, it is not immediately clear how consuming moderate amounts of alcohol improves hypnotic responding. The people being tested in the Semmens-Wheeler et al. (2013) study were only modestly responsive to hypnosis, but what made them so is not known. It was suggested in the introduction that hypnotic effects might be initiated via a mix of excitatory and inhibitory mechanisms. It is possible that people who are moderately susceptible are relatively good at initiating excitatory processes, sowing the seeds of a non-veridical experience, but they may be less good at turning off the disconfirming inhibition, the kind of inhibition that signals the effect to be coming from within, not without. If this were the case, then modest amounts of alcohol may facilitate the strategic inhibition reduction, although larger amounts would disinhibit too widely.

CONCLUSION

People who score high on hypnotic susceptibility scales appear to increase their ability to inhibit when they are hypnotized. This is taken to support models of hypnosis that explain the phenomenon in terms of focused attention and strategic deployment of frontal lobe involvement.

It is recognized that this is a small study, that may not generalize to lower levels of hypnotic susceptibility nor, possibly, to other means of testing for inhibition. However, it is an indication of an area deserving further exploration.

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Correspondence to: Peter L. N. Naish, Department of Psychology, The Open University, Milton Keynes MK7 6AA, UK
Email: peter.naish@open.ac.uk
Phone: +44 (0)1491 680 944