International Journal of Psychophysiology xxx (2009) xxx–xxx

Contents lists available at ScienceDirect

International Journal of Psychophysiology

journal homepage: www.elsevier.com/locate/ijpsycho

The effect of gamma enhancing neurofeedback on the control of feature bindings and intelligence measures

André W. Keizer [a,*], Maurice Verschoor [b], Roland S. Verment [b], Bernhard Hommel [a]

[a] *Leiden University, Institute for Psychological Research, The Netherlands*
[b] *Leiden Institute for Brain and Cognition, Leiden, The Netherlands*

ARTICLE INFO

Article history:
Received 15 June 2009
Received in revised form 21 October 2009
Accepted 26 October 2009
Available online xxxx

Keywords:
Gamma
Neurofeedback
Binding
Feature integration
Intelligence

ABSTRACT

Neural synchronization in the gamma band has been associated with feature binding and intelligence. Using neurofeedback, we aimed at changing the power of the gamma band and investigated whether these changes would influence behavioral measures of feature binding and intelligence. The results show that people are indeed able to alter the power in the gamma band if provided with neurofeedback. Moreover, the increase of gamma band power was related to a decrease of binding costs and an increase in intelligence, suggesting that the control of feature binding and intelligence share a common underlying mechanism.

1. Introduction

Visual information is analyzed by different specialized brain areas, each coding for different features in the visual field, such as shape (Kourtzi and Kanwisher, 2000) and color (Zeki et al., 1991). Even though visual information processing in the brain is so dispersed, we still perceive visual objects as integrated wholes. The question of how the brain is able to do this has often been referred to as the 'binding problem' (Treisman, 1996). A possible mechanism for the binding of features in the brain is the temporal coupling of neural populations, which is assumed to be achieved by coordinating and synchronizing the firing rates of cells referring to the same event. That is, feature conjunctions may be coded through the temporal coherence of their neural codes, presumably in the gamma frequency band (~30–100 Hz; for an overview, see Engel and Singer, 2001; Jensen et al., 2007). However, there still is an ongoing controversy regarding the functional relevance of neural synchronization in general (Pareti and De Palma, 2004) and of gamma band activity in particular (Yuval-Greenberg et al., 2008). For instance, the findings of Yuval-Greenberg et al. (2008) suggest that a specific gamma band increase after stimulus presentation, called the transient-broadband induced gamma band response (IGBRtb), might be due to miniature saccades that follow stimulus presentation rather than to true increases in neural synchrony. Findings of that sort complicate the interpretation of the relation between induced gamma responses in the time-range of 200–300 ms after visual stimulation and cognitive processes (Yuval-Greenberg and Deouell, 2009).

Even though direct links between gamma activity and feature integration are yet to be demonstrated, there is converging evidence that processes involved in the creation and maintenance of (visual) feature bindings are accompanied by, and systematically related to neural activity in the gamma band. In particular, gamma band power is correlated with visual awareness (Engel and Singer, 2001; Wyart and Tallon-Baudry, 2008) and visual working memory (Tallon-Baudry et al., 1998).

At the neural level, synchronization has been associated with activation in networks of GABAergic interneurons (Whittington et al., 1995). More specifically, gamma synchronization has been shown to arise from a combination of GABAergic and gap junction coupling (Tamás, et al., 2000). Gamma synchronization and visual feature integration seem to be related to the same neurotransmitter system. Gamma synchrony in the primary visual cortex of the cat is enhanced by muscarinic–cholinergic agonists and disrupted by muscarinic–cholinergic antagonists (Rodriguez-Bermudez et al., 2004). This fits with observations that, in humans, caffeine—a muscarinic–cholinergic agonist—facilitates visual feature integration (Colzato et al., 2005), whereas alcohol—a muscarinic–cholinergic antagonist—impairs it (Colzato et al., 2004) (These findings could be replicated with more selective, pharmacological interventions in the rat, Botly and De Rosa, 2007, and in humans, Botly and De Rosa, 2008). The relationship between muscarinic and cholinergic pathways and visual feature binding seems to be rather specific, as visual feature integration is not affected by nicotinic–cholinergic or dopaminergic manipulations (e.g.,

---

* Corresponding author. University of Amsterdam, Department of Psychology, Roetersstraat 15, 1018 WB, Amsterdam, The Netherlands. Tel.: +31 20 5256724; fax: +31 20 6391656.
E-mail address: a.w.keizer@uva.nl (A.W. Keizer).

0167-8760/$ – see front matter © 2009 Elsevier B.V. All rights reserved.
doi:10.1016/j.ijpsycho.2009.10.011

Please cite this article as: Keizer, A.W., et al., The effect of gamma enhancing neurofeedback on the control of feature bindings and intelligence measures, Int. J. Psychophysiol. (2009), doi:10.1016/j.ijpsycho.2009.10.011

Colzato and Hommel, 2008), and muscarinic–cholinergic manipulations have no impact on visuomotor integration (Colzato et al., 2004, 2005).

Given that most of the empirical evidence on the role of synchronization in feature integration points to a connection between gamma band activity and visual integration, our study focused on this possible relationship. The second variable we considered in our study was intelligence, mainly because it seems to be related to both feature integration and neural activity in the gamma band, and may thus be an important mediator in the link between integration and synchronization. Several authors have assumed and provided preliminary evidence that fluid intelligence in humans relies on gamma band synchronization (Jausovec, 2004; Jausovec and Jausovec, 2005, 2007; Lee et al., 2003; Stankov et al., 2006). With regard to binding, people high in fluid IQ have been found to be more efficient in updating feature bindings in the case of feature changes (Colzato et al., 2006a). How intelligence and binding may interact is not entirely clear however. On the one hand, it is possible that higher fluid intelligence is associated with more efficient integration processes, and one may even imagine that is this higher efficiency that allows for more intelligent behavior. On the other hand, however, it is also possible that fluid intelligence does not affect binding itself but the handling and cognitive control of bindings. Indeed, fluid intelligence correlates with executive control processes (Duncan et al., 1996; Gray et al., 2003), which are assumed to operate on cognitive structures and processes. Support for this latter possibility comes from the Colzato et al. (2006a) study where higher intelligence scores were associated with *smaller* rather than larger effects of trial-to-trial feature changes. In other words, intelligence may be related to the control of bindings rather than to their creation.

Even though theoretical considerations and converging empirical evidence support the idea that neural synchronization in the gamma band is associated with feature integration, the functional relevance of synchronization is still under heated debate (Ghose and Maunsell, 1999; Reynolds and Desimone, 1999; Shadlen and Movshon, 1999; Palanca and DeAngelis, 2005; Treisman, 1999). One of the reasons for the ongoing debate is that most of the relevant empirical evidence is correlational, which makes it difficult to rule out that synchronization is epiphenomenal to binding and other cognitive processes. A more straightforward way to investigate the functional relevance of the gamma band would be to manipulate synchronization directly and to see whether and how this affects performance, which is what we attempted to do in the present study.

One way to manipulate particular frequency bands in the EEG signal is neurofeedback (Bird et al., 1978; Vernon et al., 2003). With neurofeedback training, brain waves are measured using electrodes that are likely to pick up signals from theoretically relevant brain areas. An online spectrum analysis is performed, which makes it possible to reward participants so to guide them to increase or decrease the power in the targeted frequency band(s). Neurofeedback has been studied mainly as a possible treatment for various kinds of psychological disorders like ADHD (Gevensleben et al., 2009), migraine (Kropp et al., 2002), and epilepsy (Kotchoubey et al., 1999; Sterman and Egner, 2006). These and other studies demonstrate that people are indeed able to alter (enhance or reduce) the power of specific frequency bands in their own EEG signal.

Even though little fundamental research has been done which uses neurofeedback as a method to study cognitive processes and their relation with specific frequency bands (notable exceptions are: Egner and Gruzelier, 2001, 2003, 2004; Vernon et al., 2003), it may provide a powerful tool to study these relationships in theoretically meaningful ways. Manipulation of gamma band power by neurofeedback training has already been successfully demonstrated in a study by Bird et al. (1978). In this study, significant differences in gamma band power were demonstrated after 8 neurofeedback sessions, each lasting 30 min. In order to investigate the role of gamma in binding and intelligence-related performance, we studied whether and how this performance would change as a consequence of neurofeedback training designed to enhance or reduce the power of cortical activity in the gamma band.

One group of our subjects was thus trained to enhance gamma-related cortical activity in 8 neurofeedback sessions as in the Bird et al. (1978) study. In a second group, we intended to reduce gamma activity. One alternative would have been placebo neurofeedback training. However, this has the disadvantage that subjects may notice the absence of consistent neurofeedback and engage in elaborate hypothesis testing, which would result in uncontrollable differences between the experimental and control group. Another possibility would have been the presentation of negative feedback, that is, of feedback that directly encourages a reduction of gamma-related activity. However, little is known about how people actually manage to increase or decrease power in a particular frequency band. If the aim is to increase power in a given band, the goal is very likely the same for all participants—however they may achieve it. If the aim is to decrease the power, however, the individual goals may be very diverse: some people may manage to directly reduce the power but others may try to achieve that indirectly by targeting other frequency bands. In an attempt to encounter these problems we thus tried to equate the two investigated groups as far as possible, and we did so by rewarding one group for increasing the power in the gamma band at the cost of beta activation and the other group for increasing the power in the beta band at the cost of gamma activation. That is, we put these two frequency bands in opposition in both groups and varied the preference for one band over the other. In the following, we will refer to the group with an induced gamma preference as the *Gamma-up* group and the group with an induced beta preference as *Control* group.

There are two reasons why we chose to include beta power in the neurofeedback training procedure. First, it has been speculated that the beta band may have different binding properties than the gamma band (Colzato et al., 2005; Schnitzler and Gross, 2005). It has been considered that communication between distant brain areas relies on lower frequency bands than communication between neighboring areas (Varela et al., 2001). Indeed, whereas feature integration in perception ('local' integration) is commonly associated with the gamma band (e.g., Engel and Singer, 2001), sensorimotor binding ('global' integration) seems to be more related to neural synchronization in the beta band (e.g., Roelfsema et al., 1997). In principle, these considerations would allow for interesting hypotheses with regard to the Control group as well; however, we will see that inducing a preference for beta power was more successful in preventing an increase in gamma power than in increasing beta power, which does not permit testing beta-related hypotheses. In other words, our Control group turned out to be a true control group rather than a Beta-Up group. Second, the findings of Bird et al. (1978) show that power changes in the gamma band impact power in the beta band as well, even though the beta band was not included in their neurofeedback design. By putting the beta band power into opposition to the gamma band we intended to get a clearer distinction between the power changes in these two frequency bands.

The rationale of our design was to test the two groups on measures of visual feature binding and intelligence before the start of the first neurofeedback training (the pretest) and again after the end of the last neurofeedback training (the posttest). Note that our design includes a repetition of all relevant tests. This invites all sorts of practice- and time-related effects and thus renders main effects of time of testing equivocal. More interesting for our purposes were interactions between time of test and group, as these interactions would indicate a differential effect of neurofeedback. To tap into feature integration, we employed a version of the task developed by Hommel (1998). In this task, participants are presented with pairs of trials, in which the features of stimuli and responses vary orthogonally. The important observation is that the repetition and alternation of a given feature depends on whether other features are repeated as

Please cite this article as: Keizer, A.W., et al., The effect of gamma enhancing neurofeedback on the control of feature bindings and intelligence measures, Int. J. Psychophysiol. (2009), doi:10.1016/j.ijpsycho.2009.10.011

well. For instance, if shape and location of a stimulus repeats or alternates, shape repetition results in better performance if the location repeats as well whereas, with shape alternation, performance is better if the location alternates as well (Colzato et al., 2006b; Hommel, 1998). This suggests that shape and location are spontaneously integrated into a kind of event file, which is retrieved if at least one feature is repeated (Hommel, 2004). Along the same lines, repeating the response is beneficial if stimulus features also repeat but impair performance if stimulus features alternate (Hommel, 1998). In the present study, we will focus on the repetition and alternation of stimulus shape and location, and assume that interactions between shape- and location repetition effects are indicative of visual feature binding. The second measure of interest was intelligence, which we measured by using Raven's Standard Progressive Matrices (Raven, 1938).

If neurofeedback is successful in increasing power in the gamma band in the Gamma-up group, and if gamma band activity is associated with feature binding and intelligence, we would expect that training would have a specific effect on our binding and intelligence measures. With regard to intelligence, one might speculate that gamma training improves performance in an intelligence-related task, suggesting better Raven scores in the Gamma-up group. The predictions regarding binding depend on exactly how the training affects binding-related processes. One possibility is that it directly improves the binding process itself, suggesting that binding-related effects should increase in the Gamma-up group. Another possibility is that training improves the handling of event files (Colzato et al., 2006a), that is, the efficiency of retrieving and updating feature bindings, suggesting that binding-related effects decrease in the Gamma-up group.

2. Materials and methods

2.1. Participants

Fourteen right-handed volunteers (2 males, mean age: 22 years) participated in the experiment. Informed consent was obtained from all participants after the nature and possible consequences of the study were explained to them. The protocol was approved by the local medical ethical committee (Leiden University Medical Center).

2.2. Procedure

Each subject was randomly assigned to one of the two groups. In a double-blind procedure, both the subject and the experimenter were unaware which of the two possible neurofeedback training protocols was given to the subject until the last subject finished the experiment. Subjects filled out a questionnaire before the start of each neurofeedback training session, enquiring for any notable changes in appetite, sleep pattern, ability to concentrate, memory capacity, and mood. The order of the behavioral tests (binding and intelligence) was counterbalanced across subjects.

2.3. Neurofeedback training

Each subject completed 8 neurofeedback sessions, 1 training session per day lasting 30 min. The neurofeedback sessions were spread over a period of 10 or 11 days. For the EEG measurements, a QDS Focus amplifier (bandwith: 0.01–60 Hz) and electrodes were used (www.brain-trainer.com). The EEG signal was received from one electrode attached to the scalp of the subject, on the Oz position, according to the international 10–20 system. Reference electrodes were placed on both earlobes of the subject. A ground electrode was attached to the forehead of the subject. The EEG power spectrum analysis was calculated online with negligible delay, using the Bioexplorer software package (www.cyberevolution.com). Two elliptic filters were applied to the signal, extracting frequencies in the gamma range (36–44 Hz) and in the beta range (12–20 Hz). Two types of thresholds were implemented, an ‘upper’ and a ‘lower’ threshold. Both types were adapting to the power of the frequency band it was applied to. More specifically, the power level was based on a moving average of 30 s that was updated continuously with the average power that was calculated over epochs of 0.125 s and both types of thresholds were set to the power level that would be surpassed 75% of the time during the preceding 30 s window. The width of the gamma band was made identical to that of the beta band, to prevent possible effects of a band-width difference in the gamma and beta band. Even though the gamma band has been defined in the range of 30 and 100 Hz, we chose to operationalize the gamma band around 40 Hz, since this seems to be the most widely accepted and most referred to indicator of the gamma band (i.e. Tallon-Baudry and Bertrand, 1999).

The subjects were randomly assigned to one of the two neurofeedback groups. In the Gamma-up group, a tone was generated whenever the following two criteria were met: (1) gamma power exceeded the upper threshold and (2) beta power exceeded the lower threshold. For the Control group, a tone was generated when both the gamma power exceeded the lower threshold and the beta power exceeded the upper threshold. In other words, positive feedback was given when both thresholds were surpassed simultaneously. Both groups were instructed to attempt to increase the rate of the tone occurrences. The maximum rate of the tones was set to one tone per second. Using these criteria, subjects achieved a high rate of tones when the power of their frequency bands was recurrently exceeding the adapting thresholds.

2.4. Binding

Binding processes were tested by using a modified version of the task used by Hommel (1998), which is designed to study the behavioral effects (reaction times and errors) of feature binding. An example trial of this task is shown in Fig. 1. Subjects were instructed to respond with a left or right key press on S1 (z- or m-key), according to the preceding arrow (3.8 cm × 2.7 cm), ignoring the picture (5.0 cm × 4.3 cm) and its location (top or bottom). On S2, subjects were instructed to respond to the picture (apple or bananas) while ignoring its location, again with a left or right key press (z- or m-key, counterbalanced across subjects). The arrows were presented in the middle square of three equally sized squares (6.3 cm), placed in vertical alignment, the images of an apple or bananas were placed in either the top or the bottom square. The task consisted of 320 trials, equally divided across conditions. The rationale of this design is that on S1, the picture, location and response are integrated into an ‘event file’ (Hommel, 1998, 2004; Hommel and Colzato, 2004). On S2, the picture, location and response can independently be repeated or alternated. It has been shown in previous studies using a version of this paradigm, that the performance on S2 is impaired when the features of S1 (shape, location, and response) are partially repeated on S2 (Hommel, 1998, 2004; Hommel and Colzato, 2004). The partial repetition or binding costs can be divided into visual binding costs, which refer the binding between the two visual features (shape and location), and visuomotor binding costs, which refer to visual features and the action.

2.5. Intelligence

Intelligence was tested by using the Raven's Standard Progressive Matrices (SPM, Raven, 1938). The performance on this task correlates with Spearman's *g* factor and has also been associated with visual short-term memory capacity (Carpenter et al., 1990) and flexibility in handling event files (Colzato et al., 2006a). The test consists of 60 trials of increasing difficulty. Subjects either received the even 30

Please cite this article as: Keizer, A.W., et al., The effect of gamma enhancing neurofeedback on the control of feature bindings and intelligence measures, Int. J. Psychophysiol. (2009), doi:10.1016/j.ijpsycho.2009.10.011

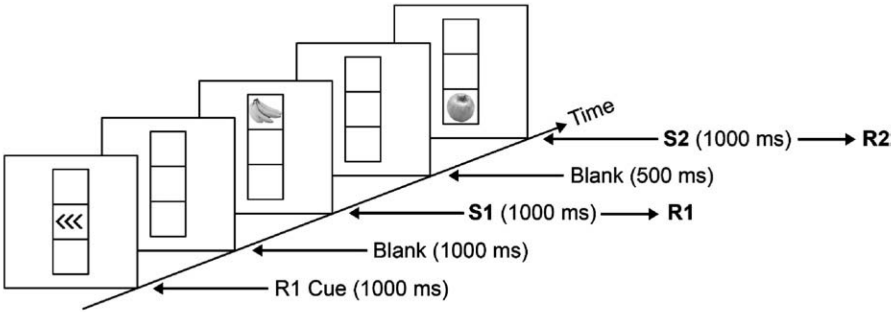

Fig. 1. Time sequence of an example trial in the binding task. Subjects had to respond with a precued response on S1 (according to the preceding arrows) and to the picture of S2 (e.g. bananas→press left, apple→press right). Performance costs were measured on the response to S2, in the conditions where features are partially repeated, in comparison with complete repetition or complete alternation of features. It is assumed that the response, picture and location are integrated on S1 and that repeating one, two or all of these features on S2 would automatically reactivate the previously associated features. In the partial repetition conditions, automatic reactivation would lead to inappropriate reactivation of previously associated features, resulting in the performance costs.

trials on the pretest and the odd 30 trials on the posttest or vice versa (counterbalanced across subjects).

3. Results

3.1. Neurofeedback training

Fig. 2A shows the percent change between the first and last neurofeedback session of power of the frequency spectrum (0.01–60 Hz, divided in 2 Hz bins). As can be seen from this figure, gamma enhancing neurofeedback mainly affected the higher frequency bands, peaking at the gamma band, (30–60 Hz). Using an ANCOVA that considers gamma power (30–60 Hz) of session 1 as a covariate, a significant difference in gamma power was found between the two groups on session 8, $F(1,13)=14.2$, $p<.005$ (Fig. 2B, Table 1), in the absence of a difference between the two groups on the pretest, $T(12)=1.3$, $p>.2$.

There also seemed to be a decrease of low frequencies (2–12 Hz) in the Control group versus an increase in the Gamma-up group (we excluded bins 48–50 Hz and 50–52 Hz from the analyses, since it contained 50 Hz AC noise). Indeed, the ANCOVA that considered 2–12 Hz power of session 1 as a covariate, a significant difference in 2–12 Hz power was found between the two groups on session 8, $F(13,2)=5.4$, $p<.05$, in the absence of a difference between the two groups on the pretest, $T(12)=1.1$, $p>.2$. However, the power of 2–12 Hz in session 8 was very similar across the two groups (Gamma-up: decrease from 16.1 uV in session 1 to 14.9 uV in session 8, Control: increase from 13.3 uV in session 1 to 14.6 uV in session 8), which may be due to a regression towards the mean.

No significant group differences were found on any of the items of the questionnaire.

3.2. Binding

Binding effects were assessed by means of repeated measures ANOVAs of reaction times and error rates with repetition versus alternation of stimulus shape, stimulus location, and response as two-level factors. Reaction times below 200 ms and above 1000 ms were considered as outliers and were discarded (3% of the data). Significant main effects were found for repetition/alternation of shape, $F(1,13)=8.96$, $p<.05$, location, $F(1,13)=11{,}75$, $p<.005$, and response, $F(1,13)=6.95$, $p<.05$. More importantly, the pretest replicated earlier findings by showing significant interactions in reaction times between the repetition/alternation of shape and location, $F(1,13)=20.79$, $p<.005$, location and response, $F(1,13)=52.13$, $p<.00005$, and shape and response, $F(1,13)=15.21$, $p<.005$, (Tables 1 and 2). All three effects were due to better performance if the stimulus feature and/or the response were both repeated or alternated as compared to the repetition of one but not the other. Error rates mirrored the reaction time results for the interaction between shape and response $F(1,13)=30.12$, $p<.0005$, and between location and response $F(1,13)=8.59$, $p<.05$, but not for the interaction between shape and location, $p>.4$. None of the main effects was significant for error rates in the pretest, $ps>.2$.

The posttest showed similar results for the main effects in reaction times of repetition/alternation of shape, $F(1,13)=6.31$, $p<.05$, and location, $F(1,13)=6.09$, $p<.05$, but not for response, $p>.1$. Similar results were also found for the interactions in reaction times between the repetition/alternation of shape and location, $F(1,13)=52.82$,

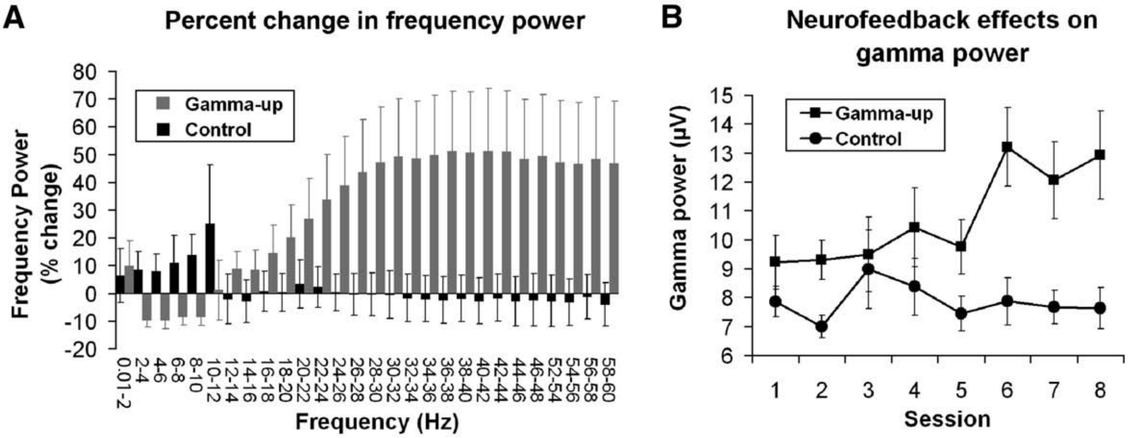

Fig. 2. The effects of neurofeedback on the power in frequency bands in the Gamma-up group and the Control group. The graph indicates the percent change in power between 0.01 and 60 Hz, divided in bins of 2 Hz (A). The increase of gamma band power (30–60 Hz) over the course of 8 neurofeedback sessions (B). Error bars depict standard error of the mean.

Please cite this article as: Keizer, A.W., et al., The effect of gamma enhancing neurofeedback on the control of feature bindings and intelligence measures, Int. J. Psychophysiol. (2009), doi:10.1016/j.ijpsycho.2009.10.011

Table 1
Means of mean reaction times and standard deviations (SD) for responses to stimulus 2 (RT; in ms) and percentages of errors on R2 (PE), as a function of group (Control vs Gamma-up), test instance (Pretest vs. Posttest), the match between response 1 and response 2, and the feature match between stimulus 1 and stimulus 2.

Pretest								
Control					Gamma-up			
	Response				Response			
	Repeated		Alternated		Repeated		Alternated	
Match	RT(SD)	PE(SD)	RT(SD)	PE(SD)	RT(SD)	PE(SD)	RT(SD)	PE(SD)
Neither	493 (43)	9,3 (11,3)	452 (50)	2,9 (4,1)	533 (57)	6,4 (6,6)	483 (41)	1,5 (2,5)
L(ocation)	509 (67)	4,4 (7,3)	488 (46)	6,6 (5,7)	575 (56)	5 (5,8)	499 (37)	3,6 (2,5)
S(hape)	466 (43)	1,4 (3,8)	488 (40)	5,8 (4,5)	492 (48)	0,8 (2)	514 (39)	2,3 (2,8)
LS	457 (35)	2,1 (3,9)	490 (52)	3,9 (5,3)	482 (46)	0 (0)	533 (67)	7,8 (9,2)
Posttest								
Match	RT(SD)	PE(SD)	RT(SD)	PE(SD)	RT(SD)	PE(SD)	RT(SD)	PE(SD)
Neither	458 (64)	7,3 (5,8)	442 (62)	1,4 (3,8)	527 (68)	5,2 (51)	488 (59)	1,4 (2,4)
L(ocation)	503 (85)	6 (7,1)	473 (34)	2,9 (4)	546 (64)	2,8 (5,4)	502 (54)	1,4 (2,4)
S(hape)	466 (53)	0,7 (1,9)	481 (53)	5 (5,8)	500 (68)	0,7 (1,9)	484 (49)	7,3 (9,9)
LS	435 (51)	1,5 (4)	471 (40)	8,8 (8,2)	468 (63)	0,7 (1,9)	514 (55)	5,2 (2,9)

Table 2
Means of mean power (uV) and standard deviations (SD) of Gamma (36–44 Hz) and Beta (12–20 Hz) in session 1 and session 2, means of mean percentages of errors (PE) of Raven's SPM performance and means of mean reaction times (RT; in ms) of binding costs1 in the pretest and posttest as a function of group (Gamma-up and Control).

Session 1 Pretest			
	Gamma	Raven SPM	Binding costs
	uV (SD)	PE (SD)	RT (SD)
Gamma-up	7,1 (2,0)	13,7 (11,1)	14,6 (18,4)
Control	6,0 (1,1)	15,1 (6,7)	13,5 (15,c)
Session 8 Posttest			
Gamma-up	10,0 (3,8)	13,4 (6,7)	10,4 (12,1)
Control	5,8 (1,5)	11,7 (6,2)	26,3 (10,8)

$p<.00001$, and location and response, $F(1,13)=76.14$, $p<.000005$, but not for shape and response, $p>.1$. Error rates mirrored the reaction time results for the interaction between shape and location, $F(1,13)=13.28$, $p<.005$, but not for the interaction between shape and response, $p>.1$, and location and response, $p>.1$. None of the main effects were significant for error rates in the posttest, $ps>.7$.

To assess the impact of neurofeedback on binding, we considered the data from the first and the second halves of pretest and posttest separately. The reason was that binding effects have been found to the particularly sensitive to practice, especially those related to the integration of visual features (Colzato et al., 2006b). The advantage of Hommel's (1998) task version is that it renders the integration process itself task-irrelevant (i.e., the task does not require any binding), which provides a relatively process-pure, strategy-free estimate of the binding process. The flipside of this advantage is that only one of the two stimulus features is actually task relevant (shape in the present case) but the other (location in the present case) can safely be ignored. People seem to learn ignoring the irrelevant feature rather quickly, so that binding effects often disappear in the course of a session (Colzato et al., 2006b). To provide a more sensitive measure of binding we thus focused on the first half of the trial,[1] that is, on the data from the first 160 trials.

From the first and second halves we calculated individual binding costs for all three feature combinations (shape–location, location–response, and shape–response) by subtracting the mean reaction times and error percentages for the complete repetition condition and the complete alternation condition from the means of both partial repetition conditions,[2] for both the pretest and posttest. In three independent ANCOVAs, the binding costs for the three feature combinations on the posttest were entered as dependent variables, the binding costs for the three feature combinations on the pretest were entered as a covariate and group (Gamma-up, Control) was entered as a fixed factor. For the reaction times, the ANCOVAs revealed a significant impact of training on the interaction between shape repetition and location repetition, that is, on the indicator of visual binding costs, $F(1,11)=10.57$, $p<.05$ (Fig. 3). In other words, there was a significant difference on the visual binding costs of the posttest between the two groups, when controlling for differences between the visual binding costs of the pretest. This effect reflects a decrease of visual binding costs in the Gamma-up group and an increase of binding costs in the Control group. No significant impact of training was found on both indicators of visuomotor binding costs, location–response: $p>.3$, shape–response: $p>.6$. The effect on visual binding costs was observed only when the first halves of the pre- and posttest were compared; on the second halves of the pre- and posttest, no significant impact of training was found on any of the binding costs, $ps>.4$. Error rates were not modulated by training, neither in the first half of the experiment, nor in the second half of the experiment.

3.3. Intelligence

There were no significant differences between the percentage correct of the Gamma-up group and the Control group, for the pretest, $p>.7$, and for the posttest, $p>.6$. An ANCOVA with the posttest percentage correct as the dependent variable, the pretest percentage correct as covariate and the two different groups as fixed factor did not reveal an effect of group, $p>.5$, suggesting that neurofeedback does not improve intelligence-related performance in a simple, linear fashion. However, the percent change of the intelligence score from pretest to posttest correlated significantly with the percent change in gamma power in the Gamma-up group, $r=.82$, $p<.05$ (two-tailed; Fig. 4A). We calculated the correlations between the percent change of the power in all the 2 Hz frequency bins and correlated this with the

---

[1] We nevertheless preferred running a relatively long session just in case the more practice-resistant bindings between relevant features (shape and response) would also be affected by neurofeedback training.

[2] For instance, the shape–location reaction time binding cost would result from $(RT_{\text{shape rep, location alt}}+RT_{\text{shape alt, location rep}})-(RT_{\text{shape rep, location rep}}+RT_{\text{shape alt, location alt}})$. This represents the interaction term, which approaches zero for additive effects of shape and location repetition but grows as the interaction increases in size.

Please cite this article as: Keizer, A.W., et al., The effect of gamma enhancing neurofeedback on the control of feature bindings and intelligence measures, Int. J. Psychophysiol. (2009), doi:10.1016/j.ijpsycho.2009.10.011

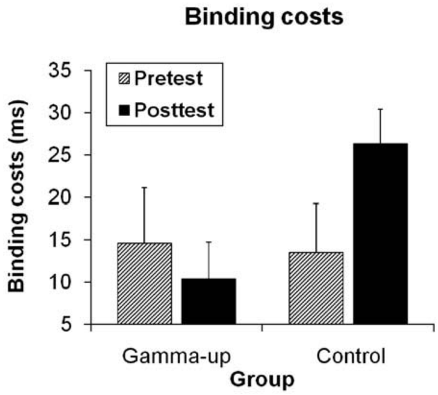

Fig. 3. The neurofeedback training resulted in a decrease of binding costs in the Gamma-up group and an increase of binding costs in the Control group (for the first half of the trials). Error bars depict standard error of the mean.

percent change of the intelligence score (Fig. 4B). The result showed that the positive correlation between the change in intelligence and the change frequency band power already peaks at 16 Hz, which suggests that both high-beta and gamma might be important for intelligence. We also observed a negative correlation between the percent change of power in the 2–4 Hz bin and percent change in intelligence that approached significance, $r = -.7$, $p = .08$. This correlation suggests that neurofeedback targets a common mechanism that is reflected in both intelligence scores and binding costs, so that training-induced changes covary.

4. Discussion

Neural synchronization in the gamma band has been proposed as the mechanism or medium that enables binding of different types of information coded in different brain areas. The aim of the present study was to investigate whether training people to increase gamma activity in their brain by means of neurofeedback would lead to any changes in binding-related performance. If this could be demonstrated, this would be relevant for theories concerning the functional role of the gamma band as well as for the development of neurofeedback as a valuable research method.

Indeed, our study provides the first direct evidence in humans that the experimental manipulation of gamma activity affects binding-related performance. In particular, we found that the performance costs associated with the only partial repetition of visual feature conjunctions (as compared to complete repetitions or alternations) are significantly smaller after neurofeedback training targeting gamma activity than after beta-targeted training. This difference reflected a decrease in binding costs in the Gamma-up group compared to an increase of binding costs in the Control group. While the decrease in binding costs seems to relate to an increase of gamma power in the Gamma-up group, increased binding costs in the Control group were not accompanied by any measurable changes in gamma or beta power. However, the Control group received a neurofeedback training protocol which was aimed at decreasing the power of the gamma band and it therefore might be that this training protocol had disturbing effects on the gamma band, which led to the increased binding costs. Notably, these changes were restricted to interactions between visual shape and location features but did not affect interactions involving the response. If we consider interactions between repetition effects to indicate binding processes, this result pattern suggests that gamma training affects visual integration but not sensorimotor integration. This dissociation fits with numerous related observations. For instance, measures of visual integration have been found to be sensitive to manipulations of muscarinic cholinergic but not dopaminergic pathways, whereas measures of visuomotor integration are sensitive to manipulations targeting dopaminergic but not cholinergic pathways (Colzato et al., 2004, 2005; Colzato and Hommel, 2008; Colzato et al., 2007a,b). It has been speculated that visual integration is mainly driven by local neural interactions in the gamma band, whereas visuomotor integration relies more on beta synchronization (Schnitzler and Gross, 2005), which may suggest a close link between cholinergic pathways and gamma synchronization on the one hand and between dopaminergic pathways and beta synchronization on the other. If so, a more successful neurofeedback training targeting beta activity may well impact measures of visuomotor binding but not visual binding. However, as our own attempt to increase beta power systematically failed, we can only speculate with regard to this issue. Although previous studies have shown differences in the power of the beta band as a result of neurofeedback (Bird et al., 1978; Vernon et al., 2003), the neurofeedback protocols that were used in these studies did not include the gamma band. More specifically, Bird et al. (1978) showed that both gamma band power and beta band power could be modulated by neurofeedback, when trained in isolation. That is, one group of subjects received neurofeedback training to modulate gamma band power, while a different group of subjects received neurofeedback training to modulate beta band power. The neurofeedback training

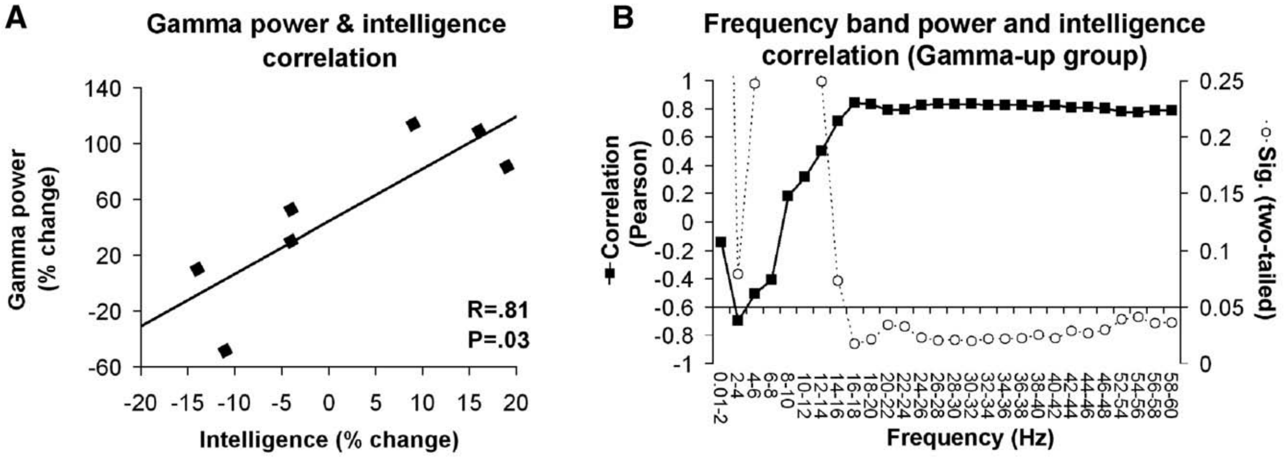

Fig. 4. There was a significant positive correlation between the percent change in gamma power (30–60 Hz) and the percent change in intelligence for the Gamma-up group (A). Figure 4B shows the correlation between the percent change in power between 0.01 and 60 Hz, divided in bins of 2 Hz with the percent change in intelligence. This graph shows that the positive correlation was present between 16 and 60 Hz, thus including both the gamma band (30–60 Hz) and the high-beta band (16–20 Hz). Error bars depict standard error of the mean.

Please cite this article as: Keizer, A.W., et al., The effect of gamma enhancing neurofeedback on the control of feature bindings and intelligence measures, Int. J. Psychophysiol. (2009), doi:10.1016/j.ijpsycho.2009.10.011

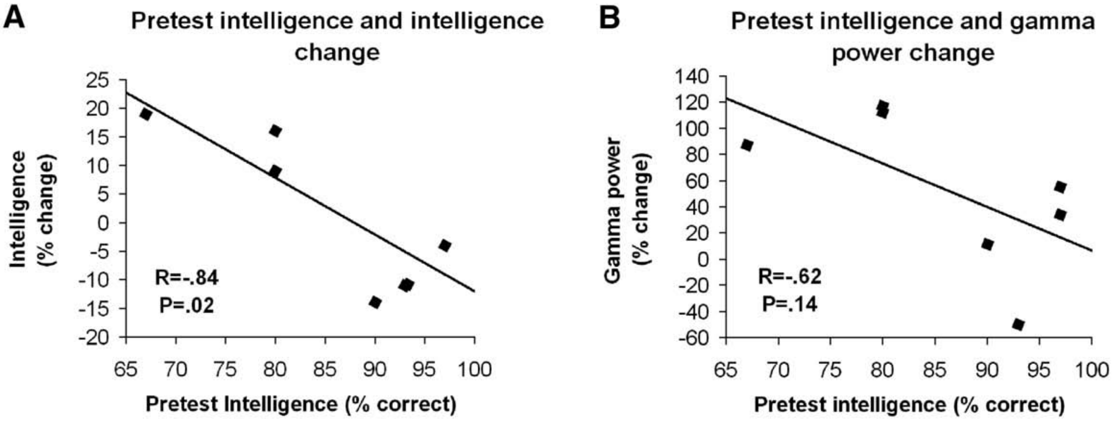

Fig. 5. Pretest intelligence correlated negatively with percent change of intelligence (A). There was no significant correlation between pretest intelligence and percent change in gamma power (B). These correlations suggest that pre-existing differences in learning ability did not underlie the positive correlation between percent change in intelligence and the percent change in gamma power.

that Vernon et al. (2003) used, was also solely aimed at modulating beta band power. Therefore, the reason for an absence of beta power modulation in our study may be the nature of the neurofeedback protocol that was used: targeting both the gamma band and the beta band.

In any case, our findings support the idea that neural synchronization in the gamma band is related to feature integration, especially to the integration of perceptual features (Engel and Singer, 2001; Jensen et al., 2007). Our findings also support the idea that intelligence is related to gamma band activation (Jausovec, 2004; Jausovec and Jausovec, 2005, 2007; Lee et al., 2003; Stankov et al., 2006). Neurofeedback training did not simply increase intelligence-related performance but improvements were correlated with, and thus presumably related to feedback-induced changes in gamma and high-beta band power. Since performance on the SPM undoubtedly depends on a myriad of cognitive functions, it is not likely that all these processes are affected by gamma enhancing neurofeedback. Rather, the effects of gamma neurofeedback on binding demonstrated in our study and the association between intelligence and binding demonstrated in the study of Colzato et al. (2006a) suggests that intelligence is codetermined by binding processes, which in turn is related to the gamma band and possibly the high-beta band as well. The fact that neurofeedback reduced rather than increased binding-related effects might provide a clue for how intelligence and integration are related. As mentioned above already, one may consider two ways of how gamma band changes may modify binding effects. Neural synchronization may play a role in the integration process itself and/or the maintenance of bindings over time (Raffone and Wolters, 2001). If so, increasing gamma power should have facilitated the creation and/or the maintenance of bindings, which should result in more pronounced binding costs. Obviously, this is the opposite of what we observed, which discounts this possibility. Alternatively, neural synchronization may represent the medium of how control mechanisms interact with local coding processes (cf., Gross et al., 2004). If so, increasing gamma power might increase the amount of control over the retrieval of bindings (Colzato et al., 2006a), which should result in less interference from previously created and now mismatching bindings. This would indeed fit with our observation. Moreover, control processes are commonly related to the frontal cortex (Duncan, 2001; Miller and Cohen, 2001), as is intelligence (Duncan et al., 2000), and control functions and intelligence arguably overlap conceptually (Kane and Engle, 2002). Gamma-targeted training may thus facilitate the handling of feature bindings by improving the communication between binding-related processes and control functions. Evidence for this hypothesis comes from findings that show a relation between high gamma band activity in the visual cortex and improved discrimination between previously presented visual stimuli and new visual stimuli (old/new effect; Gruber et al., 2002; Gruber et al., 2008; Sederberg et al., 2007). Related findings suggest that the source of this gamma band activation may be located in frontal, control related areas (Babiloni et al., 2006; Lutzenberger et al., 2002). We must, however, be careful in interpreting the correlation between gamma/high-beta and intelligence, since we cannot exclude the possibility that this correlation is mediated by a third variable. One example of such a variable is that individual differences may exist in the learning ability in the Gamma-up group, such that 'good learners' exhibit a larger increase in gamma power and a larger practice effect in SPM performance than 'bad learners'. However, if there are indeed pre-existing learning ability differences among the subjects, this would also be reflected in differences of pretest SPM performance, as learning ability arguably plays a role in fluid intelligence. Indeed, recent findings show that learning ability seems to be the single most important predictor of Raven's progressive matrices performance (e.g. Kaufman et al., 2009; Tamez et al., 2008; Williams and Pearlberg, 2006). We therefore suspect that high-learners actually show a *smaller* practice effect in SPM performance, since their performance may already close to ceiling level (certainly in our group of undergraduate students). Indeed, pretest SPM performance correlated negatively with the percent change of SPM performance in the Gamma-up group, $r = -.84$, $p = .017$ (Fig. 5A). More importantly, if pre-existing differences in learning ability would determine the change in gamma power, a positive correlation would be expected between pretest SPM performance and the percent change of gamma power. Our results do not show evidence for such a positive correlation in the Gamma-up group, $r = -.62$, $p = .14$ (Fig. 5B). We acknowledge that our relatively small sample size does not allow us to draw strong conclusions on the basis of these effects, but these negative correlations do seem to invalidate the alternative explanation that learning ability differences explain the correlation between percent change of SPM performance and percent change of gamma power.

To summarize, our study shows that neurofeedback provides a powerful method for studying the functional relevance of frequency bands in the EEG signal. Showing that alterations in the gamma band have consequences for behavioral measures is an important proof of principle, providing more explanatory power than the demonstration of correlations. Even though our results must be considered preliminary, they do suggest that binding and intelligence are jointly influenced by changes in the gamma band, which points to a common underlying mechanism.

Please cite this article as: Keizer, A.W., et al., The effect of gamma enhancing neurofeedback on the control of feature bindings and intelligence measures, Int. J. Psychophysiol. (2009), doi:10.1016/j.ijpsycho.2009.10.011

References

Babiloni, C., Vecchio, F., Cappa, S., Pasqualetti, P., Rossi, S., Miniussi, C., Rossini, P.M., 2006. Functional frontoparietal connectivity during encoding and retrieval processes follows HERA model—a high-resolution study. Brain Res. Bull. 68, 203–212.

Bird, B.L., Newton, F.A., Sheer, D.E., Ford, M., 1978. Biofeedback training of 40-Hz EEG in humans. Biofeedback Self-Regul. 3, 1–11.

Botly, L.C.P., De Rosa, E., 2008. A cross-species investigation of acetylcholine, attention, and feature binding. Psychol. Sci. 19, 1185–1193.

Botly, L.C.P., De Rosa, E., 2007. Cholinergic influences on feature binding. Behav. Neurosci. 121, 264–276.

Carpenter, P.A., Just, M.A., Shell, P., 1990. What one intelligence test measures: a theoretical account of the processing in the Raven Progressive Matrices test. Psychol. Rev. 97, 404–431.

Colzato, L.S., Hommel, B., 2008. Cannabis, cocaine, and visuomotor integration: evidence for a role of dopamine D1 receptors in binding perception and action. Neuropsychologia 46, 1570–1575.

Colzato, L.S., Erasmus, V., Hommel, B., 2004. Moderate alcohol consumption in humans impairs feature binding in visual perception but not across perception and action. Neurosci. Lett. 360, 103–105.

Colzato, L.S., Fagioli, S., Erasmus, V., Hommel, B., 2005. Caffeine, but not nicotine enhances visual feature binding. Eur. J. NeuroSci. 21, 591–595.

Colzato, L.S., van Wouwe, N.C., Lavender, T.J., Hommel, B., 2006a. Intelligence and cognitive flexibility: fluid intelligence correlates with feature "unbinding" across perception and action. Psychon. Bull. Rev. 13, 1043–1048.

Colzato, L.S., Raffone, A., Hommel, B., 2006b. What do we learn from binding features? Evidence for multilevel feature integration. J. Exp. Psychol. Hum. 32, 705–716.

Colzato, L.S., Spapé, M., Pannebakker, M.M., Hommel, B., 2007a. Working memory and the attentional blink: blink size is predicted by individual differences in operation span. Psychon. Bull. Rev. 14, 1051–1057.

Colzato, L.S., van Wouwe, N.C., Hommel, B., 2007b. Spontaneous eyeblink rate predicts the strength of visuomotor binding. Neuropsychologia 45, 2387–2392.

Duncan, J., 2001. An adaptive coding model of neural function in prefrontal cortex. Nat. Neurosci. 2, 820–829.

Duncan, J., Emslie, H., Williams, P., Johnson, R., Freer, C., 1996. Intelligence and the frontal lobe: the organization of goal-directed behaviour. Cogn. Psychol. 30, 257–303.

Duncan, J., Seitz, R.J., Kolodny, J., Bor, D., Herzog, H., Ahmed, A., et al., 2000. A neural basis for general intelligence. Science 289, 457–460.

Egner, T., Gruzelier, J.H., 2001. Learned self-regulation of EEG frequency components affects attention and event-related brain potentials in humans. NeuroReport 12, 4155–4159.

Egner, T., Gruzelier, J.H., 2003. Ecological validity of neurofeedback: modulation of slow wave EEG enhances musical performance. NeuroReport 14, 1221–1224.

Egner, T., Gruzelier, J.H., 2004. EEG biofeedback of low beta band components: frequency-specific effects on variables of attention and event-related brain potentials. Clin. Neurophysiol. 115, 131–139.

Engel, A.K., Singer, W., 2001. Temporal binding and the neural correlates of sensory awareness. Trends Cogn. Sci. 5, 16–25.

Gevensleben, H., Holl, B., Albrecht, B., Vogel, C., Schlamp, D., Kratz, O., et al., 2009. Is neurofeedback an efficious treatment for ADHD? A randomised controlled clinical trial. J. Child Psychol. Psychiatry 50, 780–789.

Ghose, G., Maunsell, J., 1999. Specialized representations in visual cortex: a role for binding? Neuron 24, 79–85.

Gray, J.R., Chabris, C.F., Braver, T.S., 2003. Neural mechanisms of general fluid intelligence. Nat. Neurosci. 6, 316–322.

Gross, J., Schmitz, F., Schnitzler, I., Kessler, K., Shapiro, K., Hommel, B., Schnitzler, A., 2004. Modulation of long-range neural synchrony reflects temporal limitations of visual attention in humans. Proc. Natl. Acad. Sci. U. S. A. 101, 13050–13055.

Gruber, T., Müller, M.M., Keil, A., 2002. Modulation of induced gamma band responses in a perceptual learning task in the human EEG. J. Cogn. Neurosci. 14, 732–744.

Gruber, T., Tsivilis, D., Giabbiconi, C.M., Müller, M.M., 2008. Induced electroencephalogram oscillations during source memory: familiarity is reflected in the gamma band, recollection in the theta band. J. Cogn. Neurosci. 20, 1043–1053.

Hommel, B., 1998. Event files: Evidence for automatic integration of stimulus–response episodes. Vis. Cogn. 5, 183–216.

Hommel, B., 2004. Event files: feature binding in and across perception and action. Trends Cogn. Sci. 8, 494–500.

Hommel, B., Colzato, L.S., 2004. Visual attention and the temporal dynamics of feature integration. Vis. Cogn. 11, 483–521.

Jausovec, N., 2004. Intelligence-related differences in induced gamma band activity. Int. J. Psychophysiol. 54, 37-37.

Jausovec, N., Jausovec, K., 2005. Differences in induced gamma and upper alpha oscillations in the human brain related to verbal/performance and emotional intelligence. Int. J. Psychophysiol. 56, 223–235.

Jausovec, N., Jausovec, K., 2007. Personality, gender and brain oscillations. Int. J. Psychophysiol. 66, 215–224.

Jensen, O., Kaiser, J., Lachaux, J.P., 2007. Human gamma-frequency oscillations associated with attention and memory. Trends Neurosci. 30, 317–324.

Kane, M.J., Engle, R.W., 2002. The role of prefrontal cortex in working-memory capacity, executive attention, and general fluid intelligence: an individual-differences perspective. Psychon. Bull. Rev. 9, 637–671.

Kaufman, S.B., DeYoung, C.G., Gray, J.G., Brown, J., Mackintosh, N., 2009. Associative learning predicts intelligence above and beyond working memory and processing speed. Intelligence 37, 374–382.

Kotchoubey, B., Strehl, U., Holzapfel, S., Blankenhorn, V., Fröscher, W., Birbaumer, N., 1999. Negative potential shifts and the prediction of the outcome of neurofeedback therapy in epilepsy. Clin. Neurophysiol. 110, 683–686.

Kourtzi, Z., Kanwisher, N., 2000. Cortical regions involved in perceiving object shape. J. Neurosci. 20, 3310–3318.

Kropp, P., Siniatchin, M., Gerber, W.D., 2002. On the pathophysiology of migraine—links for "empirically based treatment" with neurofeedback. Appl. Psychophys. Biofeedback 27, 203–213.

Lee, K.H., Williams, L.M., Breakspear, M., Gordon, E., 2003. Synchronous gamma activity: a review and contribution to an integrative neuroscience model of schizophrenia. Brain Res. Rev. 41, 57–78.

Lutzenberger, W., Ripper, B., Busse, L., Birbaumer, N., Kaiser, J., 2002. Dynamics of gamma-band activity during an audiospatial working memory task in humans. J. Neurosci. 22, 5630–5638.

Miller, E.K., Cohen, J.D., 2001. An integrative theory of prefrontal cortex function. Annu. Rev. Neurosci. 24, 167–202.

Palanca, B.J.A., DeAngelis, G.S., 2005. Does neural synchrony underlie visual feature grouping? Neuron 46, 333–346.

Pareti, G., De Palma, A., 2004. Does the brain oscillate? The dispute on neuronal synchronization. Neurol. Sci. 25, 41–47.

Raffone, A., Wolters, G., 2001. A cortical mechanism for binding in visual working memory. J. Cogn. Neurosci. 13, 766–785.

Raven, J.C., 1938. Progressive Matrices: A Perceptual Test of Intelligence. H.K. Lewis, London.

Reynolds, J., Desimone, R., 1999. The role of mechanisms of attention in solving the binding problem. Neuron 24, 19–29.

Rodriguez-Bermudez, R., Kallenbach, U., Singer, W., Munk, M.H., 2004. Short-and long-term effects of cholinergic modulation on gamma oscillations and response synchronization in the visual cortex. J. Neurosci. 24, 10369–10378.

Roelfsema, P.R., Engel, A.K., Koenig, P., Singer, W., 1997. Visuomotor integration is associated with zero time-lag synchronization among cortical areas. Nature 385, 157–161.

Schnitzler, A., Gross, J., 2005. Normal and pathological oscillatory communication in the brain. Nat. Rev. Neurosci. 6, 285–296.

Sederberg, P.B., Schulze-Bonhage, A., Madsen, J.R., Bromfield, E.B., Litt, B., Brandt, A., Kahana, M.J., 2007. Gamma oscillations distinguish true from false memories. Psychol. Sci. 18, 927–932.

Shadlen, M.N., Movshon, J.A., 1999. Synchrony unbound: a critical evaluation of the temporal binding hypothesis. Neuron 24, 67–77.

Stankov, L., Danthirr, V., Williams, L.M., Pallier, G., Roberts, R.D., Gordon, E., 2006. Intelligence and the tuning-in of brain networks. Learn. Individ. Differ. 16, 217–233.

Sterman, M.B., Egner, T., 2006. Foundation and practice of neurofeedback for the treatment of epilepsy. Appl. Psychophysiol. Biofeedback 31, 21–35.

Tallon-Baudry, C., Bertrand, O., 1999. Oscillatory gamma activity in humans and its role in object representation. Trends Cogn. Sci. 3, 151–162.

Tallon-Baudry, C., Bertrand, O., Peronnet, F., Pernier, J., 1998. Induced γ-band activity during the delay of a visual short-term memory task in humans. J. Neurosci. 18, 4244–4254.

Tamás, G., Buhl, E.H., Lörincz, A., Somogyi, P., 2000. Proximally targeted GABAergic synapses and gap junctions synchronize cortical interneurons. Nature 3, 366–371.

Tamez, E., Myerson, J., Hale, S., 2008. Learning, working memory, and intelligence revisited. Behav. Processes 78, 240–245.

Treisman, A., 1996. The binding problem. Curr. Opin. Neurobiol. 6, 171–178.

Treisman, A., 1999. Solutions to the binding problem: progress through controversy and convergence. Neuron 24, 105–110.

Varela, F., Lachaux, J.-P., Rodriguez, E., Martinerie, J., 2001. The brainweb: phase synchronization and large-scale integration. Nat. Rev. Neurosci. 2, 229–239.

Vernon, D., Egner, T., Cooper, N., Compton, T., Neilands, C., Sheri, A., Gruzelier, J., 2003. The effect of training distinct neurofeedback protocols on aspects of cognitive performance. Int. J. Psychophysiol. 47, 75–85.

Whittington, M.A., Traub, R.D., Jefferys, J.G.R., 1995. Synchronized oscillations in interneuron networks driven by metabotropic glutamate receptor activation. Nature 373, 612–615.

Williams, B.A., Pearlberg, S.L., 2006. Learning of three-term contingencies correlates with Raven scores, but not with measures of cognitive processing. Intelligence 34, 177–191.

Wyart, V., Tallon-Baudry, C., 2008. Neural dissociation between visual awareness and spatial attention. J. Neurosci. 28, 2667–2679.

Yuval-Greenberg, S., Tomer, O., Keren, A.S., Nelken, I., Deouell, L.Y., 2008. Transient induced gamma-band response in EEG as a manifestation of miniature saccades. Neuron 58, 429–441.

Zeki, S., Watson, J.D.G., Lueck, C.J., Friston, K.J., Kennard, C., Frackowiak, R.S.J., 1991. A direct demonstration of functional specialization in human visual cortex. J. Neurosci. 11, 641–649.

Please cite this article as: Keizer, A.W., et al., The effect of gamma enhancing neurofeedback on the control of feature bindings and intelligence measures, Int. J. Psychophysiol. (2009), doi:10.1016/j.ijpsycho.2009.10.011