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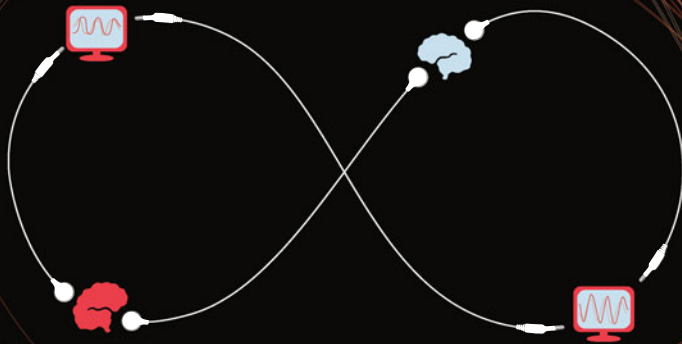
Neurofeedback

Stefanie Enriquez-Geppert, René J. Huster,
Tomas Ros and Guilherme Wood

Introduction

Already at the earliest recordings of the human EEG, the rhythmic and repetitive brain activity was one consistent detected feature (Berger 1929). As EEG time–frequency decomposition reveals, such rhythmic activity has been shown at different frequencies, ranging from delta (0–4 Hz) to gamma (30–100 Hz). Furthermore, these brain rhythms have been observed throughout different levels of neural organization, ranging from single-neuron activity, to local activity of neuronal groups, and even to activity among cortical networks of different brain areas (e.g., Buzsáki et al. 2013). These days, the study of brain oscillations is attracting substantial amount of scientific attention and is one of the fastest growing research areas in neuroscience. Oscillations represent a major mechanism of communication within the brain (Buzsáki et al. 2013) and have been consistently related to cognitive functions (e.g., Başar and Güntekin 2008; Herrmann and Knight 2001). An example of such an association is the link between frontal-midline (fm) theta

S. Enriquez-Geppert (✉)
Department of Clinical and Developmental Neuropsychology,
Faculty of Behavioural and Social Sciences, University of Groningen,
Grote Kruisstraat 2/1, 9712 TS Groningen, The Netherlands
e-mail: s.enriquez.geppert@rug.nl

R.J. Huster
Department of Psychology, University of Oslo, Oslo, Norway

T. Ros
Laboratory for Neurology and Imaging of Cognition, Department of Neurosciences,
University of Geneva, Geneva, Switzerland

G. Wood
Department of Neuropsychology, University of Graz, Graz, Austria

oscillations and executive control (Cavanagh and Frank 2014). Executive control refers to higher order functions that subserve a variety of psychological phenomena to enable adaptive and goal-oriented behavior. Fm-theta oscillations are event-related (Klimesch 1999), typically recorded over fronto-medial brain regions (Ishihara et al. 1981) and generated in the midcingulate cortex (MCC) (e.g., Cavanagh and Frank 2014). Within the network implementing executive functions (Niendam et al. 2012), the MCC is suggested to serve as a neuronal hub (Cavanagh et al. 2012). Power increases of fm-theta have been associated with enhanced cognitive processing (Mitchell et al. 2008) and can predict successful behavioral performance (Sederberg et al. 2003; Cohen and Donner 2013). Accordingly, the absence of such fm-theta power enhancements is related to reduced behavioral and cognitive performance (e.g., Donkers et al. 2011).

Mechanism of Action

Endogenous neural oscillations that show a theoretically and empirically confirmed relation to a specific cognitive function represent a direct target for the enhancement of cognition with neuroscientific approaches in general, such as neurofeedback and transcranial alternating current stimulation (tACS). However, and to put it simply, whereas tACS is an approach applying exogenous oscillations to affect endogenous neural oscillations (see Chapter "Transcranial Alternating Current Stimulation"), the goal of neurofeedback is the self-regulation of endogenous neural oscillations. Here, neural parameters of ongoing neural activity are fed back to the participant on a trial-by-trial fashion to up- or downregulate one's own brain activity (e.g., Huster et al. 2014). Thereby implementation of neurofeedback is realized by a software system and a processing pipeline consisting of five basic elements, including data acquisition, online data processing, online feature extraction, online feedback generation, and the learning participant (see Huster et al. 2014; Enriquez-Geppert et al., subm.), see Fig. 1.

Enhancement of Cognition by Neurofeedback

Based on the above-described associations of fm-theta and executive functions, neurofeedback studies have been set up. In these studies, fm-theta is extracted as scalp activity measured at mid-frontal electrodes. In one such investigation, it has been demonstrated that fm-theta neurofeedback indeed led to enhanced performance in two particular executive functions, namely task-switching and memory-updating (Enriquez-Geppert et al. 2014). In a further study, cognitive enhancements have also been shown in the domain of working memory in the elderly (Wang and Hsie 2014).
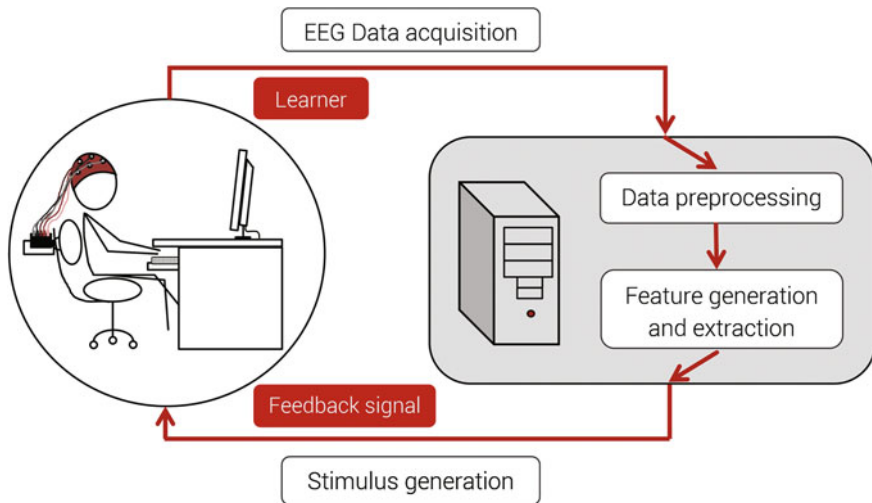
Fig. 1 Basic setup of a brain–computer interface for neurofeedback. After recording of EEG, data undergo preprocessing (e.g., artifact detection and rejection or correction), feature generation and extraction, computation, and presentation of the feedback signal. The latter step closes the feedback loop, with the participant trying to learn to use the feedback signal to alter the brain activity in accordance with the instructions

However, a considerable amount of literature reported associations of alpha oscillations with cognition, specifically focusing on the upper alpha sub-band (for a review, see Klimesch 1999). On the basis of such findings, alpha neurofeedback studies were conducted and effects were shown such as enhanced mental rotation capacity (e.g., Hanslmayr et al. 2005; Zoefel et al. 2010), as well as increased short-term memory performance (e.g., Escolano et al. 2011; Nan et al. 2012). Recently, Hsueh et al. (2016) presented evidence for an association between the amount of self-regulation capacity after neurofeedback and the amount of performance changes on cognition. Those participants who gained specifically good self-control of alpha brain activity were also those improving most regarding performance on working and episodic memory. Guez et al. (2014) performed a sham-controlled, double-blind neurofeedback study and demonstrated dissociations of two different protocols on different memory processes. Upper alpha frequency training led to enhanced strategic and top-down processes as reflected in associative memory, whereas training of the sensory motor rhythm (SMR, 13–15 Hz) led to enhanced performance in less-effortful and less-strategic memory task as reflected in improved item memory (Guez et al. 2014, see but also Kober et al. 2015a, b).

Apart from associations with working memory, alpha brain oscillations are linked to covert attention. In situations in which attention is either directed to the left or right visual hemifield, concurrent enhanced alpha is observed in the ipsilateral hemisphere, while alpha power is reduced in the contralateral hemisphere (e.g., van Gerven and Jensen 2009; Rihs et al. 2007).

Moreover, the strength of hemispheric alpha lateralization has been found to correlate with behavioral performance (Horschig et al. 2014; Thut et al. 2006). Resting upon these associations, Okazaki et al. (2015) gave their participants feedback on their posterior alpha lateralization, while they kept their attention to either the left or right hemifield. Indeed, neurofeedback training transferred to short-term changes in visual detection performance. SMR has also been observed during light non-rapid eye movement (REM) sleep, as represented by the so-called sleep spindles, which are generated in thalamo-cortical circuits (Steriade 1999). In an animal model, it was shown that learning to change SMR by conditioning transferred to facilitated sleep spindle bursts and enhanced sleep quality (Sterman et al. 1970). Thus, Hoedlmoser et al. (2008) performed an SMR neurofeedback study to investigate the effects on sleep spindles and memory consolidation in humans and demonstrated effects on memory retrieval.

Regarding high-frequency bands in the domain of gamma oscillations, evidence suggests associations to visual local feature integration, binding, as well as to visual short-term memory (Tallon-Baudry and Bertrand 1999; Engel and Singer 2001). In studies conducted by Keizer et al. (2010a, b), participants learned to either upregulate their gamma band activity or to decrease their beta activity. Enhanced performance in feature integration was shown after gamma neurofeedback training, as was reflected in reduced binding costs.

Altogether, these results provide support that the modulation of endogenous oscillations is possible by neurofeedback and that such self-regulation transfers to enhanced cognition. For further readings about cognitive outcomes after neurofeedback see the review provided by Gruzelier (2014a).

Effects of Neurofeedback on Everyday Life Performance

Apart from the investigation of neurofeedback effects on cognition, studies have been conducted to investigate transfer to everyday life performance. For instance, Ros et al. (2009) trained ophthalmic microsurgeons with SMR neurofeedback training. With this study, participants demonstrated improved surgical skills after learned self-regulation of SMR as rated by experts regarding the overall surgery technique on the one hand, and shown on the other hand by increased performance in a suture task. SMR is also related to a maintained relaxed, but focused state, which is probably due to the reduction of motor perception processes of the sensorimotor cortex (Vernon et al. 2003). Thus, neurofeedback studies have also been conducted for sport performance, such as golfing (e.g., Arns et al. 2008). Cheng et al. (2015) investigated a sham-controlled SMR training with pre-elite golfers. Indeed, self-regulation of SMR in neurofeedback transferred to enhanced SMR power during action preparation while golfing, but crucially also to increased golf putting performance. However, as studies reported of specific brain activity such as the suppression of high alpha power immediately before successful movement

initiation for striking putts (Babiloni et al. 2008; Cooke et al. 2014), the down-regulation of alpha activity in golfers might depict a further possible neurofeedback protocol (Ring et al. 2015).

A further range of neurofeedback studies analyzing the effects on real-life performance has been performed in the domain of creativity in the arts (see for a review Gruzelier 2014b). Here, so-called alpha–theta trainings arose. These are based on mainly two associations of oscillations and cognition: first, alpha activity and its association with low arousal and diffuse attention (e.g., Fink and Neubauer 2006; Grabner et al. 2007; Bazanova and Aftenas 2008) and second, oscillations in the theta domain observed in states between waking and sleeping, which are supposed to ease creative processes (Schachter 1976). The alpha–theta training is typically conducted while participants close their eyes and learn to increase their posterior theta relative to alpha amplitudes. This protocol has been found to lead to enhanced music performance in professional musicians regarding their artistic expression (e.g., Egner and Gruzelier 2004), but also increased performance of novice musicians (Gruzelier et al. 2014a). Similarly, this alpha–theta protocol increased dancing performance of professional dancers (Raymond et al. 2005; Gruzelier et al. 2014b).

We will now turn to the conceptualization of self-control of brain activity and the underlying mechanism of changes in cognition and behavior induced by neurofeedback.

Conceptualization of Self-control of Brain Activity

Generally, a circuit involving the anterior insula, middle frontal gyrus, anterior dorsal cingulate gyrus has been identified that is responsible for these more general aspects of cognitive control that are activated equally by sham (Ninaus et al. 2013, 2015) as well as effective neurofeedback (Emmert et al. 2016). Moreover, basal ganglia structures such as the striatum have been related to the core learning processes occurring during neurofeedback (Birbaumer et al. 2013). Neurofeedback learning is more complex in humans than in typical animal models that are highly motivated by deprivation and rewarded with primary reinforcement. Humans have a much richer and active mind than other animals. As such, task models, strategies, verbalizations, self-referential processes, visual, sensorial, and emotional imagery, etc. may bias or even hamper more basic procedural learning in humans and can only be switched on or off by means of purposeful cognitive control (Wood et al. 2014). Therefore, control of brain activity during neurofeedback is more than merely learning to regulate the activity in one specific neural network that is targeted directly by neurofeedback. Neurofeedback learning also involves the ability to tune the activity in other large-scale networks that are not related to the production of the brain signal being trained but can hamper the learning process (Wood et al. 2014). Accordingly, neurofeedback learning seems to be more complex than mere procedural learning and rather the result of conjugated labor of different brain

networks (Wood et al. 2014) tuned to optimize the control of the specific brain signals under training by means of feedback, thereby giving rise to different forms of brain plasticity (Ros et al. 2014).

Neuroplastic Effects of Neurofeedback

Theoretically, the observation of voluntary control of particular measure(s) of brain function (e.g., spectral power) is in itself independent of demonstrating an impact on its plasticity. For neurofeedback, and in analogy to general learning, plasticity implies a progressive and long-term change—of at least >20–30 min (Schulz and Fitzgibbons 1997)—of a measure during or after training. From a historical perspective, seminal experiments in the 1960s reporting online control of the EEG (Kamiya 2011; Sterman et al. 1969) were followed by evidence that waking SMR may be operantly conditioned to be more strongly expressed during subsequent sleep (Sterman et al. 1970). This observation of oscillatory patterns may be modified by neurofeedback within and/or between training sessions, has now been confirmed by a collection of studies, and reported to apply to theta upregulation (Enriquez-Geppert et al. 2013; Sittenfeld et al. 1976) and downregulation (Lubar and Swartwood 1995; Monastra et al. 2002; Janssen et al. 2016), alpha upregulation (Cho et al. 2008; Zoefel et al. 2010; Escolano et al. 2011) and downregulation (Ros et al. 2013; Ros et al. 2010; Regestein et al. 1973), beta upregulation (Engelbregt et al. 2016; Staufenbiel et al. 2014), and gamma upregulation (Keizer et al. 2010). What remains unclear is the physiological mechanism responsible for the plasticity of these oscillatory patterns. Given that the effects manifest in the same direction as dictated by the neurofeedback protocol, a candidate mechanism may be Hebbian plasticity, often summarized by the phrase: "synapses that fire together wire together, and synapses that fire apart wire apart". This type of associative plasticity occurs when neuronal patterns are reinforced by functional association in time, and may be explained by the fact that EEG oscillatory amplitude positively covaries with the number of synchronized neurons/synapses (Musall et al. 2014). Consequently, during amplified oscillations, the population(s) of neurons which are coherently involved in generating an oscillatory pattern would, after some time, further strengthen the connections between themselves, thus making it easier for this population pattern to emerge in the future (Knoblauch et al. 2012). Conversely, maintaining a group of neurons in a prolonged desynchronized state would weaken the correlated firing of their synapses and attenuate the connections that give rise to synchronization. This mode of action is supported by several modeling studies of Hebbian spike-timing-dependent plasticity (STDP) (Knoblauch et al. 2012; Pfister and Tass 2010; Legenstein et al. 2008), as well as in vivo experiments demonstrating lasting synchronization (Zaehle et al. 2010; Vossen et al. 2015) and desynchronization (Tass et al. 2009; Adamchic et al. 2014) of cortical oscillations using endogenous patterns of stimulation.

On the other hand, another body of research points to the existence of a complementary form of plasticity which is anti-Hebbian, or homeostatic (Hulme et al. 2013). This appears to be the consequence of intrinsic regulatory mechanisms that prevent brain activities reaching extremes, such as pathologically high/low synaptic strengths or oscillatory states (Whitt et al. 2013; Fauth and Tetzlaff 2016). Put succinctly, this form of plasticity produces changes in the very opposite direction of training (or what could be expected from Hebbian mechanisms). One of the first observations within the context of neurofeedback was made by Kluetsch et al. (2014), who reported a paradoxical rebound of spontaneous alpha rhythm following its down-training in patients with post-traumatic stress disorder (PTSD). Since these patients were found to exhibit significantly low alpha amplitude at baseline relative to healthy subjects, it was proposed that this might well be a homeostatic response (Kluetsch et al. 2014) and/or the brain self-organizing to criticality by tuning its excitation/inhibition ratio (Ros et al. 2016). The latter interpretation was based on the significant recovery of scale-free alpha amplitude fluctuations (Ros et al. 2016), as well as prior evidence that neurofeedback alpha downregulation could enhance cortical excitability and lower intracortical inhibition, as measured by a lasting increase of transcranial magnetic stimulation (TMS) motor-evoked potentials.

Finally, EEG neurofeedback-induced plasticity has also been investigated using a different set of modalities, including functional magnetic resonance imaging (fMRI) and diffusion tensor imaging (DTI). fMRI has shown that neurofeedback may lead to plastic changes in cortical regions responsible for cognitive control such as the anterior cingulate, associated with improvements in attention-deficit (Lévesque et al. 2006) or on-task mind wandering (Ros et al. 2013). DTI has yielded data that makes a promising case for neurofeedback impacting white matter pathways, in addition to changes in gray matter volume (Ghaziri et al. 2013). Taken together, this collective work indicates a basis for harnessing neurofeedback as a neuroplasticity-based technique in health and disease.

Interindividual Differences in Neurofeedback: Responders and Nonresponders

The investigation on responders vs nonresponders is a subtopic of the more general question of individual differences in neurofeedback. While review papers are full of examples of positive neurofeedback effects, only a few studies so far have investigated negative effects of neurofeedback systematically (e.g., Kober et al. 2015a). About 15–30% of participants do not show neurofeedback learning in EEG-based studies. Such estimates have not been reported yet for other brain signals such as the blood-oxygen-level-dependent (BOLD) response, but there are reports suggesting that responsivity to real-time fMRI feedback training is higher than 70%. One may distinguish at least four reasons for individual variability in the responsivity to neurofeedback, which are the following:

Physical reasons: Signal detection can be poor because of anatomical abnormalities or idiosyncrasy (Allison and Neuper 2010). EEG signal power can show dramatic variation depending on the position of the brain relative to electrodes (Rice et al. 2013). Accordingly, movement artifacts as well as brain shape may change the individual responsivity neurofeedback.

Physiological reasons: In a recent study, predictors of performance in a SMR frequency EEG neurofeedback were investigated. Findings indicate that more than age or sex, SMR signal intensity is predictive of poor learning (Reichert et al. 2016a). Participants with lower levels of SMR power over the central electrodes were less able to learn to increase the SMR power along the course of 10 training sessions.

Cognitive reasons: Reserve capacity may boost the effect of neurofeedback training (Reichert et al. 2016b, c). Some people adept some specific concentration practices seem to be more able to regulate their internal environment and to benefit more from neurofeedback learning. In a recent study, 28 triathletes learned more during even a single session of neurofeedback training than 28 matched healthy controls (Witte 2015). Accordingly, 20 Christian participants adept of intensive praying also learn more during a single session of neurofeedback than 20 matched controls (Kober et al. 2015c). A recent study also indicates that mindfulness may facilitate neurofeedback learning (Kikkert 2015).

Metacognitive reasons: Levels of perceived locus of control toward technology predict learning (Witte et al. 2013). Spontaneous strategies are associated with learning success (Kober et al. 2013). The length in words of the learning protocols of young healthy participants correlates with learning effects. The more succinct the answer to the question "what have you being doing during neurofeedback training", the better are the training outcomes ($r(65) = 0.4$, unpublished data). These pieces of evidence suggest that the individual task model (i.e., "how do I solve the task of learning from neurofeedback?") is in part responsible for individual training outcomes. The more graspable the contents of the training instructions for individuals, the more consistent are the results across individuals. Neurofeedback training protocols based on some form of mental imagery that can be easily understood and uniformly implemented will therefore also produce more uniform results than neurofeedback protocols based on some less-specific mental state such as for instance "being relaxed but concentrated". Interestingly, some brain signals seem to respond better to training when instructions are vaguer and cannot be forced to respond to a specific set of instructions (Hardman et al. 1997).

Specificity and Efficacy

The discussion about nonresponders can also be embedded in a somewhat larger context, namely when addressing those factors that determine the overall efficacy of neurofeedback. What factors constitute a training that maximizes the pre- to post-changes in neural parameters and behavioral performance measures? Whereas

there is no definitive answer to this question (yet), it is worthwhile considering some factors most likely contributing to neurofeedback efficacy. Since neurofeedback usually aims at the enhancement of a specific cognitive function, it seems straightforward to optimize those neural systems and processes that give rise to these cognitive processes. EEG-based protocols usually try to achieve this by extracting the activity of one or two frequency bands from a selected number of electrodes and instructing the participants to up- or downregulate the amplitude or the activity ratio of these bands (Gruzelier 2014c). As mentioned earlier, substantial interindividual differences in brain morphology may obscure the purity of so-derived features, as does the fact that it is rather unlikely that cognitive components can easily be tied to such narrowly defined features alone. Current approaches thus neglect the many facets of neural signals that have meanwhile been linked to cognition, such as cross-frequency coupling, inter-regional communication, and gross brain connectivity as inferred from large-scale connectivity analyses (e.g., Sauseng and Klimesch 2008; Sporns 2014). Similarly, neurofeedback could be optimized further with regards to maximizing the effects in terms of neural plasticity discussed earlier, and it is likely that induced plasticity closely relates to those neural mechanisms providing the underpinnings of cognitive processes in the first place. To date it is largely unclear how to best address any given neural system and its means of neural communication. For example, what would maximize neurofeedback efficacy aiming at cognitive control: The upregulation of fm-theta alone, or the maximization of the theta-to-alpha ratio? Such comparative studies are needed for every targeted process, yet they seem to be hardly ever conducted. Exhibiting the relevance of such work, Salari et al. (2013) found that neurofeedback for both alpha and gamma upregulation enhanced object-recognition, but object detection was more strongly influenced by the gamma-based training. Another approach to increase neurofeedback specificity, i.e., the ability to modulate the targeted system alone, may be to compute feedback in the source rather than the electrode space. Note that any recording of EEG activity at a given electrode, even that within well-circumscribed frequency bands, always reflects the activity summed across many different brain regions. Thus, combining frequency-specific feedback with EEG source analyses may well minimize the influence of cross-talk from other brain networks in our feedback signal, thereby potentially optimizing neurofeedback efficacy. A number of studies have been conducted following this notion, all combining Low Resolution Tomography (LORETA) for EEG inverse modeling with feedback based on alpha or beta activity as extracted from the midcingulate region (e.g., Congedo et al. 2004; Cannon et al. 2007, 2009; Maurizio et al. 2014). A conceptually similar methodology was tested by Zotev et al. (2011, 2014), who combined EEG feedback of beta band activity with simultaneously recorded activations of the amygdala as measured via fMRI. The common element of all these studies is that increased neurofeedback specificity may be achieved by enriching standard EEG frequency features through spatial filters. Yet again, comparative studies are needed to assess whether increases specificity indeed leads to increased efficacy of neurofeedback.

Another group of factors likely to affect neurofeedback efficacy does not so much relate to neural processes per se, but rather to the optimization of training designs in accordance with principles underlying basic learning mechanisms. For example, whereas some studies do report that significant feedback-related learning may already be found after a single training session, the majority of studies seem to indicate that reliable training effects occur after about ten training sessions (see Gruzelier 2014c, for a review). Also, established work on reinforcement schedules would suggest that a relatively early transition from continuous to intermittent reinforcement would optimize training outcome, yet this notion has not yet been tested in context of neurofeedback. This issue does relate to both the temporal spacing of feedback signals within a single session (e.g., continuous feedback vs. blocked feedback) and the scheduling of training sessions across days and weeks. Neither of these two phenomena has yet been tested systematically, although the notion that neural plasticity through synaptic consolidation and reorganization need time clearly supports the validity of these basic learning mechanisms also in neurofeedback contexts. Again, however, systematic studies are missing and only rather anecdotal evidence is available in favor of this notion (e.g., Schabus et al. 2014).

Conclusion

A necessary basis for the enhancement of cognitive functions with neuroscientific approaches are theoretically and empirically confirmed associations of brain activity and cognition. By feeding back neural parameters of ongoing neural activity to the participants on a trial-by-trial fashion, self-regulation of brain activity can be achieved. The self-control of brain activity in humans has been suggested to be more complex than in the animal model, and to exceed mere procedural learning. Thus, different brain networks might be engaged to adjust control over a brain signal during neurofeedback training. Regarding the physiological mechanism responsible for neurofeedback-induced plasticity, which might even impact brain morphology; two forms are in focus, (1) Hebbian/associative plasticity and a complementary form, which is known as (2) anti-Hebbian/homeostatic plasticity. Regarding the responsivity to neurofeedback large individual variability has been reported and four different reasons have been suggested to play a role. Notable are physical (poor signal detection), physiological (initial signal intensity of the brain feature), cognitive (reserve capacity), and metacognitive reasons (training instructions and strategies of self-regulation). The responsiveness to neurofeedback and hence its efficacy may further be moderated by methodological factors. One such group of factors considers how to best address a given neural system and its means of communication. Here, the combination of frequency-specific feedback with EEG source analysis offers one approach. A further group of factors focuses on the optimization of training designs that follow the principles of basic learning mechanisms. Thus, whereas many factors can be derived from our knowledge on

the neural underpinnings of cognition, available measurement techniques, as well as basic learning mechanisms, much more systematic work needs to be conducted to optimize neurofeedback protocols for basic research and clinical applications.

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