

Our sample was far too small and too heterogeneous regarding the variety of comorbid disorders for such subgroup analyses.

4. We did include psychometric assessments. Actually, an article on psychometric data in the same sample has already been published.² This article also reports on potentially individual changes on the psychometric level.

In conclusion, ADHD is in itself a heterogeneous disorder, making the title of the article not misleading. Further, this study addresses the efficacy of EEG neurofeedback on behavioral and global clinical functioning. The clinical points reflect the results of this research question, which are supported by a recent meta-analysis,⁶ and include a direction for future research regarding subgroups. Thus, the title and the clinical points seem accurately chosen, and adjustments are therefore regarded as unnecessary.

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Dr van Dongen-Boomsma and Colleagues Reply

To the Editor: Thank you for the opportunity to respond to the letter from Dr Cannon and colleagues about our recent article.¹

1. Reinforcement was 80% per training target, hence it occurred only when all training targets were achieved simultaneously. When no correlation in activity between the different training targets was assumed, the reinforcement was $0.8^{\text{number of training targets}}$ (eg, training θ power downward and β power upward resulted in a minimum rewarding percentage of 64%). In practice—due to overlapping targets—reinforcement was between $0.8^{\text{number of training targets}}$ and 0.8. Regarding the electroencephalographic (EEG) learning paradigm, EEG data were saved during the sessions for the active condition of the second part of the sample. For these data, we analyzed the difference between the first and the last training session. These results were published in a recent article of ours² and showed that children were not able to train their EEG targets as desired. In addition, clinical responders did not show EEG improvement on the trained EEG targets.
2. We agree that analyzing the pretreatment and posttreatment EEG would give neuroscientific insight into potential electrophysiologic changes after EEG neurofeedback. However, our main outcome variable was the severity of attention-deficit/hyperactivity disorder (ADHD) symptoms, as measured on the ADHD Rating Scale IV.³ We were unable to document the superiority of EEG neurofeedback over placebo neurofeedback on this main outcome variable. It is not typical that EEG neurofeedback studies report whether EEG changes have been brought about. In fact, if it is crucial to establish acquisition at the neural level before examining clinical effects, as these authors claim, it is surprising that this is not common practice in clinical reports on the efficacy of EEG neurofeedback.
3. Indeed, our sample was clinically heterogeneous and included children with a primary ADHD diagnosis who also had 1 or more comorbid disorders. The study was designed to allow for some comorbidity, since comorbidity is the rule rather than the exception in children with ADHD.^{4,5} As we aimed to investigate the efficacy of EEG neurofeedback in ADHD as observed in daily practice, we did not want to exclude children with common comorbid disorders. Doing so would detract from the representativeness of our sample and have a negative impact on generalizing our results to daily practice. We strongly disagree with the statement that inappropriate EEG neurofeedback protocols were used. Our protocols were determined on the basis of individual deviation patterns, making this argument invalid for not finding a treatment effect. To investigate if subgroups of children with ADHD do benefit from EEG neurofeedback is a sensible suggestion for future research, as this would enable analyses on the influence of comorbid disorders.