Modeling Behavior of Attention-Deficit-Disorder Patients in a N-Back Task

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Abstract

Cognitive performance in memory tasks, as measured by the N-back task, shows large differences between attention deficiency disorder (ADD) patients and controls. Recent findings indicate fewer anatomical differences, which, in turn, makes a cognitive modeling of the inherent information processes possible. In this article, we investigate these aspects for 0-back and 1-back problems and introduce a new cognitive model explaining differences between patients and controls. The ACT-R model explains the results by differences in the utility and reward function. The presented methodology – using cognitive modeling for controls and patient groups – works toward two goals: (1) A potential explanation of a source of differences for a medically relevant group and (2) to present and evaluate a new approach using cognitive modeling in psychiatric research areas to identify more detailed descriptions of mental differences.

Keywords: N-Back, ACT-R, Attention Deficiency Disorder, Working Memory, Modeling of Disorders

Introduction

The so-called N-back task is used as a psychological tool to determine individuals' cognitive performance on (spatial) working memory problems. The participant is presented with a sequence $a_1, \dots, a_m$ of letters one at a time. In a 1-back task, the participant is required to press a button if the letter being presented (a_i) is identical to its direct predecessor (i.e., if $a_i = a_{i-1}$ for the 1-back task). An N-back problem requires a comparison of the current letter (a_i) with the letter presented N-steps back (a_{i-N}). To keep the predecessor and successor in memory can require some sort of spatial representation. From a psychological perspective, a successful solution requires concentration and the ability to keep track of a number of items in working memory. A central finding is that response latency increases and accuracy decreases with increasing memory load (Braver et al., 1997; Cohen et al., 1994; Lovett, Daily, & Reder, 2000). Performance errors can be classified as errors of omission and errors of commission. An error of omission is committed if the participant does not press the button although the letters are identical and an error of commission if the participant presses the button although the letters to be compared are different. Errors of omission rarely appear for smaller N-back tasks (Braver et al., 1997), but are generally more likely than errors of commission.

Attention Deficiency Disorder (ADD) patients may perform more errors of commission as they are primarily characterized as having a much higher level of inattention, distractibility, and impulsiveness (Pary et al., 2002; Barkley, 1998). Although the exact biological or cognitive mechanisms are still unknown, a number of involved pathways have been discussed (Pary et al., 2002). ADD can negatively affect the educational and social performance of those suffering from its symptoms (Pary et al., 2002) and it is one of the most common mental disorders, according to data from the NIH (NHS, 2008). Although there are differences in brain development, there are no known anatomical differences. An interesting insight we can draw from these findings is that attentional problems may depend on internal information processing rather than on physiological aspects of the brain. With this in mind, it seems possible to model this effect with a symbolic cognitive model. In order to test this hypothesis we decided to model the results of a study conducted by Klein, Wendling, Huettner, Ruder, and Peper (2006). The methodological approach of comparing clinical abnormalities with controls on a level of abstraction to identify cognitive distinctions within a cognitive model is, as described in this paper, a fairly new approach. For this reason, the general procedure in this study can serve as an example for other fields of clinical diagnostics especially for cognitive disorders. Similar approaches with different starting point question could be found in Hussain and Wood (2009).

State-of-the-Art

The Experiment by Klein et al. (2006)

We briefly report the empirical findings from Klein et al. (2006). They investigated different cognitive parameters of intra-individual variability to identify subgroups of ADD–patients in comparison with controls. All members of the patient-group (57 subjects) were patients from "Caritashaus Feldberg" a clinic specializing in the treatment of ADD diagnosis and met the criteria according to ICD-10 at the time of the study. They were diagnosed by experienced clinical psychologists and psychiatrists on the basis of Conners' parent and teacher rating scales (Steinhausen, 2000). The patient group included 49 boys (85.9 %) and 8 girls (14.1 %) with a mean age of 126.4 ± 21.2 months (range: 84-169 months) and a mean IQ of 96.6 ± 13. The control group was matched to the patient-group. For this reason the controls do not differ in the mean age (126.9 months ± 21.7) or gender distribution (8 girls, 45 boys). Only the mean IQ (110.2 ± 12.82) was significantly ($t\ 108 = 5.42, p = .001$) higher than in the patient-group. Further group descriptions are given in Klein and colleagues (2006).

Design, Method & Procedure. Both groups were tested with 0-back, 1-back, and 2-back problems. In each condition

(0-back, 1-back, and 2-back tasks) the participants were presented with exactly 100 trials. Each stimulus was presented for 0.5 seconds and the next stimulus was presented 2.0 seconds later. The "event" condition in the 0-back condition was the presentation of the letter "E" while all other letters were characterized as "nonevents." In the 1-back condition, an event occurred if the letter being presented (a_i) was identical to the letter presented one step earlier (a_{i-1}), as characterized above. An event occurred randomly in about 20% of the trials. Ten practice trials preceded the 0-/1-back tasks; 20 the 2-back tasks. Subjects had to press the right-hand response button as quickly as possible for events, and the left-hand response button for nonevents.

Results. The authors decided to eliminate the 2-back condition as 17 patients and 5 controls had difficulties understanding the task. Therefore, they only report the results for 0-back and 1-back tasks. Three of the cases showed significant group differences, where the differences stand out in the 1-back condition. All results can be found in Figure 1. For further information please refer to Klein et al. (2006).

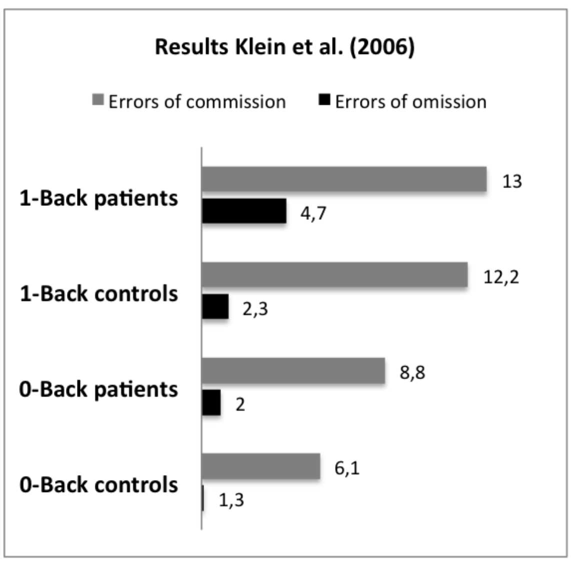

Figure 1: The empirical results for both ADD-patients and controls from the study of Klein and colleagues (2006).

ACT-R. The cognitive architecture ACT-R 6.0 (J. R. Anderson et al., 2004; J. Anderson, 2007) provides a number of working memory specific modules which are in turn associated with specific cortical regions. The smallest information unit is called a chunk. Chunks are modified by mental operations. These mental operations are associated with so-called production rules, which consist of a condition and an action part. The procedural module controls ACT-R's strictly serial behavior; only one production at a time can fire. The likelihood that a production rule can fire depends on several factors: First of all, the condition of the rule must be satisfied. If several production rules compete then noise and the production rules' utility performs the selection process.

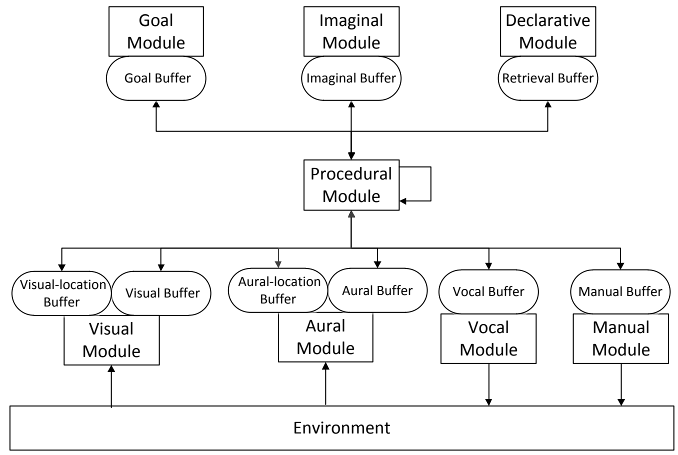

Figure 2: Overview of the different buffers and modules in the cognitive architecture ACT-R 6.0. The symbolic structure is represented by productions while subsymbolic parts control the execution of productions (based on utility) and the velocity of information retrieval from declarative memory and are therefore especially responsible for learning.

There have been several attempts to model the N-back task in ACT-R. One of the first cognitive models for the N-back was presented by Lovett et al. (2000). They identified two qualitatively different strategies used by participants: The so-called activation strategy, where participants respond 'match' if a letter seems familiar. A second strategy – the update strategy – involves actively maintaining a list of prior letters and updating that list after each letter is presented. They concluded that, working memory resources are not involved in the first strategy as no maintenance is involved, but are involved in the second. The ACT-R task model for the N-back task includes all the declarative and procedural knowledge necessary for performing this task according to the update strategy. In this model, each stored letter was represented as a declarative chunk indexed according to how many letters back it was from the current letter. They used mostly default values (see Anderson and Lebiere (1998)) and set activation noise to 0.04 and the retrieval threshold to 1.80 to optimize the fit to the data. Individual differences between participants could be captured by the previously attained source activation, parameter W, a type of attentional activation that is divided equally among the items in the current focus of attention. It spreads from these items to related chunks.

Juvina and Taatgen (2007) empirically investigated N-back tasks where subjects received feedback for each action. They successfully modeled the influence of feedback on learning rates. They found a significant change in the relation between omissions and commission error rates – as in Klein and colleagues (2006).

Aim of this research approach

The aim of this research approach was to identify relevant parameters with an influence on the deficit in cognitive performance and to provide a framework for further research approaches. Relevant symbolic processes should be identified with different kinds of modeling. The database from Klein et al. (2006), that examined ADD-patients and controls, served for the validation of the different models of the N-back task.

In addition, we briefly review the existing cognitive models for the N-back task. We discuss our findings in comparison to the experiment by Klein et al. (2006) and draw conclusions for further medical methods.

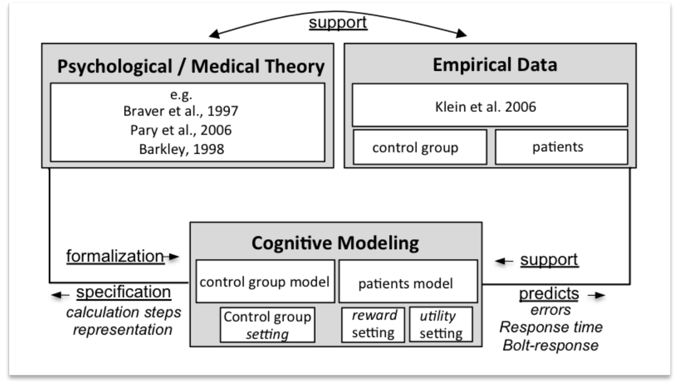

Figure 3: Research approach in this study. Empirical data for control group and patients were taken from Klein et al. (2006). Our modeling is orientated on these results. In the "modeling part" we introduce two different models for the patients group to predict the ADD group results.

Methodology

In the following our research and methodological approach are introduced (see Figure 3). Note that the following elaborations of the cognitive modeling are all based on the empirical findings by Klein (2006) described in the "state of the art" part in this paper.

The Cognitive Model

One of the main conclusions we draw from the previous experiment and the literature is that ADD-patients may have a higher inquiety (see above and Pary et al., 2006). This *inquiety* shows itself in the behavior of the participants: in a heightened probability of pressing a button (to commit an error of commission) and a slightly greater level of difficulty in retrieving information from declarative memory. This is due to a greater amount of noise and results in errors of omission. The noise levels in our two patients models vary only slightly (.35 vs .362) in comparison to the control group model. Higher inquiety has not yet been modeled (e.g., Gunzelmann, Moore, Gluck, Van Dongen, & Dinges, 2009). We decided to represent the increasing inquiety by a higher negative utility or differences in the reward parameter. Our models do not assume other differences. As shown in Figure 6, the two patient-models (reward/utility) both lead to the results found by Klein (2006). Therefore these two are different forms of the original model (control group) with differences in only one specific parameter. These parameter differences are shown in Figure 4 and Figure 5.

Stimulus presentation times were set – as in Klein (2006) – to 0.5 seconds with 2 second breaks between the stimuli. The probability for N-back occurrence was set to 20%. Each participant evaluated a trial of 100 stimuli.

The 0-back task

In the 0-back task participants were required to find a specific letter in the given series of letters.

The variability of results is caused by specific parameter settings (as indicated in Table 1). Overall, two settings were considered as a plausible explanation for the error structure of the patients group: a "reward-model" and a "utility-model." In these two models, either the reward parameter or the parameter of utility was varied (see Figure 4). Both models can explain differences in performance between patients and controls (see Figure 3). From a psychological perspective arguments for both models are possible. On the one hand, the "utility-model" justifies the higher number of errors of commission by patients through a more intense use of the "N-back-found" production than in the control group model. On the other hand, the "reward-model" explains this particular error pattern with different reward setting. On a psychological basis this would mean the use of the "N-back-not-found" production has a more negative reward. Each "no-event-sequences" will decrease the use of the "N-back-not-found" production and this results in a higher amount of commission errors.

In this sense patients and controls differ in the following sub-symbolic parameters (see Table 1 and Table 2). As already mentioned in the "Cognitive Model" section you can see the different parameter settings in the differences between the two patient-models in Figure 4. For example a Δ0 in the parameter reward (Δ R: 0) means that the fitting in this parameter for that model is not different from the two patient models or the control group model. A Δ U: 1.95 in the "N-back-found" production means that the patient utility model has an 1.95 higher utility parameter than the control group model and also than the reward model, because their utility was not modeled.

Table 1: Parameters used to model patients and controls for the 0- & 1-back task.

	egs	:esc	:rt	:lf	:ans	:bll
Controls	1	t	-.5	0.25	**0.35**	0.5
Patients	1	t	-.5	0.25	**0.362**	0.5

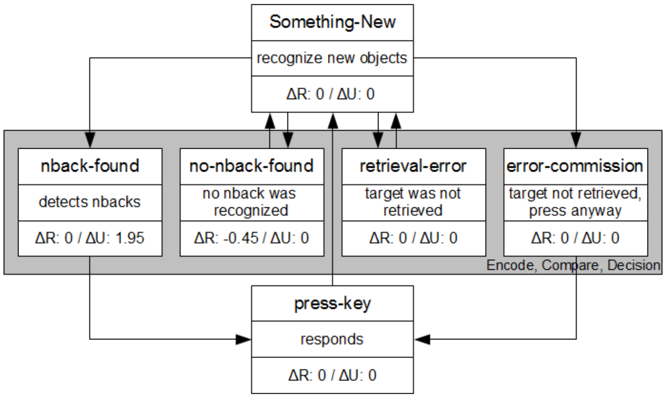

Figure 4: Diagram of the 0-back model "Something-new" and "press-key" productions and the encode-, compare- and decision-layer with its productions in the center. Δ R describes the difference between the patient and control group models in the parameter "reward" (R(patients)–R(control group) = ΔR) for each specific production. This situation also applies to the alternatively designed utility model (ΔU) of the 0-back task.

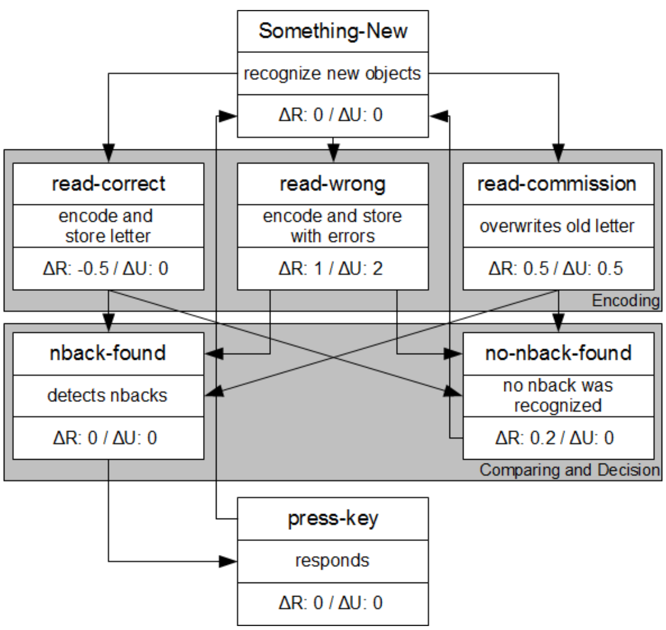

Figure 5: Diagram of the 1-back model. "Something-new" and "press-key" productions and the encode-, compare- and decision-layer with its productions in the center. Δ R describes the difference between the patient and control group models in the parameter "reward" (R(patients)–R(control group) = ΔR) for each specific production. This situation also applies to the alternatively designed utility model (ΔU) of the 1-back task.

The 1-back task

In the 1-back task participants have to press a button, if the letter being presented is identical to the previous letter. This task differs to the previous one so we included more production rules – because it cannot be reduced to a simple "retrieve-model" like the 0-back-task.

For this reason we introduced a more complex representation. Thus, the encoding layer is now separated from the comparing and decision layer and is also a possible source of errors.

As described in the explanation of the 0-back-task, we created two models that explain the differences between patients and controls. One model only uses the differences in utility and one uses the reward parameter (see Figure 5).

Table 2: The base level activations for each stimulus in the 0-back task. Target stimulus for the remaining letters.

	target stimulus	remaining letters
Baselevel activation	50	0.5

Results and Discussion

The present modeling replicates data from Klein (2006) satisfactorily (see Figure 6). Although both models match the original data in a sufficient way, the "reward-model" provides the better data. On the one hand, the scatter of the data in the "reward-model" is less around the mean of the behavioral data, and also comes closer to this. Also working memory components in the "compare and decision" layer in the 0- and 1-back-models like the "nback-found" production do not differ in the two groups looking at the "reward-model"(see Figure 6). This showed that there are no parameter differences in this "execution parts" of the "reward-model", but therefor in the reward of inhibitory parts like the "no-nback-found" production, which results in this specific arrangement of errors. This so created inquiety or impatience is the main cause of the specific error pattern. Impatience is also mentioned in relation with a phenomenon called "delay aversion." Delay aversion is a behavior that was investigated in connection with ADD and ADHD (Sonuga-Barke, Taylor, Sembi, & Smith, 1992). Our results indicate that the specific error pattern that ADD patients produce in an N-back task could be traced back to variations in the reward parameter of production rules. This could mean that even at this subconscious level "reward similar actions" are responsible for the underlying pathological behavior. The fact that this small variations in the reward parameters, could affect working memory capacity in a cognitive task was shown by the "reward model". A selective control of the occurrence time concerning the N-back targets could clarify this question more. Longer "no-event phases" in the N-back task should lead to larger number of errors in the patient-groups than in control groups.

General Discussion and Outlook

There are only a few approaches to modeling different patient groups in ACT-R. Thus far, there are – to the best of our knowledge – no (symbolic) cognitive models of working

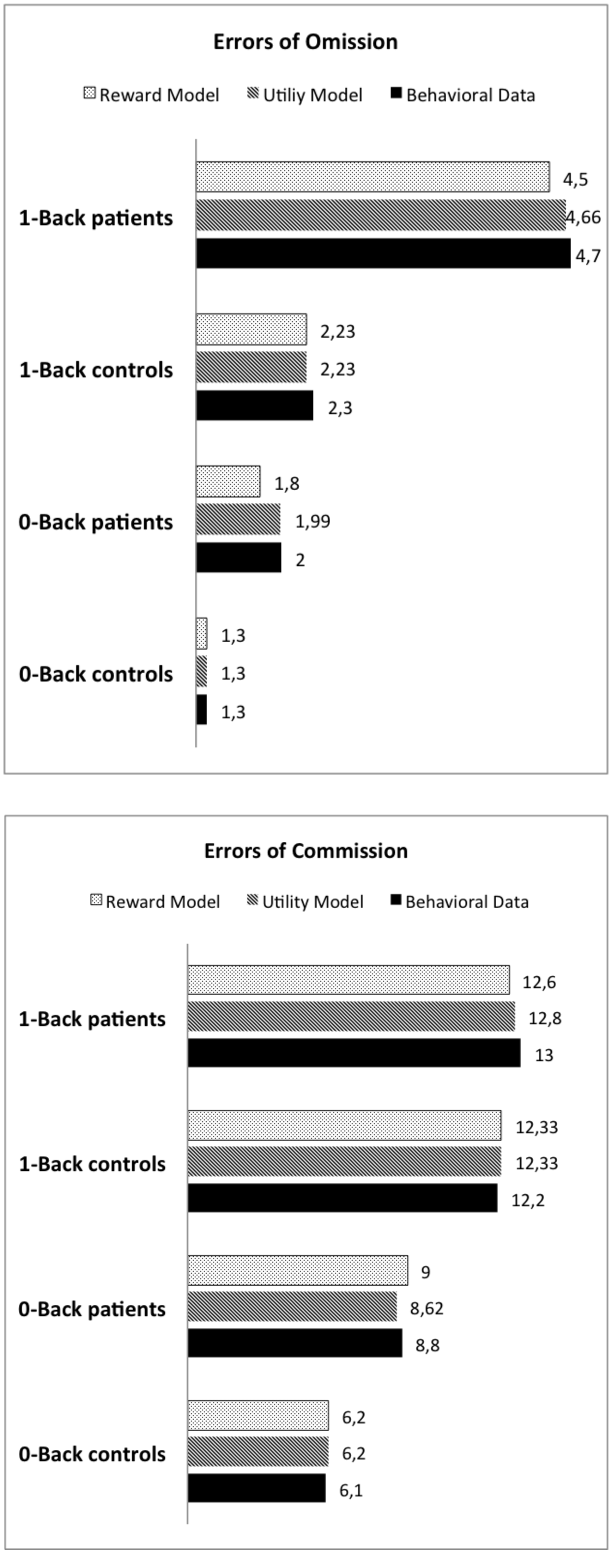

Figure 6: The identified errors of omission- and commission both for ADD-Patients and the predicted ACT-R data for the 0-back and 1-back task.

memory differences in ADD-Patients. One reason might be the difficulty of assessing differences in the attention span. Most cognitive models have assessed this purely by differences in parameter settings. ADD-patients represent an interesting and increasingly important population. By analyzing empirical data we decided to model increasing inquiety (resulting in a heightened probability of pressing a button (to commit an error of commission) and a slightly greater level of difficulty in retrieving information from declarative memory) by increasing negative utility. In the modeling process, it became apparent that there is at least an additional explanation pattern, namely, in differences with reward (keeping utility constant). A second "reward"-model was able to capture the empirical differences. Both models make a satisfactory prediction of the results and not only in parametric distances. This goes along with analysis from (Roberts & Pashler, 2000). He argued that parameters might give a good hint about a cognitive model if they constrain the outcomes. In this way, we have differentiated between two likely model. But, both models differ regarding an important aspect, namely the length of the presented sequence. The higher the length of the total sequence of presented letters, the higher the errors the reward-model would predict but not the utility model. A further prediction would be: Most psychological studies have presented participants with a random function based on the probabilities with which an event occurs. But, based on the cognitive modeling, we would predict that higher pauses between events might trigger more commission errors in the patient groups than in the controls. These questions have not yet been investigated empirically or reported in the literature. This shows the power of the cognitive model approach's ability to make clear predictions based on an algorithmic implementation, which can, in turn, be empirically investigated in human experiments and suffice to discern different theories.

Our modeling approach is purely information-theoretic, i.e., it does not require anatomical differences nor does it explain the effects solely by parameter settings. Recently, Gunzelmann and colleagues (2009) modeled the decline of cognitive ability by sleep-deprived soldiers in an attention task. They applied a Psychomotor Vigilance Test (Dinges & Powell, 1985; Dorrian, Rogers, & Dinges, 2005). The task of the participants was to monitor a known location on a computer screen and to press a response button each time a stimulus appeared at random intervals between 2 seconds and 10 seconds. Sleep deprivation had a severe effect on performance due to decreased alertness. Differences between groups were explained by differences in parameters. Certainly the parameter fitting process provides no explicit information about prevalent biological differences, but several findings can still be derived in this sort of modeling.

Unsurprisingly, results showed that a higher level of complexity in the N-back task leads to a change in the relationship between omission- and commission errors. Our hypothesis in this context argues that at whenever a complexity-related omission error is committed, a commission error is counted. This only applies to 2-Back and higher N-backs because of a probable miscounting. This has not been reported so far, but might indicate that this aspect has led to a distortion of reported empirical findings in the literature. These findings indicate that there is a great importance to develop a trustworthy method to identify the reasons for an error to occur. Effects of training of working memory by a dual-version N-back tasks on intelligence has been investigated (Jaeggi, Buschkuehl, Jonides, & Perrig, 2008). Nab et al. (2009) reported changes on a neuronal level using the N-back task also for cognitive training. These two findings reveal further investigations on the N-back task to develop new therapeutic methods for ADD. Future work will investigate the differences regarding neurological predictions (fMRI-Analysis) and the modeling of subgroups of ADD-Patients. Cognitive disorders might be – at least in a non-negligible part – traced back to differences in mental model operations, which are linked to the production buffer that has been associated with basal ganglia. Further psychological and medical research (to explain the underlying medical condition) is necessary.

Acknowledgments

The authors are grateful to Christoph Klein (University of Bangore, UK) for various helpful discussions. This research was partially supported by the DFG (German National Research Foundation) in the Transregional Collaborative Research Center, SFB/TR 8 within project R8-[CSPACE].

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