

**Cognitive Control Strategies in ADHD: Shifting Between Proactive and Reactive
Modes in Dual-Task Conditions.**

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Abstract

Dual-task interference arises when consolidating a first target (T1) while needing to respond to a secondary target (T2) in rapid succession. This either leads to delayed responding to the secondary target (proactive interference) or reduced accuracy in primary target performance (retroactive interference), depending on the probability of interference between them. The present study examined how proactive and reactive control processes differ between individuals with elevated Attention-Deficit/Hyperactivity Disorder (ADHD) symptoms and controls. Our sample (N = 69) completed an online experiment in which the probability of T2 occurrence (50% vs. 100%) and stimulus onset asynchrony (SOA) were manipulated, while T1 accuracy on a memory consolidation task and T2 reaction times on a numeric task were measured. A significant interaction effect was observed between T2 probability and SOA for both T1 Accuracy and T2 RT, indicating that cognitive control is modulated by the probability that T2 occurs in those with low ADHD symptoms; a proactive shift when interference is certain, and a reactive shift when it is uncertain. No evidence was found for high ADHD scores leading to reduced retroactive or proactive interference modulation by T2, providing no evidence for reduced adjustment of cognitive control based on interference.

Keywords: ADHD, cognitive control, dual-task interference

Cognitive Control Strategies in ADHD: Shifting Between Proactive and Reactive modes in Dual-Task Conditions.

Working memory, defined as the ability to hold and simultaneously process information over short periods of time (Baddeley & Hitch, 1974), is limited in its capacity. Dual-task interference occurs when a primary consolidation task (T1) is shortly followed by a secondary numeric reaction-time task (T2). Earlier studies show that while perceptual processes operate in parallel for different stimuli at the same time, later operations, such as selecting a response or consolidating a stimulus into working memory, involve a central process that operates serially (Zylberg et al., 2010). Some studies explain dual-task interference through an attentional bottleneck; a strict, serial all-or-none consolidation process responsible for the postponement of a secondary task (T2) until the primary task (T1) is fully consolidated (Jolicoeur & Dell'Acqua, 1998).

Conversely, capacity-sharing models suggest that T1 and T2 can be processed in parallel, while they compete for a limited capacity of attentional resources. Here, interference arises from how much of that limited capacity is allocated to each task, rather than from a structural bottleneck (Tombu & Jolicoeur, 2003). Instead of an all-or-none process, people strategically reallocate attention by either interrupting T1 consolidation to respond to T2 or finishing T1 consolidation before processing T2 (Tombu & Jolicoeur, 2003; Ricker et al., 2018). Thus, bottleneck and capacity-sharing theories seem to offer different explanations for the same dual-task interference effects, whereby the former sees central processing as serial and indivisible, while the latter views it as a flexible, graded allocation of resources.

Nieuwenstein and colleagues (2025), in their adaptive attentional shielding hypothesis, specify how secondary task expectancy shapes the trade-off in dual-task interference. When the secondary task T2 is certainly expected, the system “shields” consolidation of the first task (T1), leading to proactive interference. In this case, T1 accuracy is intact, while T2 reaction

time suffers. Conversely, when T2 is not certainly expected, T1 does not get shielded, and retroactive interference occurs; reaction time on T2 is preserved, while T1 accuracy suffers.

The interference between T1 and T2 is contingent on SOA and the probability that T2 appears, where at short SOAs, T1 accuracy is worse when T2 appears only 50% of the time, than when it appears 100% of the time, and this difference vanishes at long SOAs. On the other hand, reaction times on T2 show large-SOA dependent slowing when T2 appears 100% of the time, compared to the 50% condition, where T2 reaction times do not change much between SOAs. In short, the mechanism suggests that shielding is deployed adaptively, such that which target is shielded against interference depends on the probability that such interference occurs (Nieuwenstein et al., 2025).

The Dual Mechanisms of Control (DMC) framework is a contemporary theory of underlying mechanisms in adjusting response strategies and consists of two separate modes. Proactive control reflects the prolonged and foreseeing maintenance of goal-relevant information within the lateral prefrontal cortex (PFC) to enable optimal cognitive performance, whereas reactive control reflects transient stimulus-driven goal reactivation that recruits lateral PFC on interference demands or episodic associations (Braver, 2012). The DMC provides a comprehensive explanation of three sources of variation encountered in cognitive control research: intra-individual (task-related), inter-individual (trait-related), and between-group (population-related).

The intra-individual source suggests that individuals adjust their use of cognitive control based on expectations about interference during working memory processing (Braver, 2012; Burgess & Braver, 2010). In a recent probes task where PFC activity was measured, there was an indication of proactive control engagement in high interference conditions, whereas there was reactive engagement in low expectancy conditions (Burgess & Braver, 2010).

It seems that people engage proactively with high expected interference on trials, like expecting a high proportion of incongruent words on a list, as seen in the Stroop task (e.g., the word “red” printed in green ink), by filtering out the color from the meaning of the word (Bugg, 2012). Reactive engagement may be observed during low expected conflict situations, such as a list with a low probability of incongruent items. Here, conflict between the word meaning and color is not anticipated; participants recruit control in a stimulus-driven manner, with slower reaction times and more errors on the incongruent words (Bugg, 2012).

These findings are parallel to those in the AX-CPT task literature, where participants provided with cues about upcoming probes showed shifts toward proactive control, maintaining task goals in advance and preparing responses, reflected by improved performance on anticipated BX trials and decreased performance on subsequent AY trials (Paxton et al., 2006; Braver et al., 2009; Edwards et al., 2010).

Intra-individual observations and the aforementioned findings suggest a shift towards proactive reliance under high interference expectancy, as when T1 accuracy was protected when T2 appeared 100% of the time, reflecting sustained task-goal maintenance. A reactive control shift occurs when interference is not anticipated, leading to transient stimulus-driven responding, as evidenced by less effective T1 shielding when T2 was present 50% of the time, reflecting less sustained goal maintenance in working memory (Braver, 2012; Bugg, 2012; Nieuwenstein et al., 2025).

The between-group assumption of the DMC suggests that there is variability in cognitive control function between people, with a generic shift from proactive towards reactive control across some populations - not as a global impairment, but as a differential reliance (Braver, 2012). There seem to be altered cognitive control strategies in clinical populations, including people with schizophrenia, depression, and people with Attention-Deficit/Hyperactivity Disorder (ADHD) (Burgess et al., 2010; Braver, 2012).

ADHD is a neurodevelopmental disorder, typically marked by difficulties in executive function and compromised functions such as those of planning and response inhibition (Willcutt et al., 2005). Attentional control and working memory function depend on similar neural structures in the brain, many of which are found to be impaired in individuals with ADHD, such as the dorsolateral prefrontal cortex (DLPFC). (Dickstein et al., 2006). Since attentional control relies on brain areas shown to have deficits in ADHD, identifying differences between neurodivergent and neurotypical people in cognitive control biases would contribute to a better understanding of attentional control deployment.

Evidence from fMRI measures suggests that individuals with ADHD compensated for weaker task-set maintenance with later-stage attentional processes in Stroop incongruent trials where conflict was expected, such as those often observed in reactive control (Burgess et al., 2010). Such findings have been interpreted via the DMC as a shift towards greater reliance on reactive, stimulus-driven control processes, shifting away from proactive engagement (Braver, 2012). Sidlauskaite and colleagues (2019) examined ADHD adult symptomatology and cognitive control according to the DMC principles, using a cued-switching task in which cue informativeness was manipulated and measured via EEG. Results in fact suggested proactive control difficulties in adults with ADHD, evidenced by less use of cued advance information and abnormal preparatory processes for upcoming tasks. Despite that, no evidence for altered reactive control was found.

Engelhardt and colleagues (2008) examined working memory function using a classic Stroop-task paradigm and reported that ADHD participants made more errors on incongruent Stroop trials than neurotypicals. This would indicate increased reactive control, under the assumption that mistakes on incongruent trials would be less likely if proactive control were strongly applied (Bugg, 2012). However, no significant interaction effect was observed

between group status (ADHD vs. non-ADHD) and the Stroop condition (congruent vs. incongruent trials).

The present study

In light of the aforementioned results, conclusions remain ambiguous regarding differences in control engagement strategies between individuals with ADHD and neurotypical people. A large proportion of results come from neuroimaging studies or Stroop congruency effects, rather than from behavioral indices examining reaction time and accuracy measures.

This paper aims to address this gap in the literature by addressing the following research question: how do individuals with low versus high ADHD scores differ in their deployment of proactive and reactive control under dual-task conditions? In addition to examining proactive and reactive engagement, the study aims to replicate the findings of Nieuwenstein et al. (2025) of an adaptive attentional shielding mechanism in our sample.

We hypothesized that individuals with low ASRS scores would show modulation of proactive and retroactive interference by T2 expectancy. Proactive interference (slowed T2 RT in the 100% vs. 50% T2 condition across SOAs) is expected, whereas retroactive interference (reduced T1 Accuracy in the 50% than the 100% T2 condition across SOAs) is expected (H1). This will indicate modulation of cognitive control by T2 expectancy; when T2 probability is high, they will engage proactive control by effectively shielding T1, whereas when T2 probability is low, they will shift toward stimulus-driven reactive reliance by not shielding T1.

Conversely, we hypothesized that people with high ASRS scores will show reduced modulation of proactive and retroactive interference by T2 expectancy (H2). They will have smaller differences in T1 Accuracy and T2 reaction times between the two T2 probability conditions. This would indicate reduced modulation of cognitive control by T2 expectancy,

with less flexible shifting between the two control modes: a general tendency toward reactive responding, with a diminished ability to employ proactive control.

These hypotheses will be tested by examining how participants with varying levels of ADHD severity, as measured by the Adult ADHD Self-Report Scale (ASRS-v1.1) (Kessler et al., 2005), differ in cognitive control strategies during a first-target consolidation task (T1) and a subsequent reaction time task (T2).

Method

Participants

The participants for this study were recruited using a convenience sample through the SONA system of the University of Groningen, social media, and family/friends. After removing some cases, 69 participants' data were used. A target sample size of 100 participants was desired based on an a priori power analysis, to replicate the effects reported by Nieuwenstein et al. (2025). The inclusion criterion was a minimum age of eighteen years. Of the participants, 17.4% were male ($n = 12$), 79.7% were female ($n = 55$), and 2.9% identified as neither male nor female ($n = 2$). 33 participants completed trials with a 100% chance of T2 first, followed by trials with a 50% chance of T2, while 36 participants completed trials with a 50% of T2 first, followed by trials with a 100% chance.

Materials

The study was programmed and administered using OpenSesame (Mathôt et al., 2012). The experiment was conducted online using JATOS, a server-based platform compatible with OpenSesame's online export module (OSWeb), allowing the study to be completed remotely. The display resolution was set to 1980 x 1020 pixels, with a refresh rate of 60 Hz. Participants accessed the experiment in their web browser on a personal computer

or laptop and responded using their keyboard. They were instructed to complete the experiment in a quiet environment and in one uninterrupted session.

Stimuli

The experimental stimuli consisted of two target stimuli, T1 and T2. Target 1 (T1) consisted of a string of four uppercase letters, presented for 100 milliseconds. Vowels and the letters “Q”, “W”, and “M” were excluded, since they are more recognizable due to their shape than other consonants, leaving a set of 18 possible letters. The letters were randomly sampled without replacement from the alphabet. A mask of four characters, made up of three overlapping symbols (@, #, []), was used to obscure T1. The mask was also displayed for 100 milliseconds and was visually similar to target 1 in size and font, though the symbols were not within the target set. Target 2 (T2) consisted of a single digit, selected randomly from 2 to 9, and presented for 100 milliseconds. All stimuli were presented centrally in black 20-point Droid Sans Mono font on a white background.

ADHD Questionnaire

Attention-Deficit/Hyperactivity Disorder (ADHD) symptoms were measured with the Adult ADHD Self-Report Scale (ASRS v1.1) (Kessler et al., 2005). The ASRS consists of 18 items assessing inattention and hyperactivity/impulsivity symptoms according to DSM-5 criteria. Responses are made on a 5-point Likert scale (0 = never to 4 = very often). The scale demonstrates good psychometric properties, with internal consistency ranging from $\alpha = .70-.88$ and test-retest reliability of $r = .68-.80$. The total ASRS score was calculated by summing all 18 items' scores.

Design

Participants underwent a within-subjects experiment where each participant experienced every level in the study. The order of the two T2-probability conditions (50% T2 first, followed by 100% T2 or 100% T2 first, followed by 50% T2) was manipulated between groups. The SOA had two levels (50ms and 800ms), both used as a within-subject variable, where SOA was randomised in all trial blocks.

All participants completed 144 trials of which the first 16 trials served as practice trials in the 50% T2 presence condition and 72 trials of which the 8 served as practice trials in the 100% T2- Presence condition. The trials with 50% T2 presence contained twice the number of trials of the 100% T2 presence condition to equate the number of T2-present trials across conditions. As T2 appeared on only half of the trials in the 50% T2 condition, increasing the total number of trials ensured that participants completed an equal number of T2-present trials in both the 50% and 100% T2 probability conditions. This resulted in 64 trials where T2 was present, 64 trials where T2 was not present in the 50% T2 condition, and 64 trials in the 100% T2 condition.

Procedure

The participants were asked to provide their informed consent in using personal data before taking part in the study, such as their age and gender. They were then asked to complete the Adult Self-Report Scale (ASRS v1.1), reporting their symptomatology in their everyday life. The participants were asked to take the experiment in a quiet environment where distractions would be kept to a minimum, so as to ensure accurate responses and reaction times. The experiment was expected to take no more than 20 minutes to complete, while the entire study itself should not take more than 30 minutes, including completing the questionnaire.

After completing the ASRS, participants received instructions for the experiment. The participants were told to respond as fast as possible on the T2 reaction time task, indicating

whether the number presented on screen was odd or even, using the left and right arrow keys respectively. They were then asked to respond as accurately as possible on the T1 response task, by typing in the letters they saw on the screen on their keyboard, for which there was no time limit. Each trial in the experiment began with the presentation of a fixation dot, and a progress bar indicating how many trials had been completed at that point. Participants were instructed to fixate on the fixation dot and to then press the spacebar with their left hand to initiate the trial sequence. Once a key is pressed, the participant is presented with a string of four letters, after which they see a masking sequence of a combination of multiple symbols, followed by the number. T2 was presented either at an SOA of 50ms and 800ms after T1. Participants then received written feedback after each response, prompting them to be faster or more accurate with their T2 responses, in case their performance was too slow or incorrect. A progress bar was shown at the top of the screen, indicating how far along the respondent was in the sequence of trials. The participant had to press a random key on the keyboard to start the next trial. Once the respondents finished the experiment, a debriefing was included to ensure they were fully informed about the intentions of the study. They were then able to exit the experiment once the data was uploaded, using the “esc” keyboard button.

Data exclusions, Analyses, and Indices

In this study, we only included participants who responded correctly in the T2 reaction-time task on more than 55% of the trials, and who had more than 1 letter correct on average for T1 accuracy on trials in which T2 was present. Out of a total of 88 participants who initially completed the study, 19 participants had to be removed based on these criteria. In the end, we were left with 69 participants that were included in the final analysis.

Target SOAs of 50 ms and 800 ms resulted in corresponding ISIs of 45 ms and 795 ms due to the screen refresh rate. In the results, these conditions are referred to as 50 ms and 800 ms ISI, referring to the approximate values.

In measuring proactive (slowed T2 RT) and retroactive (reduced T1 accuracy) interference according to H1, the mean values for T1 accuracy and T2 reaction time were used for every participant across all possible combinations of ISI (50 ms vs. 800 ms) and T2 probability (50%, 100%). We conducted a repeated measures ANOVA analysis, for the two ISI levels and two T2 probability conditions, alongside paired-samples *t* tests to characterize any observed interaction effects. To test that people with ADHD traits will show less modulation of retroactive and proactive interference by T2 probability according to H2, we performed two correlational analyses examining the association between the total ASRS score and the modulation of retroactive and proactive interference by T2 probability. Retroactive was calculated by subtracting T1 Accuracy at the short ISI from T1 accuracy at the long ISI for each T2 probability condition. Proactive was computed by subtracting T2 RT at the long ISI from T2 RT at the short ISI. Using these indices, the modulation by T2 probability for each type of interference was computed, specifically, how much T1 suffered at 100% T2 probability vs 50% T2 probability (modulation of retroactive interference) and how much T2 slowed down at 50% T2 probability vs 100% T2 probability (modulation of proactive interference).

Results

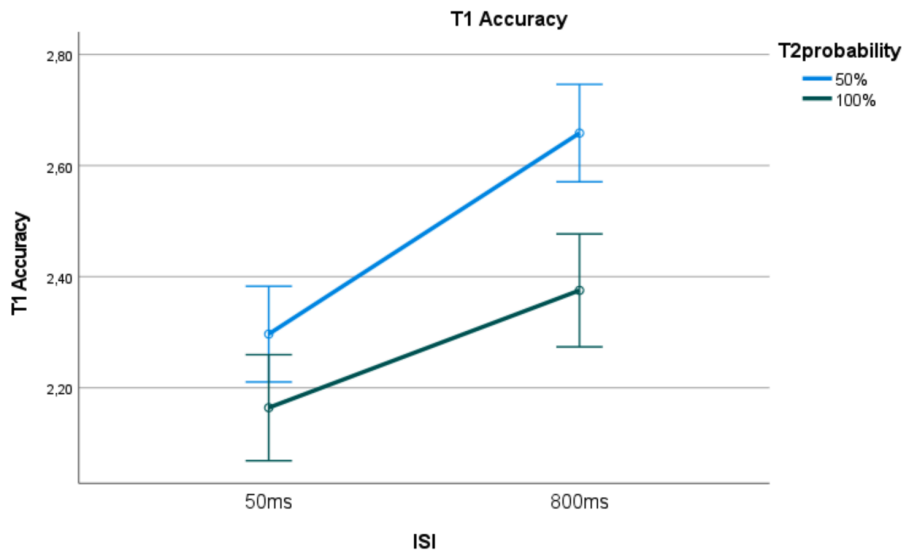
T1 Accuracy. In Figure 1, we can see that T1 accuracy suffered more in the 50ms ISI condition vs. the 800ms condition, when T2 was present in both the 100% condition and the 50% condition. However, there was a sharper drop when T2 was present only 50% of the time. Consistent with these observations, the results of a 2 (T2 probability: 50 vs. 100%) by 2 (ISI: 50, 800) repeated measures ANOVA analysis showed a significant interaction effect between T2 probability and SOA, $F(1, 68) = 6.76, p = .01, \eta^2 = .09$. The analysis also showed a significant main effect of ISI, $F(1, 68) = 47.92, p < .001, \eta^2 = .41$, and a significant main effect of T2 probability, $F(1, 68) = 11.68, p = .001, \eta^2 = .15$. A

paired-samples *t*-test was conducted to compare the mean difference in T1 accuracy between short (50ms) vs. long (800ms) ISI under 50% T2 and 100% T2 expectancy separately. The mean difference in T1 accuracy between short and long ISIs was larger in the 50% T2 condition ($M = -.36, SD = .39$), $t(68) = -7.53, p < .001$, 95% CI [-0.46, -0.27] than the 100% T2 expectancy condition. ($M = -.21, SD = .44$), $t(68) = -3.99, p < .001$, 95% CI [-0.32, -0.11]. This output showcases reduced T1 Accuracy from 800 to 50 ISI in the 50% compared to the 100% T2 probability, consistent with the retroactive interference account of H1.

T2 RT. In Figure 2, we can see that T2 reaction time decreased markedly across ISIs in the 100% T2 condition, whereas it decreased less across ISIs in the 50% condition. Consistent with these observations, the results of the 2 (T2 probability: 50 vs. 100%) by 2 (ISI: 50, 800) repeated measures ANOVA demonstrated a significant interaction effect between ISI and T2 probability, $F(1, 68) = 59.53, p < .001, \eta^2 = .47$. There was also a significant main effect of T2 probability, $F(1, 68) = 5.85, p = .018, \eta^2 = .079$, as well as a statistically significant main effect of ISI, $F(1, 68) = 189.54, p < .001, \eta^2 = .736$. A paired-samples *t*-test was conducted to compare T2 RT between the long and short ISI conditions at 50% and 100% T2 probability separately. T2 RT mean differences were significantly smaller between ISIs in 50% T2 probability ($M = 61.86, SD = 84.28$), $t(68) = 6.098, p < .001$, 95% CI [41.62, 82.11] than in 100% T2 probability ($M = 192.50, SD = 120.63$), $t(68) = 13.25, p < .001, CI [163.4, 221.4]$. These results indicate shorter T2 RT going from 50 to 800 ISI, at 50% vs. 100% T2 probability, consistent with the proactive interference account of H1.

Figure 1.

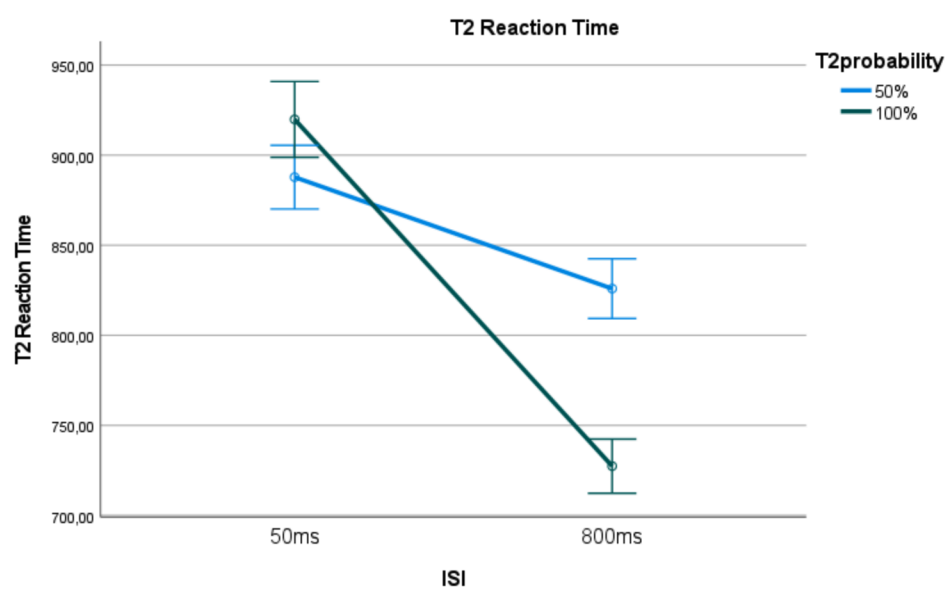
Line graph showing the effect of ISI and T2 presence probability on T1 accuracy.



Note: Error bars show the 95% confidence interval.

Figure 2.

Line graph showing the effect of ISI and T2 presence probability on T2 RT.



Note: Error bars show the 95% confidence interval.

ADHD and Retroactive interference modulation by T2 probability. A Pearson correlation was conducted to examine the relationship between ADHD symptom severity (ASRS total score) and the modulation of retroactive interference by T2 probability. The correlational analysis yielded a weak negative correlation, which did not reach statistical significance $r(67) = -.17, p = .17$. As seen in Figure 3, this yield is supported in how the data are scattered in a flat fashion, converging towards 0, suggesting no association between the ASRS scores and T2 probability modulating retroactive interference.

ADHD and Proactive interference modulation by T2 probability. A Pearson correlation was conducted in examining the relationship between ADHD symptom severity (ASRS total score), and the modulation of proactive interference at T2 probability. The correlational analysis yielded a weak positive correlation, which did not reach statistical significance either $r(67) = .20, p = .10$. As seen in Figure 4, the data points are scattered in a flat fashion, mostly converging towards 0. This visual representation also indicates no clear association between the scores in the ASRS and T2 probability modulating proactive interference.

These results indicate that ultimately the total ASRS score, indicative of ADHD symptomatology in our sample, is not associated with reduced modulation of retroactive interference or proactive interference by T2 probability. This in line, ultimately rejects H2.

Figure 3.

Scatterplot showing the relationship between the ASRS and the modulation of retroactive interference by T2 probability.

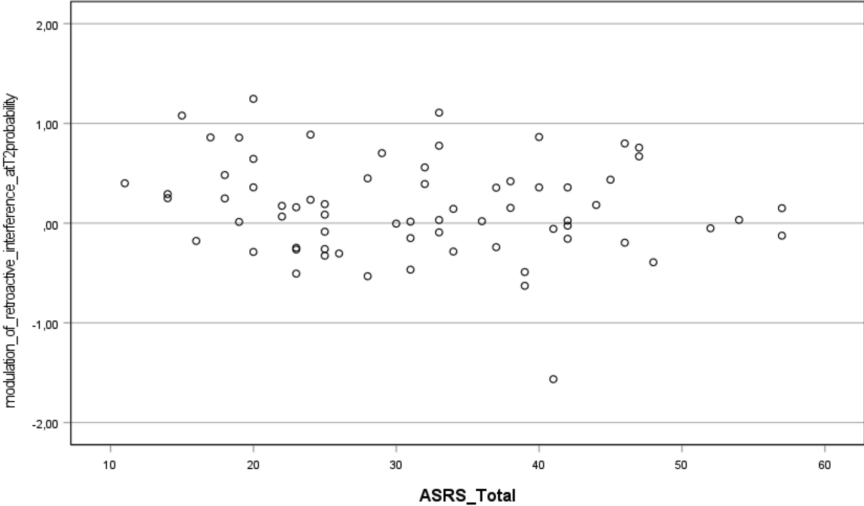
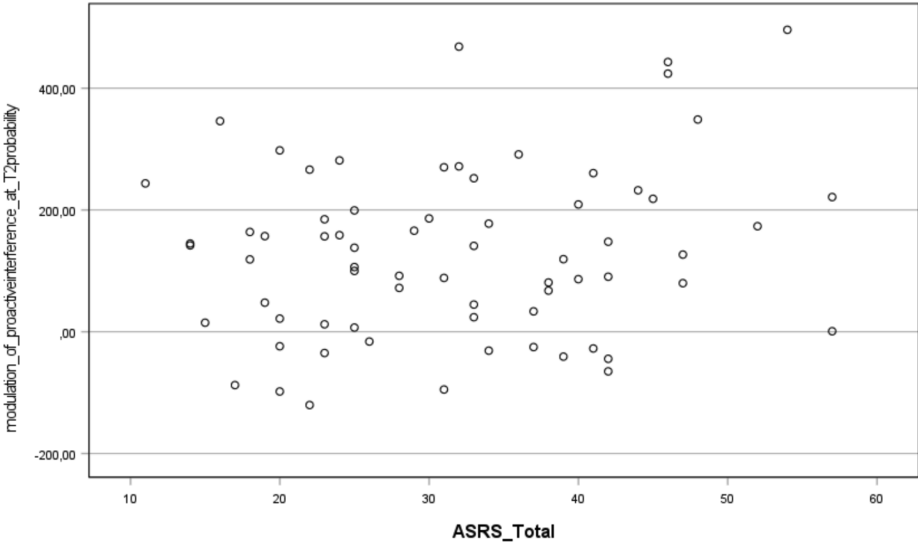


Figure 4.

Scatterplot showing the relationship between the ASRS score and the modulation of proactive interference by T2 probability.



Discussion

The aim of this paper was to examine how participants with high ADHD likelihood, as measured by the ASRS-v1.1, differ in how they deploy proactive vs. reactive control in dual-task conditions. T2 interference expectancy, and stimulus-onset asynchrony (SOA) were manipulated as within-subject variables, while performance on a primary consolidation task (T1) and reaction time on a secondary numeric task (T2) were measured. ADHD symptomatology was manipulated as a continuous variable by summing participants' test scores on the ASRS-v1.1.

Participants with low scores on the ASRS were expected to show increased proactive interference when T2 is always present across SOAs, and increased retroactive interference when T2 is present only half of the time across SOAs (H1), demonstrating proactive control adjustment when T2 appeared certainly, while switching towards reactive strategies when T2 did not always appear. Conversely, participants with high scores on the ASRS were expected to show decreased adjustment of proactive and retroactive interference by T2 probability, reflected in similar performance in the T1 and T2 tasks, whether T2 is expected or not (H2). This would illustrate decreased modulation of cognitive control by T2, suggesting diminished context-dependent proactive control engagement, and greater reliance on reactive control mechanisms.

As the results illustrate, the findings of Nieuwenstein et al. (2025) were replicated in our study. There was a significant modulation of T1 accuracy and T2 reaction time under conditions of different T2 expectancy and SOAs. A significant interaction effect was observed for T2 probability x SOA for both T1 and T2. Participants' T2 response times were indeed slower in the 100% T2 condition at short SOAs vs. long SOAs than in the 50% T2 condition, where there was not a lot of variation between SOAs, signifying proactive interference. (See Figure 2). Additionally, T1 recall was progressively less accurate in the 50% T2 condition,

going from long to short SOAs, than the 100% T2 condition, where there was not as much variation between SOAs, indicating retroactive interference. (See figure 1). A significant main effect of T2 probability on both T1 accuracy and T2 reaction times signifies that the expectancy of a secondary task does indeed influence which target gets shielded against interference (Nieuwenstein et al., 2025).

An interesting observation, which was not evident in Nieuwenstein et al. (2025) is the overall worse performance of T1 in the 100% than the 50% T2 probability (See Figure 1). Importantly, this is different from the retroactive interference pattern, showing worse progressive accuracy of T1 across SOAs when T2 was not always present (50% probability) than in the condition where it is always present (100% probability). A plausible explanation for this may be feedback effects. In our study, whenever participants responded incorrectly or had very long responses to T2, they received a message asking them to be faster, or more accurate. Since the participants were only corrected on their performance on T2, and not T1, they may have prioritized responding to the number over typing in as many letters as possible.

According to the intra-individual assumption in the DMC, comparable to proactively filtering out any irrelevant information from the meaning of the word in the high proportion of incongruent Stroop trials (Bugg, 2012), T1 Accuracy was shielded when T2 was expected on every trial. This argues in favour of attentional proactive control, preparing to prevent interference before it occurs, when it is strongly expected (Braver, 2012), implying that the shielding of T1 in high anticipated interference might be an instance of expectancy-driven, proactive control manifestation, comparable to the Stroop interference observed under high incongruency proportions (Bugg, 2012). Conversely, when T2 was not expected on every trial, T1 accuracy was not shielded, reflecting a shift toward stimulus-driven reactive responding, as evidenced by how T2 was processed more rapidly in the 50% T2 condition.

Our second hypothesis, assuming that ADHD symptomatology will be associated with

reduced modulation of cognitive control by T2 expectancy, was rejected. The correlational analysis provided no evidence for reduced modulation of proactive or retroactive interference by T2 probability. These observations suggest that individuals with test scores indicative of ADHD do not differ from those with low scores in how they adjust their T1 and T2 responses based on T2 expectancy. There is therefore no evidence for people with pronounced ADHD traits showing overall diminished proactive adjustment or greater reliance on reactive compensatory strategies.

Brain imaging studies showed that people with ADHD may partially offset reduced sustained goal maintenance by engaging control processes that operate later, particularly when conflict is salient, which aligns with reactive control mechanisms when the conflict actually appears (Burgess et al., 2010). Within the DMC, this pattern has been taken as evidence for preference for stimulus-triggered control over anticipatory, proactive strategies (Braver, 2012). Instead, our study found no evidence of default reactive reliance in participants scoring high on ADHD. Despite that, our behavioural measures may reflect our paradigm's inability to detect more subtle reactive control adjustments in individuals with high ADHD traits, rather than a true absence of effect.

Overall, our dual-task paradigm operationalizes proactive and reactive control differently than paradigms using neuroimaging measures or cue-informativeness tasks. Literature relying on neuroimaging data may reflect more sensitive differences across ADHD participants and control groups (Burgess et al., 2010; Sidlauskaite et al., 2019). Thus, any evidence for weak proactive engagement in ADHD under these paradigms renders the absence of a group difference in our study not entirely surprising. We did not measure cognitive control modes directly, such as with EEG, but rather measured behavioural markers of cognitive control modulation from expectancy-driven interference patterns, similar to studies measuring interference in the Stroop task (Braver et al., 2012; Bugg, 2012).

Considerable limitations in generalizing these findings concern the unequal gender distribution in our study (female: $n = 55$; male: $n = 12$). Any conclusions should therefore be approached cautiously, since our sample is not representative of the gender distribution in the actual population. This definitely limits the generalizability of our results, and any future studies examining how ADHD symptoms interact with cognitive control adjustment strategies in dual-task conditions should aim for a better gender distribution.

Moreover, our small sample size ($N = 69$) may be underpowered, which increases the likelihood of Type II errors. The null finding for H2 may instead reflect insufficient power to detect subtle associations between ADHD symptomatology and a reduced adjustment of cognitive control, rather than evidence that such associations are truly absent. In addition, participants were collected via the SONA software, an online platform primarily used by 1st-year psychology students. It is likely that university-level psychology students with indicative ADHD scores may not functionally represent the typical ADHD population in their cognitive abilities (Weyandt & DuPaul, 2006), which may explain our study finding no evidence for altered cognitive control processes in people with pronounced ADHD symptoms.

Lastly, although the ASRS demonstrates good reliability and validity in assessing symptoms, it is crucial to note that the score is only indicative and can not be viewed as an official ADHD diagnosis. As a result, although participants may have high scores, they may not actually have ADHD. Any comparison between our results and those of other studies should be approached cautiously, since other papers examining control biases in ADHD included people with clinical diagnoses (Burgess et al., 2010; Englehardt et al., 2008; Sidlauskaite et al., 2019). Here, it is important to discuss the possibility of people with an actual ADHD diagnosis being on medication while doing our study, since an unmedicated ADHD group was responsible for failure in consistently engaging proactive control in other research (Sidlauskaite et al., 2019). Thus, our results suggesting preserved modulation of

cognitive control by interference expectancy in people indicative of ADHD may be partly attributed to performance resembling that of neurotypical functioning.

The strengths of the study lie in the application of a 2x2 ANOVA, which revealed significant main and interaction effects, allowing for a thorough examination of the combined influence of SOA and T2 probability on the T1 Accuracy and T2 Reaction Time tasks. Methodological strengths of the study lie in participants experiencing every level of SOA (50 vs. 800ms) and T2 probability (50% vs. 100%), reducing inter-individual variability, which is especially valuable in our modest sample size of 69 participants. In addition, the practice trials ensured that participants were familiar with the demands of the task and the overall procedure, reducing noise in the main trials.

A key future direction would be to identify robust behavioral markers that provide reliable and independent indices of proactive and reactive control, since so far, this is not the case. In the present study, proactive and reactive control were inferred through performance on a T2 reaction time and T1 consolidation task, as a function of T2 probability and SOA. However, as with other paradigms, these measures are unlikely to be free from bias, as performance reflects multiple cognitive processes that intersect. Future research could employ paradigms such as the AX-CPT, or neuroimaging data shown to detect more subtle attentional control shifts to further investigate cognitive control strategies in ADHD.

In conclusion, the aim of the study was to identify whether proactive and reactive control is deployed differently between people indicative and not indicative of ADHD. The study partly served as a follow-up replication study of that of Nieuwenstein et al., (2025) adaptive attentional shielding hypothesis, suggesting effective cognitive control modulation in shifting between proactive and reactive modes based on secondary task expectancy. The paper also aimed to follow up with inconsistent findings in the literature regarding any proactive or reactive control biases in ADHD individuals. Despite the study's limitations, it provides clear

evidence for the adaptive attentional shielding hypothesis demonstrating effective switching between proactive and reactive control in people low in ADHD symptoms, while it does not provide evidence for participants with ADHD modulating their control less effectively based on interference expectancy. Nevertheless, our paper contributes to a growing body of literature investigating encoding and consolidation processes in working memory, which seem to operate adaptively according to anticipated interference, regardless of the possibility of ADHD traits in people.

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Appendix

Task Instructions

