Working memory capacity preferentially enhances implementation of proactive control

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Abstract

Previous research has linked working memory capacity (WMC) with enhanced proactive control. However, it remains unclear the extent to which this relationship reflects the influence of WMC on the tendency to engage proactive control, or rather, the ability to implement it. The current study sought to clarify this ambiguity by leveraging the Dual Mechanisms of Cognitive Control (DMCC) version of the AX-CPT task, in which the mode of cognitive control is experimentally manipulated across distinct testing sessions. To adjudicate between competing hypotheses, Bayesian mixed modeling was used to conduct sequential exploratory and confirmatory replication analyses involving two separate datasets. Posterior parameter estimates obtained from the exploratory analysis were entered as informed priors during the replication analysis to evaluate the influence of new data on previous estimates. Results yielded strong evidence demonstrating that the influence of WMC on proactive control is most robust under experimentally controlled conditions, during which utilization of proactive control is standardized across participants via explicit training and instruction. Critically, the observed pattern of findings suggests that the relationship between WMC and proactive control may be better characterized as individual differences in the ability to implement proactive control, rather than a more generalized tendency to engage it.

Keywords: *individual differences, dual mechanisms of control, cognition, cognitive control*

Introduction

Cognitive control is a fundamental ability that enables coordination and adaptive execution of goal-directed behavior (Egner, 2017). Given the central role of cognitive control in navigating wide domains of human functioning, it is unsurprising that control is considered to be a dynamical process sensitive to changing demands and circumstances related to the environmental context. One theory which aims to systematically parse this variability is the Dual Mechanisms of Cognitive Control (DMC) framework (Braver, 2012; Braver et al., 2021). The DMC proposes that cognitive control can be deployed in two distinct modes: proactive and reactive. Proactive control involves preparatory, sustained activation of task rules and goal representations; whereas reactive control involves transient, stimulus-driven task/goal activation.

Importantly, each mode of control is theorized to confer unique costs and advantages, such that successful utilization is likely to require flexible adoption of both control strategies. Conversely, disruption of this adaptability has been shown to underlie psychological impairment and behavioral dysfunction. For example, psychological and neurocognitive disorders such as schizophrenia and dementia have been associated with diminished use of proactive control (Barch & Ceaser, 2012; Braver et al., 2005), while developmental risk for anxiety has been linked to overreliance on reactive control (Troller-Renfree et al., 2019). Furthermore, individual differences in personality and cognitive ability are likewise thought to exert systemic influence on control dynamics (Braver, 2012; Braver et al., 2007). Within this domain, one of the most well studied individual difference constructs is working memory capacity (WMC), commonly defined as the ability to temporarily store, manipulate, and retrieve goal-relevant information (Unsworth & Engle, 2008).

Investigating WMC & Cognitive Control using the AX-CPT

In particular, a growing number of studies have leveraged the AX version of the continuous performance test (AX-CPT; Braver et al., 2001; Servan-Schreiber et al., 1996), to investigate the influence of WMC on cognitive control strategy use. Briefly, the AX-CPT is a widely used task of context processing, during which participants respond to trials of sequential cue-probe letter pairs. A target response is required only when an A cue is followed by an X (AX trials); conversely, a non-target response is required for all other cue-probe pairs including AY, BX, and BY trials such that B and Y represent any letters except A and X. Proactive control is indexed by preparatory response patterns influenced by the A cue (e.g., committing more correct non-target responses, following a non-A cue, such as on BX trials, but more incorrect target responses on AY trials); whereas reactive control is indexed by "just-in-time" response patterns that are influenced by the X-probe (e.g., committing more correct non-target responses on AY trials, but slower and potentially more error-prone responses on BX trials).

Using this paradigm, investigators examining the relationship between WMC and cognitive control have consistently reported that adult individuals with higher WMC exhibit greater use of proactive control relative to lower WMC participants (Belletier et al., 2019; Boudewyn et al., 2015; Redick, 2014; Redick & Engle, 2011; Richmond et al., 2015; Stawarczyk et al., 2014; Wiemers & Redick, 2018). There is also emerging evidence that this relationship extends to young children and may begin to develop as early as age 5 (Gonthier et al., 2019; Troller-Renfree et al., 2020; Wang et al., 2021). Importantly, these findings provide empirical support for the theoretical implications of WMC in the context of the DMC framework—namely, that WMC enables active maintenance of task and goal relevant information; and because

proactive control essentially relies on this capacity, it stands to reason that higher WMC should facilitate the use of proactive control.

Conceptual Ambiguities & Methodological Challenges

With that said, the studies to date have primarily relied on extreme-group comparison (Redick, 2014; Redick & Engle, 2011; Wiemers & Redick, 2018) or correlational (Belletier et al., 2019; Boudewyn et al., 2015; Gonthier et al., 2019; Richmond et al., 2015; Stawarczyk et al., 2014; Troller-Renfree et al., 2020; Wang et al., 2021) designs. Although it is instructive to compare AX-CPT performance across low vs. high WMC individuals, the salient limitation of these methods is that it precludes the ability to discriminate between the *tendency* to engage in proactive control and the *ability* to implement it. Without stringent experimental controls to standardize control strategy use and establish appropriate comparisons, the admittedly porous but potentially important boundary conditions between the tendency and ability to implement different modes of control is likely to remain analytically indistinguishable. Specifically, experimental manipulations that involve explicit instruction of proactive/reactive control are critical for interpreting observed individual differences in ability. This is because failure to specify and standardize cognitive control strategy use leaves open the unwanted possibility that participants may spontaneously vary their mode of control during task performance. In other words, under conditions for which cognitive control strategy is not explicitly instructed and/or constrained, it is possible that observed individual differences may reflect the tendency to spontaneously adopt a control mode, or to flexibly switch among them, rather than the ability to implement a singular mode of control when the task requires it.

In addition to resolving these ambiguities, a related but broader challenge involves circumventing the psychometric and analytic limitations commonly associated with using cognitive experimental behavioral paradigms when conducting individual differences research. Here, we briefly highlight four pervasive and well-documented problems. First, use of conventional summary scores derived from averaging across aggregated trial performance overlooks trial-level variability, and as a consequence, can result in poor reliability and underestimated effect sizes (Rouder & Haaf, 2019). Second, the equally common practice of using subtraction-based difference scores can increase measurement error, constrain between-subject variance, and attenuate reliability (Caruso, 2004; Cronbach & Furby, 1970; Draheim et al., 2019; Hedge et al., 2018). Third, classic experimental psychology statistical approaches such as ANOVA and ANCOVA often violate assumptions of independence, and do not model subject-level variability as a unique source of variance, potentially leading to overestimated effect sizes and increased risk of Type I error (Judd et al., 2012; Singmann & Kellen, 2019). Fourth, the practice of null hypothesis testing (NHST), which has been subject to increasing scrutiny (Cumming, 2014; Halsey et al., 2015; Ioannidis, 2005; Simmons et al., 2011), only weighs evidence against the plausibility of the null hypothesis but not evidence in favor the alternative hypothesis, precluding inferential evaluation of both the existence and magnitude of the hypothesized effect (Wagenmakers, 2007; Wagenmakers et al., 2018).

Study Rationale

With these challenges in mind, the primary aims of the present study are twofold: (1) to clarify the conceptual ambiguities regarding the nature of the relationship between WMC and proactive control; and (2) to remediate the common methodological issues pertaining to

measurement and analysis described above. As alluded to above, the key to achieving the first aim lies in deriving methods to place cognitive control mode under direct experimental manipulation. Toward this end, one of the most comprehensive efforts to date involves the development of the DMCC task battery (see Braver et al., 2021; Tang, Bugg, Snijder, Conway, & Braver, 2021). In particular, the DMCC version of the AX-CPT was designed to elicit selective utilization of proactive, reactive, and baseline control across three separate namesake testing sessions.

Briefly, the proactive session leverages prior work by explicitly instructing participants to utilize contextual cue information in preparing their responses (Gonthier, Macnamara, et al., 2016), effectively training participants to implement a proactive strategy during task performance. On the other hand, consistent with other approaches to reactive control, the reactive session uses an implicit, item-specific cueing manipulation (Bugg & Crump, 2012; Gonthier, Braver, et al., 2016). Specifically, in this session, the probe is presented in a distinct location with unique borders on AY, BX, and no-go trials, serving as an accentuated "just-in-time" stimulus signal to implement reactive control during high demand conflict trials. Finally, the baseline session excludes the prior manipulations but retains no-go trials (presented across all sessions), during which the probe is replaced by a numerical digit. Importantly, implementation of no-go trials decreases the predictive utility of cue information, and is designed to reduce the proactive bias observed in healthy young adults (Gonthier, Macnamara, et al., 2016)—rendering the baseline session an ideal "active control" condition from which intended shifts of cognitive control can be evaluated via cross-session comparison (e.g., contrasting proactive vs. baseline performance).

Importantly, the multi-session, within-subject design of the DMCC AX-CPT affords a powerful experimental-correlational approach to clarify the influence of WMC on cognitive control (Cronbach, 1957). By standardizing cognitive control strategy use via experimental

manipulation, variability associated with the natural tendency adopt a preferred control mode is minimized, enabling any observed associations between WMC and performance within a given session to be interpreted as between-subject differences in the ability or effectiveness of using a given control strategy instead. Moreover, the addition of multiple sessions enables comparative analyses to ascertain the relative *specificity* of the purported influence of WMC on control. For example, data can be aggregated across sessions to explicitly test whether a hypothesized relationship between higher WMC and enhanced proactive control is observed preferentially within the proactive session, over and above any relationships present in the baseline and reactive sessions.

Together, these design features provide the inferential ability to parse the extent to which observed correlations between WMC and proactive control reflect the influence of WMC on the ability to implement proactive control or the tendency to engage it. For example, finding a specific association between higher WMC and enhanced proactive control preferentially in the proactive session (during which all participants are trained and instructed to engage in proactive control) would link higher WMC with superior proactive control ability. On the other hand, observing an association between higher WMC and enhanced proactive control during the baseline but *not* the proactive session would signal that WMC influences the tendency to use proactive control during unconstrained conditions where strategy use is not actively manipulated. Lastly, a third possibility involves finding that higher WMC is associated with enhanced proactive control in both the proactive and baseline sessions, suggesting that WMC may influence both the tendency and ability to use proactive control.

To adjudicate between these competing possibilities, we pre-registered a set of exploratory analyses aimed at broadly replicating past findings linking WMC with enhanced proactive control.

We hypothesized that higher WMC scores would be associated with enhanced proactive control metrics in the baseline and or proactive sessions (see https://osf.io/n9mqw). Specifically, analyses focused on three indices of proactive control: (1) A-cue bias, (2) BX interference, and (3) the d-prime context effect. Briefly, A-cue bias assesses the propensity to commit target responses across "A" trials, including both AX (hits) and AY (false alarms) trials—rendering it a collective measure of response bias based on contextual cue utilization and response preparation. Because WMC is needed to store and actively maintain contextual cue information, while proactive control enables preparatory cue utilization (i.e., activation of the expected target response), we predicted that higher WMC would be associated with stronger A-cue bias (i.e., more target responding across "A" trials). The BX interference effect contrasts errors and RT across BX and BY trials, enabling measurement of the interference associated with the presentation of an "X" probe following a *nontarget* ("B") cue. Here, enhanced B-cue utilization afforded from proactive control should elicit greater preparation of correct nontarget responses and attenuate "X" probe interference. Higher WMC is therefore expected to be associated with reduced BX error (i.e., fewer errors) and RT interference (i.e., faster RTs) on high conflict BX trials, referenced to the low conflict BY trials. Lastly, the d-prime context effect compares target responding across AX and BX trials, measuring the degree to which the response to "X" probes discriminates target ("A") and nontarget ("B") cues. Similarly, greater cue utilization should elicit *more* target responses on AX trials (i.e., correct hits) relative to BX trials (i.e., false alarms)—a metric referred to as d-prime sensitivity. Extending the rationale developed above, higher WMC is expected to be associated with enhanced d-prime sensitivity. Taken together, these three indices provide a strong signature of proactive control from which to observe sensitivity to individual differences in WMC, and how these effects are impacted by the session manipulation of cognitive control mode. Specifically, the presence of session

specific patterns that emerged to support these predictions (e.g., WMC positively correlated with A-cue bias in only the proactive session but not the baseline session) were identified in the exploratory dataset. These were then treated fixed predictions to be retested in a pre-registered follow-up replication analysis, using a separately collected holdout dataset (https://osf.io/x96wn).

Regarding the second aim, we endeavored to address the aforementioned methodological and analytic issues by adopting a trial-level Bayesian mixed modeling approach. First, we used complete trial-level data across all task conditions to appropriately capture trial-level variability and obviate use of difference scores. Second, we leveraged mixed modeling with random slopes and intercepts to circumvent non-independence and account for subject-level variability in task performance. Third, we employed a Bayesian regression approach to quantify and assess evidence in favor of the predicted effects against the null. Moreover, as mentioned above, we utilized this approach to showcase how results from a pre-registered exploratory analysis can be subject to sequential replication with Bayesian updating procedures. Although Bayesian statistical approaches are far from new, and have become increasingly popular within various areas of psychological science (van de Schoot et al., 2017), to our knowledge, the use of Bayesian approaches to investigate cognitive control remains relatively underutilized relative to traditional frequentist approaches. Consequently, a final motivation for the current study was to provide a practical demonstration of the advantages and implementational features of this analytic and inferential approach, as applied to the research prerogatives of the DMC framework and of cognitive control studies more generally. Below, we separately detail the specific methods and results of the exploratory and replication analyses before synthesizing the collective implications of the findings in the general discussion section at the end.

Exploratory Analysis

Method

Participants

One-hundred thirty-seven participants were recruited via the Amazon Mechanical Turk (MTurk) on-line platform. Data collection was completed in March 2018 and included two rounds of testing, during which participants completed a retest assessment several weeks after initial testing. Participants were not restricted with regard to age (22-64, M = 37.05, SD = 10.88; 85 females, 52 males). Excluded participants included those who did not complete the sessions on time, experienced prohibitive technical issues, or failed to comply with task instructions. Lastly, participants with overall accuracy below 50% and or 3 SDs below the sample mean were excluded. The TurkPrime interface was used for all aspects of participant recruitment and management (e.g., advertisement, communication, and payment). Prospective participants were given a link to review and sign the consent form. Upon consenting, the web-links for the tasks were made available over MTurk. Participants were compensated a total of $122 for full completion (includes test and retest). The study protocol was approved by the institutional review board of Washington University, St. Louis.

Design and Procedure

The study protocol consisted of thirty testing sessions lasting 20-40 minutes (15 sessions for test, and 15 sessions for retest), including the baseline, proactive, and reactive sessions of the AX-CPT among other components of the DMCC battery. For the purposes of the current study (i.e., to maintain continuity with the replication analysis described below), analyses focused exclusively on the test data; the retest data were excluded from all analyses and not examined. The

DMCC task battery was originally developed for the neuroimaging environment (Braver et al., 2021); the online behavioral protocol and task variants (Tang et al., 2021), which included additional self-report/demographic questionnaires, are fully described elsewhere (see Etzel et al., 2021). Participants were asked to complete the sessions at a rate of 5 per week in fixed sequential order (with baseline conditions completed first, followed by reactive, then proactive), taking about 3 weeks to complete the full protocol. Each 5-session set was posted at the beginning of the week and two reminder emails were sent to remind subjects to complete the set by the end of the week. Completed sessions were checked for accuracy and compliance (see Tang et al., 2021). Subjects who did not complete the weekly set or failed to comply with instructions were discontinued from future sessions and received a prorated payment for sessions completed.

Tasks

AX-CPT

Participants were instructed to make either target ("/") or nontarget (".") button press responses to visually presented cue-probe pairs. Consistent with previous versions of the AX-CPT, a target response was required to the probe on AX trials, whereas a nontarget response was required to the probe all other trial types (AX, BX, BY), as well as to the cue on all trials. As mentioned above, this version of the task also included no-go trials, which required withholding response to the probe; no-go trials were indicated by a digit (1-9) rather than letter probe (Gonthier et al., 2016). There was a total of 216 trials, encompassing 72 AX trials, 72 BY trials, 18 AY trials, 18 BX trials and 36 no-go trials (18 following an A-cue, 18 following a B-cue). The task was performed in three 72 trial blocks with all trials presented in random order. Subjects were instructed to take a minimum 1-minute rest break between blocks to mitigate fatigue. Across all

trials, the cue was presented at the center of a white screen for 500 milliseconds (ms). After a fixed blank duration of 4000 ms, the target probe was presented for 500 ms which was preceded by a bounding box presented 250 ms earlier. Each trial concluded with a 1500 ms inter-trial interval during which a triangle arrangement of fixation crosses was presented at the center of the screen.

Baseline Session. The baseline session was identical to the description above. Participants performed a 12-trial practice block before beginning the actual session.

Proactive Session. Participants completed two phases of strategy training prior to beginning the session (Gonthier et al., 2016). In the first phase, an audio clip instructed participants how to prepare their button presses in response to the cue across 6 hypothetical trials (i.e., to prepare a target response following A cues and nontarget otherwise). In the second phase, participants completed 6 practice trials by typing out "left" or "right" to indicate the button they were preparing to press. Feedback was provided after incorrect responses, reminding participants of the cue letter and requesting them to try again. Finally, participants were prompted with the visual message "Use the strategy!" during the inter-trial interval periods across the actual testing session. All other task components were identical to the baseline session.

Reactive Session. The reactive session featured a new AX-CPT variant that was adopted to preferentially encourage and enhance utilization of reactive control (for further discussion of this version see Braver et al., 2021; Tang et al., 2021). High conflict trials (AY, BX, no-go) were preceded by a unique border color and further accentuated by placing the probe in a distinct spatial location. Specifically, on low-conflict AX and BY trials, the probe was presented on the upper half of the screen, whereas the probe was presented on the lower half of the screen during the high-conflict AY, BX, and no-go trials. Furthermore, a black border preceded the probe on AX and BY trials, whereas a red border preceded the probe on AY, BX, and no-go trials. Cues were presented

at the center of the screen. All other trial parameters were identical to the baseline and proactive sessions.

Working Memory Tasks

Operation Span Task (OSPAN). The OSPAN (Turner & Engle, 1989; Unsworth, Heitz, Schrock, & Engle, 2005) was used to assess working memory capacity (WMC). During each trial, participants were required to verify the accuracy of a mathematical equation before being presented with a random letter to remember. The number of math-letter sequences (i.e., set size) varied from three to seven per trial. At the end of the trial, participants selected the presented letters in the order that they had appeared. The task consisted of three trials of each set size for a total of 15 trials.

Symmetry Span Task (SYMSPAN). The SYMSPAN (Unsworth, Redick, Heitz, Broadway, & Engle, 2009) is another measure of WMC. Similar to the structure of the OSPAN, participants were required to judge whether a displayed shape is symmetrical along its vertical axis before being presented with a red square in a 4x4 grid of potential locations to remember. At the end of each trial, participants selected the location of the red squares in the order of presentation. The number of symmetry-location sequences (i.e., set size) ranged from two to five per trial. The task consisted of three trials of each set size for a total of 12 trials.

Statistical Analyses & Predictions

Bayesian linear regression models were fit using the brms package in the R software environment (Bürkner, 2017). WMC was quantified as a composite score and mean-centered by way of z-scoring the average of OSPAN and SYMSPAN scores (each span task was also z-scored

first). Categorical variables (trial type, session) were effect coded. As further detailed below, the data were filtered so that trial type was rendered as a binary variable contingent upon the metric of interest. Logistic regression on trial-level data was then used to model interference effects on response type (target vs. nontarget) and accuracy (correct vs. incorrect). Similarly, trial-level reaction times (RTs) were modeled using shifted lognormal functions (Haines et al., 2020), with mixed-effects linear regression used to estimate interference effects. Trial type, session (baseline, proactive, reactive) and WMC score were predictors in the model. Subject-level variability was modeled by entering the intercept and trial type as random effects nested within subject. Due to the exploratory nature of the analyses, we conservatively elected to use uniform priors on the fixed effects across all models. Random effects were weakly informative based on brms defaults.

Below, we describe how each control metric was modeled and tested. Models involving response type and accuracy was run with 4 Monte Carlo chains, each containing 2,000 sample iterations and 1000 warm-up iterations, with the warmup iterations discarded. The modeling of RT was likewise run with 4 chains, but each containing 4,000 sample iterations and 2000 warm-up iterations—sample iterations were doubled to ensure sufficient effective sample sizes (ESS) across model parameters. For every parameter estimate, we report the mean, standard deviation, and the 95% credible interval (CI; quantile-based equal tailed interval) of the posterior distribution, as well as the R-hat and ESS values. Where applicable, log-odds were exponentiated to odds and presented in the model summary tables. The pre-registration for this exploratory analysis is accessible at https://osf.io/n9mqw. Moreover, all data, materials, and analysis code are openly available at https://osf.io/xfe32/.

A-Cue Bias

A-cue bias was modeled as the log-likelihood of committing a target, relative to nontarget, probe response on AX/AY trials. To test the general prediction that higher WMC would be associated with stronger A-cue bias, we ran the following model on AX/AY trial data aggregated across all sessions: *probe response ~ trial type x WMC x session + (1 + trial type | subject)*. Specifically, we expected to observe that higher WMC would be associated with higher log-odds of committing target responses in either the baseline session, proactive session, or both (i.e., WMC x session interactions). Although finding a main effect of WMC was also a possibility (i.e., that higher WMC is associated with higher log-odds of target responding irrespective of session manipulations), we nonetheless expected that the effect of WMC on A-cue bias would be most salient in the baseline or proactive sessions based on prior work and theoretical grounds (Richmond et al., 2015). Failure to obtain evidence for either effect would serve as grounds for falsification that WMC is related to A-Cue bias.

BX Interference

BX interference on trial accuracy was modeled as the log-likelihood of committing a correct, relative to incorrect, response on BX/BY trials; whereas interference on RT was modeled using lognormal linear regression on BX/BY trial RTs. To test the prediction that higher WMC would be associated with reduced BX interference, we applied the same predictors specified above to estimate trial accuracy and RT. Here, we expected that higher WMC would be associated with higher log-odds of correct responses and faster RTs on BX trials relative to BY trials (i.e., reduced BX error and RT interference, respectively) in either the baseline session, proactive session, or

both. Note that in contrast to the A-cue bias, predictions the BX interference predictions are 3-way interactions that involve trial type (i.e., session x WMC x trial type). Again, it was also possible, but not predicted, that higher WMC would be associated with reduced BX error and RT interference, collapsing across session (i.e., WMC x trial type). The absence of these 3-way interaction effects would fail to support the hypothesized relationship between WMC and BX interference.

D-Prime Context Effect

Lastly, the d-prime context effect was modeled as the log-likelihood of committing a target, relative to nontarget, probe response on AX/BX trials. Similar to above, we leveraged the same predictors to test the hypothesis that higher WMC would be associated with enhanced d-prime sensitivity. Specifically, we expected that higher WMC would be associated with higher log-odds of target responding on AX relative to BX trials (i.e., more correct hits relative to fewer false alarms) in either the baseline session, proactive session, or both. Similar to the BX interference predictions (but distinct from A-cue bias predictions), the d-prime context predictions are 3-way interactions that involve trial type (i.e., session x WMC x trial type). Once again, we acknowledged the possibility, but did not predict, that higher WMC would be associated with enhanced d-prime sensitivity collapsing across all sessions (i.e., WMC x trial type). Failure to obtain evidence for these 3-way interaction effects would fail to support the hypothesis that WMC is related to d-prime sensitivity.

Results

Full model summaries involving all parameter estimates are provided in Tables 1-4. To maintain focus and tractability, we circumscribe descriptive reporting to only the predicted effects below.

A-Cue Bias

Consistent with predictions, higher WMC was not significantly associated with a general tendency to make a target response (i.e., non-significant main effect), but instead was uniquely associated with higher log-odds of target responding in the proactive session (b = 0.16, sd = 0.03, 95% CI = [0.09, 0.23]). Conversely, WMC was associated with relatively lower log-odds of target responding in the reactive (b = -0.08, sd = 0.04, 95% CI = [-0.15, -0.01]) and baseline session (b = -0.08, sd = 0.04, 95% CI = [-0.15, -0.01]).

BX Interference

In analyzing trial accuracy, higher WMC was associated with relatively higher log-odds of correct responses on BX trials relative to BY trials (i.e., reduced BX error interference, 3-way interaction) in the baseline session (b = 0.14, sd = 0.05, 95% CI = [0.06, 0.23]), but not in the proactive (b = -0.09, sd = 0.05, 95% CI = [-0.19, 0.00]) or reactive session (b = -0.05, sd = 0.04, 95% CI = [-0.13, 0.04]). In contrast for RT however, higher WMC was associated with faster RTs on BX relative to BY trials in the proactive session (i.e., reduced BX RT interference, 3-way interaction; b = -0.006, sd = 0.003, 95% CI = [-0.011, -0.001]) [1], but not the baseline (b = 0.001,

[1] Because RTs were fit to a lognormal distribution, effect estimates involved small values. Consequently, we report parameter estimates to the third decimal place.

sd = 0.003, 95% CI = [-0.004, 0.007]) or reactive session (*b* = 0.005, *sd* = 0.003, 95% CI = [0.000, 0.010]).

D-Prime Context Effect

Contrary to expectations, WMC was unrelated to target responding on d-prime sensitivity across all sessions (i.e., no significant WMC x trial or 3-way interactions; *b*s < |0.05|, all CIs contain 0]).

Summary

Briefly, the exploratory analyses broadly supported the hypothesized relationship between higher WMC and enhanced proactive control. In particular, higher WMC was associated with stronger A-cue bias and reduced BX RT interference in the proactive session. Interestingly and somewhat inconsistently, higher WMC was associated with reduced BX error interference in the *baseline* session, with no significant effect in proactive. Finally, WMC was unrelated to d-prime sensitivity. Although in need of further testing, this constellation of findings suggested that the relationship between WMC and enhanced proactive control may be relatively more specific to the proactive session as opposed to the baseline session. Moreover, there is limited evidence in favor of a "session general" effect of WMC on control metrics (i.e., that WMC is related to enhanced indices of proactive control irrespective of session).

With these possibilities in mind, we aimed to replicate the results prior to extrapolating the significance and implications of the pattern of findings. Toward this end, all observed effects were subject to retesting in a replication analysis, with the posterior parameter estimates obtained above entered as informed priors to fully utilize the advantages of Bayesian updating and hypothesis

testing. Critically, the specific predictions involving WMC and control metrics were narrowed to mirror the results obtained in the exploratory analyses. Below, we detail the model specifications and statistical analytic procedures associated with the replicatory testing of each prediction, including use of Bayes factors and probability of direction to quantify the strength of evidence for the expected effects.

Replication Analysis

Method

Participants

One-hundred thirty-three participants completed the MTurk task battery in October 2020. The TurkPrime interface was again used for all aspects of participant recruitment and management. All participants reviewed and signed the consent form prior to study enrollment. Similarly, participants were not restricted to age (18-77, M = 39.30, SD = 11.28; 73 females, 59 males, 1 prefer not to answer), and the exclusion criteria remained identical to that outlined in the previous section. Participants were paid $51 for full completion of the study (which did not include a retest component). The study protocol was approved by the institutional review board of Washington University, St. Louis.

Design and Procedure

The second wave of data collection did not include a retest phase and was therefore comprised of 15 sessions. Likewise, in this wave of data collection, a fixed session order was used, but this was slightly different from the 2018 wave in that although the baseline sessions were first,

the proactive sessions came before reactive. All other aspects of the tasks, materials, and procedures were otherwise identical to what was previously described regarding the first wave.

Statistical Analyses & Predictions

Again, all Bayesian linear regression models were fit using the brms package in the R software environment (Bürkner, 2017). Coding of variables and mixed effect modeling procedures were identical to what was specified in the exploratory analyses. The key difference is that posterior parameter estimates obtained from the exploratory dataset were modeled as informed gaussian priors (specified as the mean and SD of the distribution) to evaluate the influence of new data on previous estimates. Indeed, the central aim of the analysis was to replicate the key findings involving WMC and proactive control. Models involving response type and accuracy were again run with 4 Monte Carlo chains, each containing 2,000 sample iterations and 1000 warm-up iterations, whereas modeling of RT utilized 4,000 sample iterations and 2000 warm-up iterations. Similarly, for every parameter estimate, we again report the mean, standard deviation, and the 95% credible interval (CI) of the posterior distribution.

Novel to the replication analysis, however, is the use of Bayes factors (BFs)—specifically, the Savage-Dickey density ratio (SDR) and evidence ratio (ER)—and the probability of direction (PD) metric to evaluate the strength of replication. For each predicted effect outlined below, the SDR was computed to evaluate the extent to which the mean parameter estimates obtained from the exploratory dataset differed as a function of incorporating the replication sample. Briefly, the SDR is formalized as the posterior density divided by the prior density at the specified point value, which for our purposes will be the mean value of the *prior* distribution (i.e., mean parameter estimate obtained from the exploratory analysis). Moreover, we calculate the ER to evaluate the

overall amount of evidence favoring the presence of the predicted effect against the null. Contextualized within the scope of the research question, this involves testing the extent to which effects involving WMC and proactive control metrics are greater or less than 0, contingent upon the specific directionality of the original finding. For example, when attempting to replicate a hypothetical main effect of WMC on A-cue bias (i.e., higher WMC associated with stronger A-cue bias), the ER is the ratio of the posterior probability that WMC > 0 (i.e., alternative hypothesis) relative to the posterior probability of WMC < 0 (i.e., null hypothesis). Lastly, we present the PD, computed as the posterior probability that a parameter estimate is positive or negative, to evaluate the amount of evidence that the effect falls within the expected direction.

Together, the SDR, ER, and PD provide a quantification of point estimate replicability (i.e., the degree to which the parameter means changed after incorporating replication data), as well as the overall strength of evidence favoring the presence of the predicted effect (i.e., the relative proportion of the posterior distribution falling above or below 0 in the predicted direction). SDRs greater than one indicate that the weight of evidence is in favor of the prior mean, whereas SDRs less than one indicate that the prior and posterior mean may differ. ERs greater than one indicate evidence in favor of the tested hypothesis whereas values less than one indicate evidence against. Per Lee & Wagenmakers (2014), values between 1 – 3 (1 – 1/3) suggest anecdotal evidence, 3 – 10 (1/3 – 1/10) moderate evidence, 10 – 30 (1/10 – 1/30) strong evidence, greater than 30 (less than 1/30) very strong evidence. The PD has a direct correspondence to the frequentist p-value, such that a PD of 95%, 97.5%, and 99.5% is approximately equivalent to a two-sided p-value of 0.10, 0.05, and 0.01, respectively. To further aid interpretation of these metrics, we provide plots of the prior and posterior distributions as a visual supplement.

Lastly, we plotted predicted probability figures of each model, graphed as a function of WMC and session, and separated by trial type using the *interactions* package in R (Long, 2019). It should be noted here that the transformation of logits to predicted probabilities is nonlinear and that probability slope estimates are derived as an aggregated function of *all* model parameters (Ai & Norton, 2003; Osborne, 2019). Consequently, some of the hypothesized effects, which are based linearly in logits and involve higher order interactions, are not always visualizable in the probability domain. Nonetheless, we present these graphs to provide a more comprehensive portrayal of our models and to solicit engagement of other researchers whom might be interested in extending this work more formally into the probability domain (e.g., via Stata methods outlined in Mitchell & Chen, 2005). The pre-registration for this replication analysis is available at https://osf.io/x96wn, while all data and code used to conduct the analyses are openly available at https://osf.io/xfe32/.

Results

Full model summaries involving all parameter estimates are provided in Tables 5-8. We again circumscribe descriptive reporting to only the effects of interest. Visualization of the prior and posterior distribution for each predicted effect is presented on Figure 1. Predicted probability graphs for each model are presented sequentially on Figures 2-5.

A-Cue Bias

Confirming the original finding, higher WMC was uniquely associated with higher log-odds of target responding in the proactive session (b = 0.12, sd = 0.02, 95% CI = [0.08, 0.16], ER

= Inf[2], PD = 100%), but not in the baseline condition (*b* = -0.02, *sd* = 0.03, 95% CI = [-0.06, 0.04]). Interestingly, higher WMC was associated with relatively *lower* log-odds of target responding in the reactive session (*b* = -0.12, *sd* = 0.03, 95% CI = [-0.17, -0.06]). The SDR comparing the posterior and prior density at the prior mean (*b* = 0.16) for the predicted effect that WMC would be associated with stronger A-cue bias in the proactive session was 0.25, indicating that the prior mean estimate may have been overestimated in the original dataset. Nonetheless, given that the ER approached infinity and the PD was 100%, there was decisive evidence supporting the presence of the predicted effect (see Fig. 1A).

BX Interference

Replicating the initial finding, higher WMC was associated with higher log-odds of correct responses on BX trials relative to BY trials (i.e., reduced BX error interference) in the baseline session (*b* = 0.09, *sd* = 0.03, 95% CI = [0.03, 0.16], ER = 443.44, PD = 99.78%), but not in the proactive (*b* = -0.02, *sd* = 0.03, 95% CI = [-0.08, 0.04]) and reactive sessions (*b* = -0.05, *sd* = 0.03, 95% CI = [-0.11, 0.00]). The SDR comparing the posterior and prior density at the prior mean (*b* = .14) for the predicted effect that higher WMC would be associated with reduced BX interference in the baseline session was .64, suggesting that the prior mean may have been slightly overestimated. The ER of 443.44 and PD of 99.78% provided very strong evidence in favor of the predicted effect, indicating that the vast majority of the distribution fell above 0 (see Fig. 1B).

The RT interference effect was also replicated. Consistent with the finding from the exploratory analysis, higher WMC was associated with faster RTs on BX relative to BY trials (i.e., reduced BX RT interference) in only the proactive session (*b* = -0.006, *sd* = 0.002, 95% CI = [-

[2] An ER approaching infinity indicates that the entire posterior distribution exceeded the test value in the predicted direction.

0.010, -0.003], ER = 799, PD = 99.99%). In contrast, higher WMC was associated with comparatively slower RTs on BX relative to BY trials in the reactive session (b = 0.005, sd = 0.002, 95% CI = [0.001, 0.008]). WMC did not influence BX-BY RTs in the baseline session (b = 0.001, sd = 0.002, 95% CI = [-0.002, 0.005]). The SDR comparing the posterior and prior density at the prior mean (b = -0.006) was 1.42, indicating increased evidence in support of the prior mean estimate. The ER of 799 and PD of 99.99% decisively confirmed the predicted effect (see Fig. 1C).

D-Prime Context Effect

Notably, the null effects involving d-prime sensitivity were not replicated. Instead, higher WMC was associated with higher log-odds of target responding on AX trials relative to BX trials (i.e., enhanced d-prime sensitivity) in the proactive session (b = 0.06, sd = 0.02, 95% CI = [0.02, 0.10], ER = 499, PD = 99.80%). The emergence of this finding is not necessarily surprising given that there was already trending evidence in support of this relationship in the exploratory analysis (see Fig. 1D for comparison of the prior and posterior distributions). Interestingly, higher WMC was associated with relatively lower log-odds of AX-BX target responding in the reactive session (i.e., reduced d-prime sensitivity; b = -0.08, sd = .02, 95% CI = [-0.12, -0.04]), whereas WMC did not influence d-prime sensitivity in the baseline session (b = 0.04, sd = 0.02, 95% CI = [-0.01, 0.08]). As seen in Fig. 1D, the SDR comparing the posterior and prior density at the prior mean (b = 0.05) for the effect that higher WMC would be associated with enhanced d-prime sensitivity in the proactive session was 1.32, demonstrating that incorporation of additional data narrowed the posterior distribution closer to the prior mean estimate. Importantly, the ER of 499 and PD of 99.80% provided decisive evidence for the presence of the effect.

General Discussion

The current study sought to advance understanding of the nature of the relationship between WMC and proactive control using the theoretically optimized DMCC version of the AX-CPT task. Importantly, the DMCC AX-CPT places cognitive control mode under experimental manipulation across three testing sessions, each designed to elicit selective engagement of proactive and reactive control in addition to a baseline reference condition. Capitalizing upon this core task feature, we adopted a unique experimental-correlational investigative approach to parse the extent to which WMC influences the *tendency* to engage proactive control relative to the *ability* to implement it. Moreover, to address broader relevant methodological issues pertaining to the ambiguities of using summary/subtraction-based difference scores, statistical nonindependence of repeated subject-level observations, and the inferential limitations of NHST, we leveraged trial-level Bayesian mixed modeling to conduct initial hypothesis testing and subsequent replication analysis across two separate datasets.

Conceptual and Methodological Implications

Overall, our findings were consistent with prior work (Belletier et al., 2019; Boudewyn et al., 2015; Redick, 2014; Redick & Engle, 2011; Richmond et al., 2015; Stawarczyk et al., 2014; Wiemers & Redick, 2018)—higher WMC was indeed broadly associated with enhanced proactive control, as indicated by stronger A-cue bias, reduced BX RT interference, and enhanced d-prime context sensitivity. Central to the novel aim of the investigation, we observed that most of these associations were specific to the proactive session relative to the baseline and reactive sessions. Consequently, our findings demonstrate that the influence of WMC on proactive control is most

robust under regularized conditions during which participants are explicitly trained and instructed to use proactive control across task performance. Although the tendency to use proactive control is undoubtedly intertwined with the ability to implement it, this pattern suggests that within the current experimental approach, the relationship between WMC and proactive control may be more influenced by between-subject variation in the ability to implement proactive control than the preferential tendency to engage it.

With that said, the notable exception is that WMC did not influence BX error interference in the proactive session but was rather associated with reduced error interference in the baseline session. To contextualize this unexpected finding, it is important to note that relative to the baseline session, the proactive session yielded higher overall likelihood of committing correct responses collapsing across trial type, but did not preferentially increase the log-odds of BX trial accuracy (no interactive effect of the proactive session on trial type; see table 6). Together, this introduces the possibility that the proactive manipulation may have instituted a ceiling effect on trial accuracy, thereby restricting the variability needed to detect the expected relationship between WMC and BX error interference.

It is further worth mentioning that the RT interference analysis showed that higher WMC was associated with *reduced* BX RT interference in the *proactive* session, suggesting that although the potency of the proactive manipulation may have instituted a ceiling effect on trial accuracy, participants with higher WMC were nonetheless able to respond more quickly when utilizing proactive control. On the other hand, not only did the baseline session produce slower overall RTs, WMC was associated with slower RTs in the baseline session relative to the other sessions. Rather than reflecting increased tendency to engage in proactive control *per se*, the possible presence of a speed-accuracy tradeoff suggests that that individuals with higher WMC may have adopted a

more cautious style of responding during the baseline session, slowing down to the benefit of increased accuracy. Given these considerations, the overall pattern of results appears consistent with the emerging picture that the relationship between WMC and proactive control is more circumscribed to the proactive session.

Interestingly, WMC was demonstrably associated with *less* A-cue bias, d-prime sensitivity, and BX interference attenuation in the *reactive* session relative to the other sessions. Considering that one of the major aims of the DMCC task battery is to empirically dissociate between proactive and reactive control, the opposing directionality of effects consistently observed in the reactive session introduces the intriguing possibility that between-session dissociations may be evidenced in relation to individual differences. One specific interpretation of this pattern is that individuals with higher WMC possess more flexibility in shifting toward or away from a prospective cue-based strategy (i.e., proactive control) to meet the contextual demands of the situation.

Indeed, this possibility is an extension of the emergent notion that higher WMC does not necessarily translate to greater tendency to engage proactive control. Even though higher WMC may enable relatively more effective utilization of proactive control, it does not appear to lead to overreliance of proactive control during situational conditions that call for the use of reactive control. Put another way, higher WMC is not associated with more proactive control at the expense of less reactive control *per se*. Although the current data cannot fully resolve whether proactive and reactive control are independent constructs or polar ends of a dimensional continuum (see also Gonthier et al., 2016), it does demonstrate that experimentally induced shifts in proactive and reactive control can yield differential relationships between performance and a common individual difference variable. From this broader perspective, the results support and further illustrate the extent of orthogonality between these two modes of control.

Methodologically speaking, the findings reflect the strengths and unique advantages of our analytic approach, enabling us to dovetail exploratory and replication analyses to rigorously test open-ended hypotheses while mitigating common statistical concerns prevalent in the field. First, aggregated trial-level mixed modeling allowed us to leverage the entire dataset while appropriately accounting for non-independence and subject-level performance variability. Consequently, the results obtained here make full use of the data without relying on the computation of summary and difference scores, and do not involve arbitrary partitioning of the data or imposing multiple sets of analyses unnecessarily (e.g., separating across session). Second, we applied Bayesian regression to derive posterior distribution estimates for each model parameter. Furthermore, we showcased how Bayesian updating procedures can be used to conduct confirmatory replication analyses by modeling the posterior parameter distributions obtained from the exploratory dataset as informed priors in analysis of separate holdout data. Collectively, this allowed us to visually and quantitatively assess how incorporation of new data influences the likelihood or amount of evidence favoring the predicted effect relative to the null as opposed to traditional NHST.

With all that said, it must be acknowledged that our conclusions are not unequivocal. In particular, a very recent study utilizing a similar experimental-correlational approach reported null findings, observing that the relationship between WMC and proactive control was not moderated by experimental condition (Rosales et al., 2022). In fact, WMC was unrelated to proactive control performance across baseline, proactive, or reactive conditions. Instead, higher WMC was associated with generally faster RTs across all trial types, leading to the contradictory conclusion that WMC may have a domain general influence on performance but is not related to the specific tendency (or ability) to utilize proactive control. In attempting to reconcile the mixed nature of

these findings, it may be valuable to consider several key methodological factors that might differentiate the current study from Rosales et al (2022).

First, although the proactive control manipulation was essentially identical across the studies, the baseline and reactive conditions differed substantially. Specifically, Rosales et al. (2022) used no-go trials to induce reactive control, whereas we implemented an item-specific cuing approach while including no-go trials across *all* conditions. As detailed elsewhere (Braver et al., 2021; Tang et al., 2021), the purpose of this change was to explicitly distinguish selective *enhancement* of reactive control from reduced engagement of proactive control (i.e., the effect of no-go trials)—our baseline condition was therefore equivalent to the reactive condition in Rosales and colleague's study. Consequently, the experimental distinctions across the control conditions, in combination with our aggregated trial-level mixed modeling approach (designed specifically to assess for session-level interactions) may have increased performance variability and enhanced sensitivity to detect relationships between WMC and proactive control. Second, the mode of study recruitment and task administration (i.e., online MTurk vs. in-person university study) may have also played an influential role, specifically by increasing sample heterogeneity in the current study. Finally, the current study was not age restricted and as a consequence had a relatively older participant sample. As also noted by Rosales and colleagues (2022), an older as opposed to younger sample might also result in greater performance variability, and could buffer against issues related to range restriction.

Although further research is needed to adjudicate between these different possibilities, we ultimately view these studies as complementary efforts toward elucidating the nature of the relationship between WMC and cognitive control. In fact, our findings strongly support Rosales and colleagues' astute postulations that: (1) individuals with higher WMC may be better at

adapting/shifting mode of control in response to changing task demands; and (2) stronger and more explicit manipulations, such as item-specific cueing and strategy training, may be better suited to induce respective reactive and proactive control strategy shifts, than manipulations such as the inclusion of no-go trials. Indeed, it appears reasonable that the ability to detect relationships between WMC and proactive/reactive control (and the strength of the relationship itself) may be contingent upon the potency of the manipulations. Taken together, there is considerable promise in conducting follow-up investigations that seek to validate and build off our approach in service of testing and extending these possibilities. Toward this end, we outline some specific directions for future research below.

Conclusion & Future Directions

The current study leveraged the design innovations of the DMCC task battery to show that the influence of WMC on proactive control may be better characterized as individual differences in the ability to implement control rather than the tendency to engage it. Furthermore, we also found that although individuals with higher WMC exhibited more proactive control in the proactive session, they also exhibited greater shifts away from proactive control in the reactive session—suggesting that proactive and reactive control may be partially dissociable in relation to WMC. Indeed, from the perspective that proactive and reactive control represent orthogonal dimensions of control, it stands to reason that neither the ability nor tendency to engage in proactive control should *necessarily* have to come at the expense of reactive control (and vice versa), even though some individual differences may very well confer specific influence on only one mode of control.

With these considerations in mind, three immediate and potentially fruitful future directions involve: (1) leveraging the retest component of the exploratory dataset to determine the extent to which the reported associations are reliable across repeated testing over time within the same individuals; (2) examining whether the current pattern of findings can generalize to other DMCC tasks such as the Stroop or Cued Task-Switching; and (3) investigating how other individual differences, ideally ones that are both theoretically relevant to the DMC framework and fall within close nomological proximity to WMC, might be related to control using the DMCC task battery. Critically, these three directions synergize to enable incremental evaluation of the construct validity of the DMC framework across repeated assessment, different tasks, and other individual difference constructs, all the while providing a natural avenue through which to adjudicate between the mixed findings mentioned above. Obtaining convergent evidence across these domains would not only strengthen confidence in the validity and generalizability of our conclusions here but would further substantiate the explanatory power of the DMC framework and methodological sensitivity of the DMCC task battery in predicting and testing within-individual and between-individual variability in cognitive control (Braver et al., 2021).

Finally, to bolster these efforts, we encourage future researchers to consider adopting our Bayesian mixed modeling approach which makes full use of trial-level data and computes probabilistic evidence for both the presence and magnitude of predicted effects. For example, the posterior parameter distributions reported here can be used to develop reasonable priors for future studies, including direct replication attempts or new investigations involving different DMCC tasks or individual difference measures. As we have shown here, this enables an accretive approach to research, continuously quantifying and reassessing evidence for the alternative hypothesis or effect of interest across new studies and accumulated data. In conclusion, we hope that other

investigators will take interest in using the DMCC task battery and applying these methods to their own work. Together, we look forward to furthering understanding of the role of individual differences in shaping the development and use of cognitive control.

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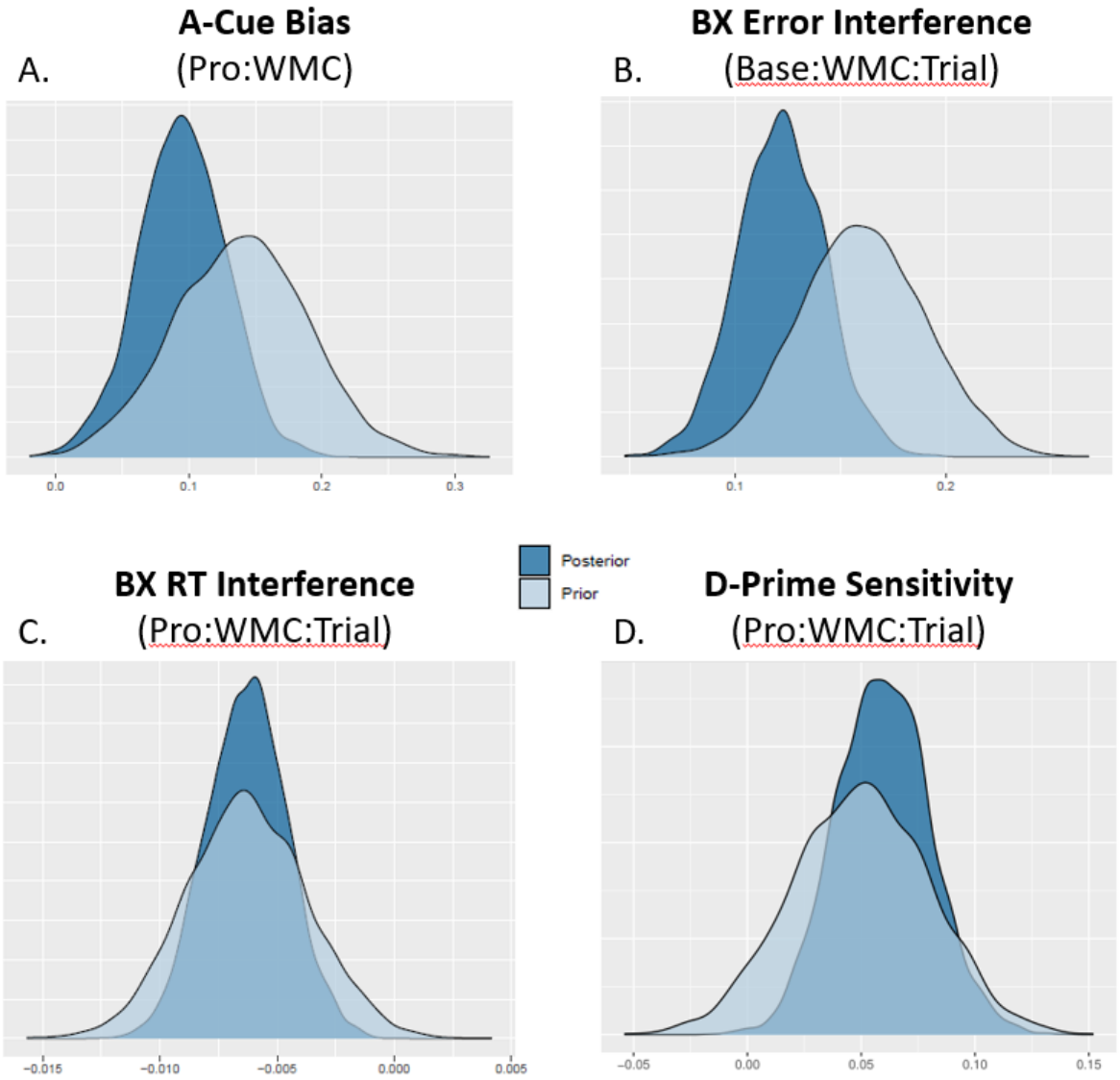
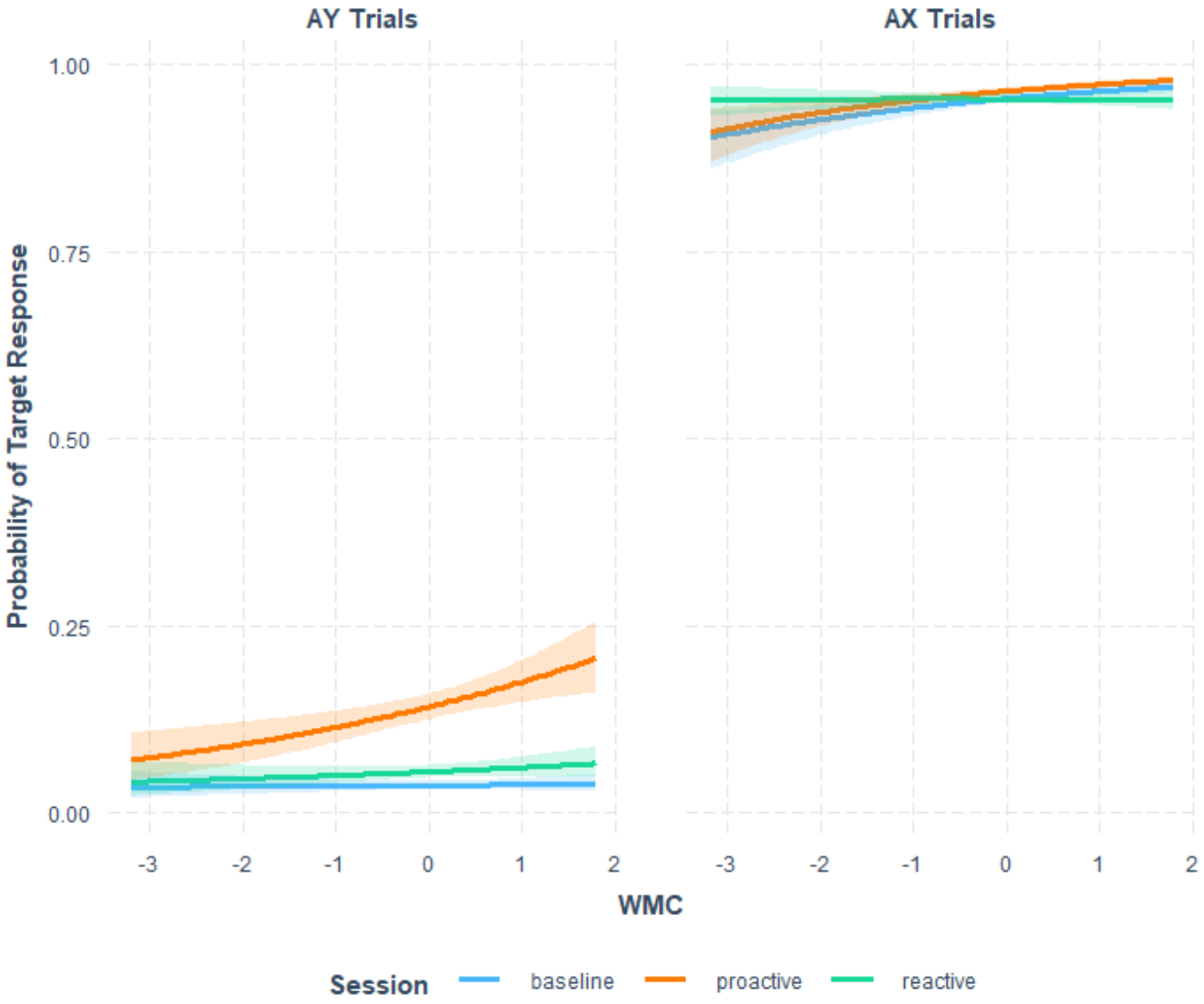
Figure 1. Posterior and prior density distributions of A-cue bias, BX error and RT interference, and d-prime sensitivity estimates. Prior distributions reflect parameter estimates obtained during exploratory analysis, which were entered as informed priors to derive the posterior distributions of the replication analysis.



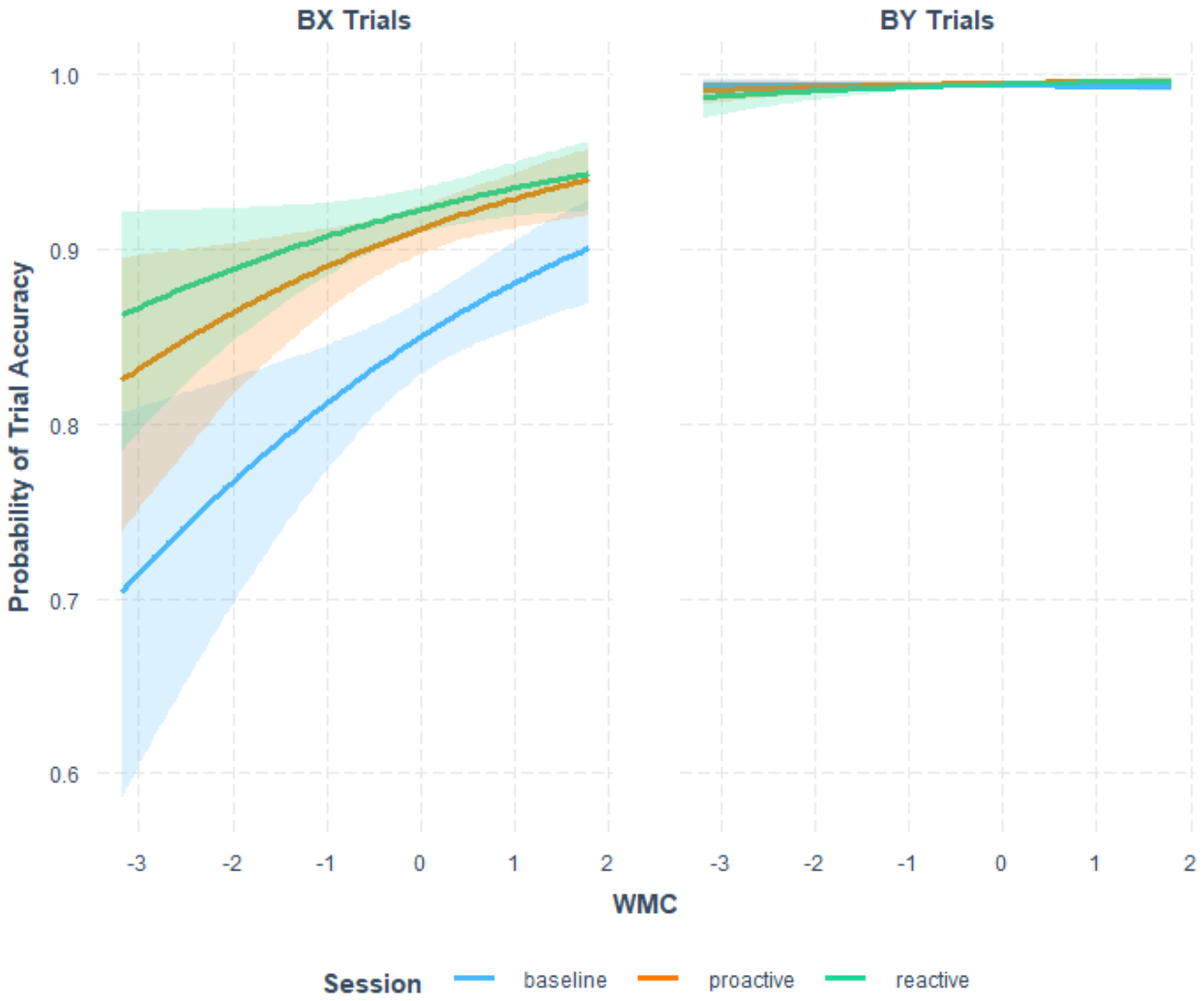
Note. WMC = Working Memory Capacity, Base = Baseline Session, Pro = Proactive Session

Figure 2. Predicted probability of target responding plotted as a function of WMC & Session separated across AY and AX trials.



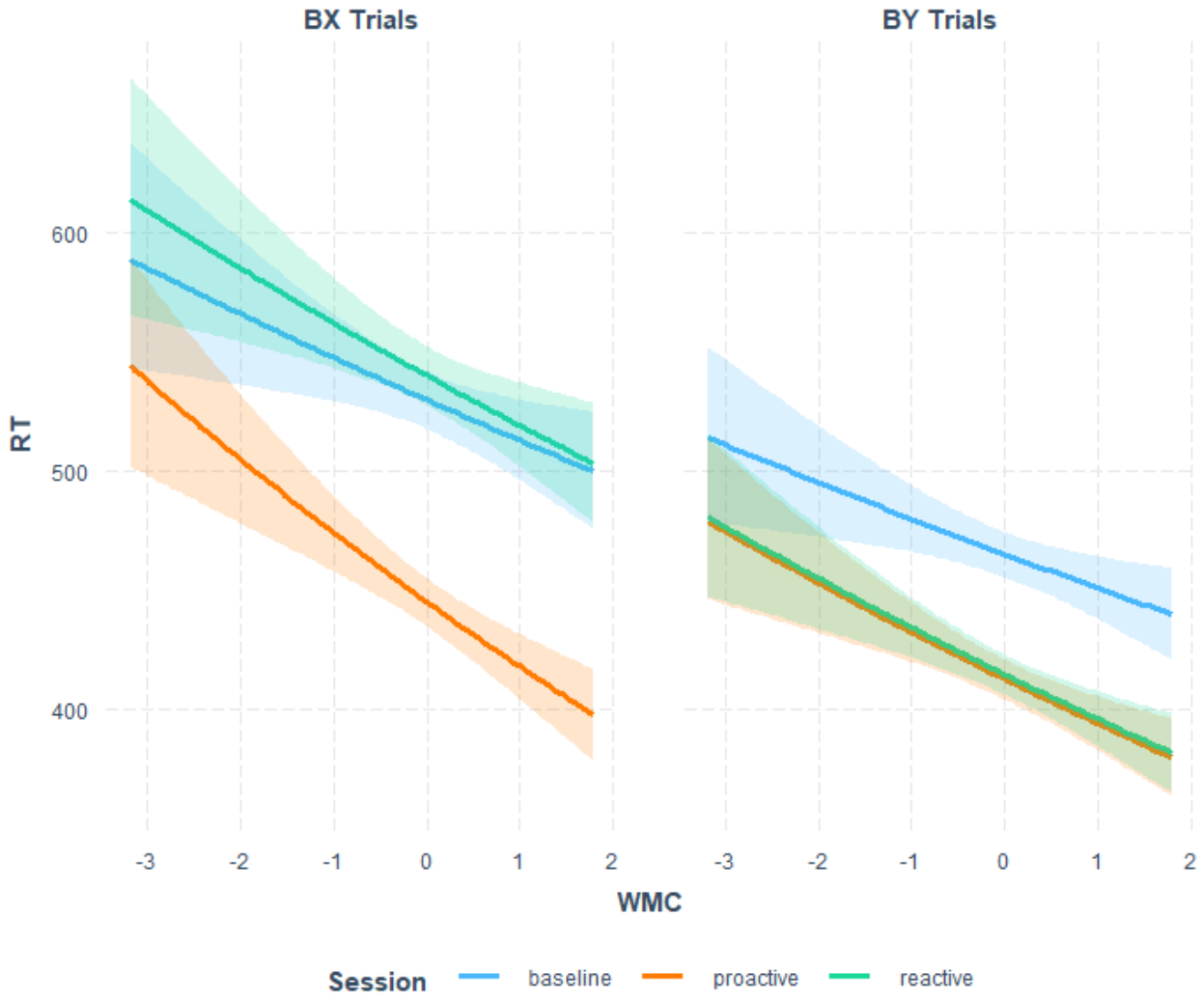
Note. WMC = Working Memory Capacity

Figure 3. Predicted probability of trial accuracy plotted as a function of WMC & Session separated across BX and BY trials.



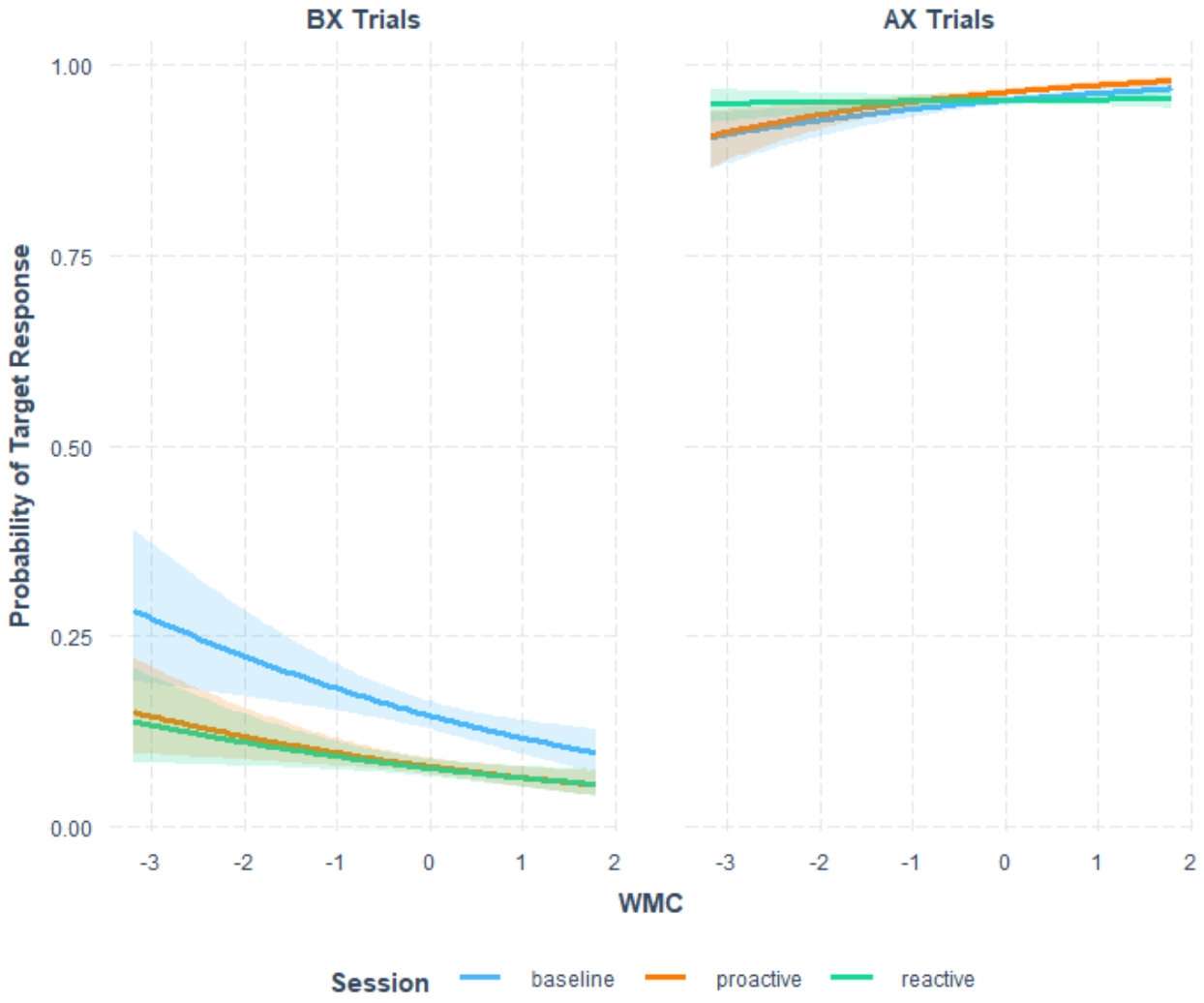
Note. WMC = Working Memory Capacity

Figure 4. Predicted trial RT plotted as a function of WMC & Session separated across BX and BY trials.



Note. WMC = Working Memory Capacity

Figure 5. Predicted probability of target responding plotted as a function of WMC & Session separated across BX and AX trials.



Note. WMC = Working Memory Capacity

Table 1. *Model output from exploratory A-cue bias logistic regression analysis*

Model	Fixed Effects	Estimate (SD)	95% CI	Odds (SD)	R-hat	Bulk ESS	Tail ESS
A-Cue Bias	Intercept	0.23 (0.06)	[0.11, 0.35]*	1.26 (0.08)	1.00	1918	2895
	Base	-0.37 (0.04)	[-0.45, -0.30]*	0.69 (0.03)	1.00	6016	3417
	Pro	0.61 (0.03)	[0.55, 0.68]*	1.85 (0.06)	1.00	5707	3062
	Rea	-0.24 (0.04)	[-0.32, -0.17]*	0.79 (0.03)	1.00	6740	3256
	WMC	0.08 (0.06)	[-0.04, 0.19]	1.08 (0.06)	1.00	1995	2486
	Trial	-2.89 (0.07)	[-3.04, -2.76]*	0.06 (0.00)	1.00	1268	2010
	Base:WMC	-0.08 (0.04)	[-0.15, -0.01]*	0.92 (0.04)	1.00	5157	3114
	Pro:WMC	0.16 (0.03)	[0.09, 0.23]*	1.17 (0.04)	1.00	6173	3238
	Rea:WMC	-0.08 (0.04)	[-0.15, -0.01]*	0.92 (0.03)	1.00	6359	3325
	Base:Trial	-0.24 (0.04)	[-0.32, -0.16]*	0.79 (0.03)	1.00	5223	3385
	Pro:Trial	0.36 (0.03)	[0.30, 0.43]*	1.44 (0.05)	1.00	6690	3196
	Rea:Trial	-0.12 (0.04)	[-0.20, -0.04]*	0.89 (0.03)	1.00	6402	3213
	WMC:Trial	0.06 (0.07)	[-0.09, 0.20]	1.06 (0.08)	1.01	1394	2198
	Base:WMC:Trial	0.00 (0.04)	[0.04, -0.07]	1.00 (0.04)	1.00	5696	3615
	Pro:WMC:Trial	-0.01 (0.03)	[-0.07, 0.06]	0.99 (0.03)	1.00	6126	3451
	Rea:WMC:Trial	0.00 (0.04)	[-0.07, 0.08]	1.00 (0.04)	1.00	6423	3446

Note. WMC = Working Memory Capacity, Base = Baseline Session, Pro = Proactive Session, Rea = Reactive Session, Trial = Trial Type. * denotes 95% CI does not contain 0.

Table 2. *Model output from exploratory BX error interference logistic regression analysis*

Model	**Fixed Effects**	**Estimate (SD)**	**95% CI**	**Odds (SD)**	**R-hat**	**Bulk ESS**	**Tail ESS**
BX Error Interference	Intercept	3.62 (0.10)	[3.24, 3.82]*	37.4 (3.80)	1.00	1319	1858
	Base	-0.31 (0.05)	[-0.39, -0.21]*	0.74 (0.03)	1.00	5241	3466
	Pro	0.30 (0.05)	[0.21, 0.40]*	1.36 (0.07)	1.00	6133	3419
	Rea	0.00 (0.05)	[-0.09, 0.10]	1.01 (0.05)	1.00	6441	3549
	WMC	0.06 (0.10)	[-0.14, 0.24]	1.06 (0.10)	1.00	1299	2118
	Trial	-1.51 (0.07)	[-1.66, -1.38]*	0.22 (0.02)	1.00	2604	2520
	Base:WMC	-0.12 (0.05)	[-0.21, -0.03]*	0.89 (0.04)	1.00	5360	3385
	Pro:WMC	0.02 (0.05)	[-0.08, 0.11]	1.02 (0.05)	1.00	5827	3616
	Rea:WMC	0.10 (0.05)	[0.01, 0.19]*	1.11 (0.05)	1.00	6588	3362
	Base:Trial	-0.13 (0.05)	[-0.21, -0.03]*	0.88 (0.04)	1.00	5741	2882
	Pro:Trial	0.05 (0.05)	[-0.05, 0.15]	1.06 (0.05)	1.00	5603	3275
	Rea:Trial	0.07 (0.05)	[-0.02, 0.16]	1.08 (0.05)	1.00	6425	3394
	WMC:Trial	0.08 (0.06)	[-0.03, 0.21]	1.09 (0.07)	1.00	2230	2744
	Base:WMC:Trial	0.14 (0.05)	[0.06, 0.23]*	1.15 (0.05)	1.00	4982	3488
	Pro:WMC:Trial	-0.09 (0.05)	[-0.19, 0.00]	0.91 (0.05)	1.00	6007	3424
	Rea:WMC:Trial	-0.05 (0.04)	[-0.13, 0.04]	0.96 (0.04)	1.00	5703	3571

Note. WMC = Working Memory Capacity, Base = Baseline Session, Pro = Proactive Session, Rea = Reactive Session, Trial = Trial Type. * denotes 95% CI does not contain 0.

Table 3. *Model output from exploratory BX RT interference lognormal regression analysis*

Model	Fixed Effects	Estimate (SD)	95% CI	R-hat	Bulk ESS	Tail ESS
BX RT	Intercept	6.107 (0.014)	[6.079, 6.135]*	1.00	410	804
Interference	Base	0.060 (0.003)	[0.055, 0.065]*	1.00	12119	6662
	Pro	-0.107 (0.003)	[-0.112, -0.102]*	1.00	11393	7121
	Rea	0.048 (0.003)	[0.043, 0.053]*	1.00	11705	6661
	WMC	-0.040 (0.015)	[-0.069, -0.010]*	1.00	509	1557
	Trial	0.078 (0.004)	[0.069, 0.085]*	1.00	1598	3684
	Base:WMC	0.008 (0.003)	[0.003, 0.013]*	1.00	12995	6254
	Pro:WMC	-0.008 (0.003)	[-0.014, -0.003]*	1.00	11512	6380
	Rea:WMC	0.000 (0.003)	[-0.005, 0.005]	1.00	12489	6641
	Base:Trial	-0.012 (0.003)	[-0.017, -0.007]*	1.00	12269	6723
	Pro:Trial	-0.036 (0.003)	[-0.041, -0.031]*	1.00	12586	6798
	Rea:Trial	0.048 (0.003)	[0.043, 0.053]*	1.00	11946	6420
	WMC:Trial	-0.001 (0.004)	[-0.009, 0.008]	1.00	1737	3776
	Base:WMC:Trial	0.001 (0.003)	[-0.004, 0.007]	1.00	13278	7375
	Pro:WMC:Trial	-0.006 (0.003)	[-0.011, -0.001]*	1.00	11650	6620
	Rea:WMC:Trial	0.005 (0.003)	[0.000, 0.010]	1.00	12653	6804

Note. WMC = Working Memory Capacity, Base = Baseline Session, Pro = Proactive Session, Rea = Reactive Session, Trial = Trial Type. * denotes 95% CI does not contain 0.

Table 4. *Model output from exploratory d-prime sensitivity logistic regression analysis*

Model	**Fixed Effects**	**Estimate (SD)**	**95% CI**	**Odds (SD)**	**R-hat**	**Bulk ESS**	**Tail ESS**
D-Prime Sensitivity	Intercept	0.47 (0.05)	[0.36, 0.57]*	1.60 (0.08)	1.00	1476	2231
	Base	0.15 (0.03)	[0.08, 0.21]*	1.16 (0.04)	1.00	5853	3421
	Pro	-0.04 (0.03)	[-0.11, 0.02]	0.96 (0.03)	1.00	6355	3382
	Rea	-0.10 (0.03)	[-0.17, -0.04]*	0.90 (0.03)	1.00	5199	3565
	WMC	-0.05 (0.05)	[-0.14, 0.05]	0.96 (0.05)	1.00	1911	2477
	Trial	2.66 (0.08)	[2.51, 2.81]*	14.3 (1.13)	1.00	902	1657
	Base:WMC	-0.05 (0.03)	[-0.11, 0.01]	0.95 (0.03)	1.00	6758	3303
	Pro:WMC	0.11 (0.03)	[0.05, 0.18]*	1.12 (0.04)	1.00	6501	2955
	Rea:WMC	-0.06 (0.03)	[-0.13, 0.00]	0.94 (0.03)	1.00	6580	3280
	Base:Trial	-0.28 (0.03)	[-0.34, -0.22]*	0.76 (0.02)	1.00	5412	3213
	Pro:Trial	0.30 (0.03)	[0.23, 0.36]*	1.12 (0.04)	1.00	5867	3809
	Rea:Trial	-0.02 (0.03)	[-0.08, 0.04]	0.98 (0.03)	1.00	5874	3320
	WMC:Trial	0.08 (0.08)	[-0.07, 0.23]	1.08 (0.08)	1.01	982	2015
	Base:WMC:Trial	-0.03 (0.03)	[-0.09, 0.03]	0.97 (0.03)	1.00	6100	3221
	Pro:WMC:Trial	0.05 (0.03)	[-0.01, 0.12]	1.05 (0.04)	1.00	6081	3246
	Rea:WMC:Trial	-0.02 (0.03)	[-0.08, 0.04]	0.98 (0.03)	1.00	7043	3548

Note. WMC = Working Memory Capacity, Base = Baseline Session, Pro = Proactive Session, Rea = Reactive Session, Trial = Trial Type. * denotes 95% CI does not contain 0.

Table 5. *Model output from replication A-cue bias logistic regression analysis*

Model	Fixed Effects	Estimate (SD)	95% CI	Odds (SD)	R-hat	Bulk ESS	Tail ESS
A-Cue Bias	Intercept	0.24 (0.04)	[0.16, 0.32]*	1.27 (0.05)	1.00	2842	2989
	Base	-0.36 (0.03)	[-0.41, -0.31]*	0.70 (0.02)	1.00	6231	2683
	Pro	0.51 (0.02)	[0.47, 0.55]*	1.66 (0.04)	1.00	7774	2906
	Rea	-0.20 (0.03)	[-0.25, -0.14]*	0.82 (0.02)	1.00	7548	3102
	WMC	0.16 (0.04)	[0.07, 0.24]*	1.17 (0.05)	1.00	3503	3416
	Trial	-2.89 (0.05)	[-2.99, -2.80]*	0.06 (0.01)	1.00	2677	2821
	Base:WMC	-0.02 (0.03)	[-0.06, 0.04]	0.99 (0.03)	1.00	6950	2467
	Pro:WMC	0.12 (0.02)	[0.08, 0.16]*	1.13 (0.02)	1.00	6080	3203
	Rea:WMC	-0.12 (0.03)	[-0.17, -0.06]*	0.89 (0.02)	1.00	7543	2930
	Base:Trial	-0.28 (0.03)	[-0.33, -0.23]*	0.76 (0.02)	1.00	7602	3369
	Pro:Trial	0.34 (0.02)	[0.29, 0.38]*	1.40 (0.03)	1.00	7895	3090
	Rea:Trial	-0.08 (0.03)	[-0.14, -0.03]*	0.92 (0.02)	1.00	7978	2615
	WMC:Trial	-0.03 (0.05)	[-0.12, 0.07]	0.97 (0.05)	1.00	3054	2643
	Base:WMC:Trial	-0.08 (0.03)	[-0.13, -0.03]*	0.92 (0.02)	1.00	7344	3282
	Pro:WMC:Trial	0.00 (0.02)	[-0.04, 0.04]	1.00 (0.02)	1.00	6596	3052
	Rea:WMC:Trial	0.07 (0.03)	[0.02, 0.12]*	1.07 (0.03)	1.00	6875	3136

Note. WMC = Working Memory Capacity, Base = Baseline Session, Pro = Proactive Session, Rea = Reactive Session, Trial = Trial Type. * denotes 95% CI does not contain 0.

Table 6. *Model output from replication BX error interference logistic regression analysis*

Model	Fixed Effects	Estimate (SD)	95% CI	Odds (SD)	R-hat	Bulk ESS	Tail ESS
BX Error Interference	Intercept	3.64 (0.07)	[3.50, 3.78]*	38.2 (2.76)	1.00	1177	1968
	Base	-0.29 (0.03)	[-0.35, -0.22]*	0.75 (0.02)	1.00	4473	3030
	Pro	0.14 (0.03)	[0.07, 0.20]*	1.15 (0.04)	1.00	5091	3192
	Rea	0.09 (0.03)	[0.02, 0.15]*	1.09 (0.04)	1.00	4795	3433
	WMC	0.18 (0.07)	[0.05, 0.32]*	1.20 (0.08)	1.00	1111	2032
	Trial	-1.46 (0.05)	[-1.56, -1.37]*	0.23 (0.01)	1.00	2417	2801
	Base:WMC	-0.06 (0.03)	[-0.12, 0.00]	0.94 (0.03)	1.00	4836	3301
	Pro:WMC	0.02 (0.03)	[-0.04, 0.09]	1.02 (0.03)	1.00	5041	3070
	Rea:WMC	0.03 (0.03)	[-0.03, 0.09]	1.04 (0.03)	1.00	5300	3021
	Base:Trial	-0.16 (0.03)	[-0.22, -0.10]*	0.85 (0.03)	1.00	4978	3266
	Pro:Trial	0.02 (0.03)	[-0.05, 0.08]	1.02 (0.03)	1.00	5372	2828
	Rea:Trial	0.13 (0.03)	[0.07, 0.20]*	1.14 (0.04)	1.00	5352	3020
	WMC:Trial	0.05 (0.04)	[-0.03, 0.13]	1.05 (0.04)	1.00	2619	2797
	Base:WMC:Trial	0.09 (0.03)	[0.03, 0.16]*	1.10 (0.04)	1.00	4645	3236
	Pro:WMC:Trial	-0.02 (0.03)	[-0.08, 0.04]	0.98 (0.03)	1.00	5107	3106
	Rea:WMC:Trial	-0.05 (0.03)	[-0.11, 0.00]	0.95 (0.03)	1.00	4971	2801

Note. WMC = Working Memory Capacity, Base = Baseline Session, Pro = Proactive Session, Rea = Reactive Session, Trial = Trial Type. * denotes 95% CI does not contain 0.

Table 7. *Model output from replication BX RT interference lognormal regression analysis*

Model	Fixed Effects	Estimate (SD)	95% CI	R-hat	Bulk ESS	Tail ESS
BX RT Interference	Intercept	6.106 (0.010)	[6.085, 6.126]*	1.00	590	1037
	Base	0.065 (0.002)	[0.062, 0.068]*	1.00	14614	6412
	Pro	-0.082 (0.002)	[-0.086, -0.079]*	1.00	15302	6154
	Rea	0.028 (0.002)	[0.025, 0.032]*	1.00	16177	6493
	WMC	-0.043 (0.011)	[-0.065, -0.022]*	1.00	678	1514
	Trial	0.079 (0.003)	[0.073, 0.084]*	1.00	3099	4688
	Base:WMC	0.011 (0.002)	[0.008, 0.015]*	1.00	16724	6402
	Pro:WMC	-0.012 (0.002)	[-0.015, -0.008]*	1.00	14934	6591
	Rea:WMC	-0.001 (0.002)	[-0.005, 0.002]	1.00	14234	6162
	Base:Trial	-0.013 (0.002)	[-0.017, -0.010]*	1.00	15536	6168
	Pro:Trial	-0.041 (0.002)	[-0.044, -0.037]*	1.00	16074	6138
	Rea:Trial	0.056 (0.002)	[0.053, 0.060]*	1.00	16418	6613
	WMC:Trial	-0.002 (0.003)	[-0.008, 0.004]	1.00	3760	5265
	Base:WMC:Trial	0.001 (0.002)	[-0.002, 0.005]	1.00	15483	6575
	Pro:WMC:Trial	-0.006 (0.002)	[-0.010, -0.003]*	1.00	14834	6715
	Rea:WMC:Trial	0.005 (0.002)	[0.001, 0.008]*	1.00	16286	5892

Note. WMC = Working Memory Capacity, Base = Baseline Session, Pro = Proactive Session, Rea = Reactive Session, Trial = Trial Type. * denotes 95% CI does not contain 0.

Table 8. *Model output from replication d-prime sensitivity logistic regression analysis*

Model	Fixed Effects	Estimate (SD)	95% CI	Odds (SD)	R-hat	Bulk ESS	Tail ESS
D-Prime Sensitivity	Intercept	0.45 (0.04)	[0.37, 0.52]*	1.57 (0.06)	1.00	1942	2755
	Base	0.19 (0.02)	[0.15, 0.23]*	1.21 (0.03)	1.00	7194	3071
	Pro	-0.02 (0.02)	[-0.06 ,0.02]	0.98 (0.02)	1.00	7464	3254
	Rea	-0.14 (0.02)	[-0.18, -0.10]*	0.87 (0.02)	1.00	5820	2748
	WMC	-0.02 (0.04)	[-0.09, 0.06]	0.99 (0.04)	1.00	2023	2323
	Trial	2.69 (0.05)	[2.59, 2.80]*	14.80 (0.80)	1.00	1545	2460
	Base:WMC	0.01 (0.02)	[-0.03, 0.05]	1.01 (0.02)	1.00	6905	2925
	Pro:WMC	0.07 (0.02)	[0.02, 0.11]*	1.07 (0.02)	1.00	7043	3113
	Rea:WMC	-0.08 (0.02)	[-0.12, -0.04]*	0.92 (0.02)	1.00	7339	2919
	Base:Trial	-0.28 (0.02)	[-0.32, -0.24]*	0.76 (0.02)	1.00	7868	3077
	Pro:Trial	0.20 (0.02)	[0.16, 0.24]*	1.22 (0.03)	1.00	7911	3166
	Rea:Trial	0.03 (0.02)	[-0.01, 0.07]	1.03 (0.02)	1.00	6507	2984
	WMC:Trial	0.21 (0.05)	[0.11, 0.31]*	1.24 (0.07)	1.00	1414	2175
	Base:WMC:Trial	0.04 (0.02)	[-0.01, 0.08]	1.04 (0.02)	1.00	5887	2520
	Pro:WMC:Trial	0.06 (0.02)	[0.02, 0.10]*	1.06 (0.02)	1.00	7166	3171
	Rea:WMC:Trial	-0.08 (0.02)	[-0.12, -0.04]*	0.92 (0.02)	1.00	5450	3228

Note. WMC = Working Memory Capacity, Base = Baseline Session, Pro = Proactive Session, Rea = Reactive Session, Trial = Trial Type. * denotes 95% CI does not contain 0.