When effort matters: Expectations of reward and efficacy guide cognitive control allocation

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Abstract

The amount of mental effort we invest in a task is influenced by the reward we can expect if we perform that task well. However, some of the rewards that have the greatest potential for driving these efforts (e.g., jobs, grants) are partly determined by factors outside of one's control. In such cases, effort has more limited *efficacy* for achieving one's desired outcome. We have proposed that people integrate information about the expected reward and efficacy for effort to determine the expected value of control, and then adjust their control (i.e. mental effort) allocation accordingly. Here we test key predictions this makes about behavior and neural activity. We show that participants invest more control in a task when this control is more rewarding and more efficacious, and that these incentive components separately modulate EEG signatures of incentive evaluation and subsequent control allocation. Our findings support the prediction that individuals combine considerations of worth and worthwhileness to determine how they will invest their efforts.

Cognitive control is critical to one's ability to achieve most goals [1-3]–whether to complete a paper in time for its deadline or sending that birthday message amidst a busy workday – but exerting control appears to come at a cost. We experience cognitive control as mentally effortful[4,5], and therefore require some form of incentive to justify investing control in a task[6,7]. For instance, a student is likely to study harder for an exam that has higher stakes (e.g., worth half of their grade) than a lower-stakes exam. Accordingly, research has shown that participants generally exert more mental effort on a cognitive control task (e.g., Stroop, flanker) when they are offered higher rewards for performing well, as evidenced by improved task performance and increased engagement of relevant control circuits [7-16].

In the real world, increased control may not always seem helpful, i.e. *efficacious*, for achieving a desired outcome. For instance, even when the stakes are high, that same student is likely to exert less effort studying if they think that those efforts have little bearing on their score on that exam, say if they felt that grading for the exam is driven mostly by factors out of their control (e.g., random chance). While previous work has closely examined the mechanisms by which people evaluate the potential rewards to expect for a certain control allocation, much less is known about how they evaluate the efficacy of that control, nor how these two values are integrated to determine how much control is invested.

We have recently developed a model that formalizes the roles of evaluation, decision-making, and motivation in allocating and adjusting cognitive control [17,18] (Fig.1). Our model describes how cognitive control can be allocated based on the overall worth of executing different types and amounts of control, which we refer to as their Expected Value of Control (EVC). The EVC of a given control allocation is determined by the extent to which the costs that would need to be incurred (mental effort) are outweighed by the benefits. Critically, these

benefits are a function of both the expected outcomes for reaching one's goal (*reward*, e.g., money or praise) and the likelihood that this goal will be reached with a given investment of control (*efficacy*) (Fig. 1A). The amount of control invested is predicted to increase monotonically with a combination of these two incentive components (Fig. 1B).

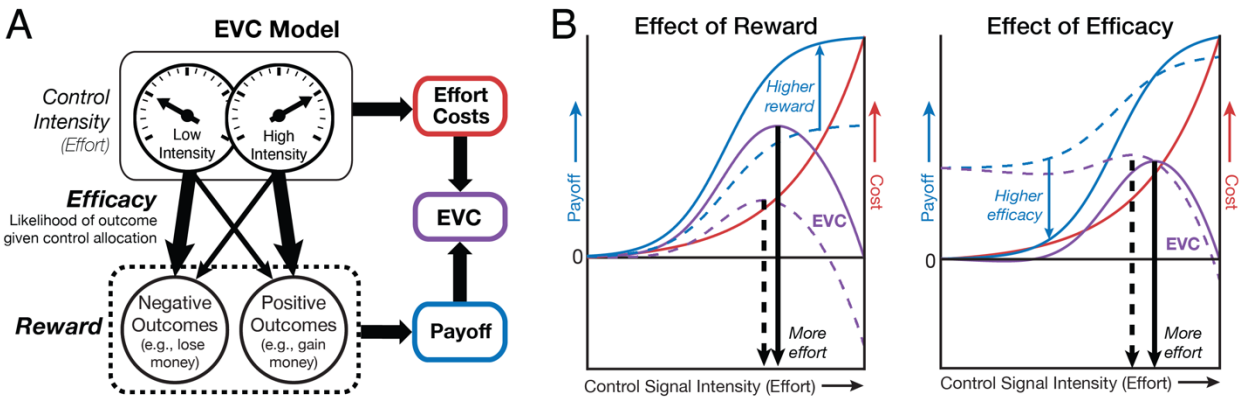

Figure 1. The EVC model predicts that control should increase with expected reward and efficacy. **A**. EVC model. Control intensity is chosen to optimize the trade-off between effort costs and payoff, maximizing the expected value of control. The payoff of a given control signal is determined by the expected reward and efficacy for a given control intensity. **B**. Increasing control signal intensity (x-axis) is associated with greater effort costs (red) but often leads to greater payoffs (blue). The EVC of a given control intensity (purple) increases with expected payoff and decreases with expected costs, determining the optimal (EVC-maximizing) amount of control to invest in a task (black arrows). The model predicts that control should increase when reaching a goal is more rewarding (**left**) and when control is more efficacious for reaching that goal (**right**).

The EVC model integrates over and formalizes past theories that posit roles for reward/utility and/or efficacy/controllability/agency in the motivation to engage in a particular course of action [19-26]. In so doing, our model enables a description of the computational and neural mechanisms of control allocation. For instance, past research has shown that behavioral and neural markers of proactive control increase with increases in anticipated task difficulty[27-32]. Through the lens of the EVC theory (Fig. 1), these difficulty-related increases in control intensity can be accounted for by changes in expected reward (i.e., the harder the task, the less likely you are to achieve a positive outcome) and/or changes in expected efficacy (i.e., the harder the task,

the less helpful a given level of control is for meeting your goal). The latter explains why the relationship between control intensity and task difficulty is nonmonotonic - once the task exceeds a certain difficulty (i.e., once effort is no longer efficacious; [33,34]), a person stops intensifying their mental efforts and instead starts to disengage from a task.

Our theory therefore makes the novel prediction that variability in efficacy (holding expected reward and difficulty constant) should itself be sufficient to drive changes in behavioral and neural signatures of control allocation. The theory makes the further prediction that reward and efficacy should shape incentive processing and associated neural correlates at multiple stages, including during the initial evaluation of each of these incentive components and at the point when those components converge to determine control allocation based on their combined value (EVC).

Here, we test these predictions across two studies using a novel paradigm that explicitly dissociates expectations of reward and efficacy associated with a cognitive control task (the Stroop task; Fig. 2), allowing us to isolate their individual and joint contributions to control allocation. To further examine how reward and efficacy are encoded at different stages of incentive processing, in Study 2 we measured EEG and pupillometry while participants performed this task, allowing us to separately measure the extent to which reward and efficacy are reflected in signals associated with the initial evaluation of the incentives available on a given trial (putatively indexed by the post-cue P3b; [27,35]) versus those associated with preparatory allocation of the control deemed appropriate for that trial (putatively indexed by the contingent negative variation [CNV] occurring prior to the presentation of the target stimulus; [27,30,32,36-38]). Confirming our predictions, both studies found that participants adaptively increased their control allocation (and thus performed better at the task) when expecting higher levels of

reward and efficacy. Study 2 showed that both incentive components amplify event-related potentials (ERPs) associated with distinct stages of incentive processing: incentive evaluation (indexed by the P3b following cue presentation) and control allocation (indexed by the contingent negative variation [CNV] prior to Stroop target onset). Critically, only the CNV reflected the integration of reward and efficacy. The amplitude of both ERPs, but more so the CNV, predicted performance on the upcoming trial, supporting the assumption that these neural signals index different stages in the evaluation and allocation of control.

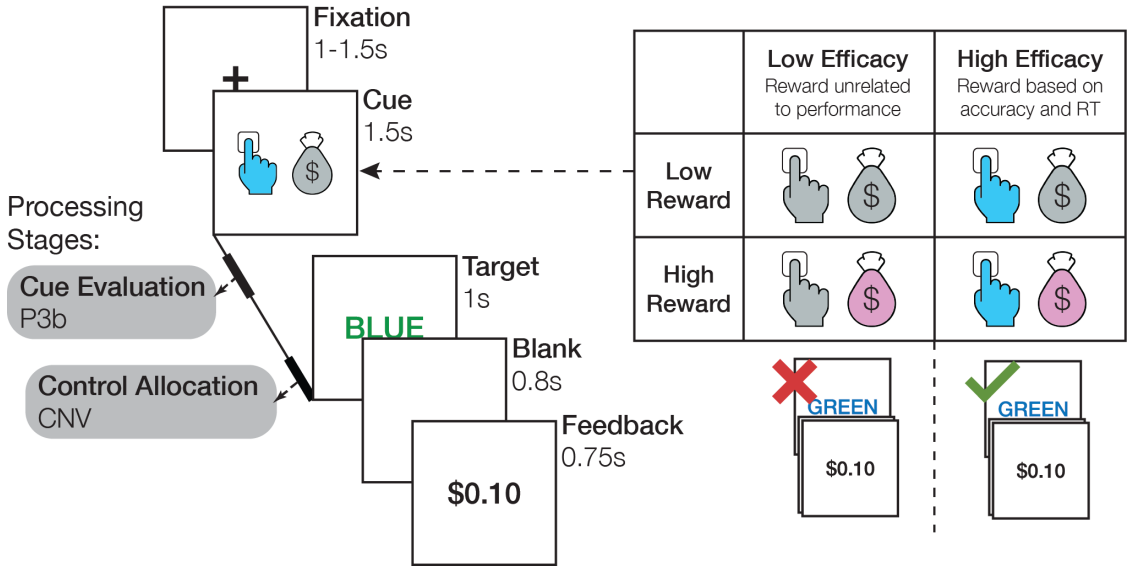

Figure 2. Manipulation of expected reward and efficacy. On each trial, participants view an incentive cue followed by a Stroop stimulus (target) and then feedback indicating how much reward they received. Four different cues signal whether a trial is high or low in reward and efficacy. The P3b and CNV event-related potentials are measured during the interval between cue and target, as indices of cue evaluation (P3b) and control allocation (CNV).

Results

To test the prediction that reward and efficacy together shape cognitive effort investment and task performance, we developed and validated a novel paradigm that manipulates efficacy independently from expected reward (Fig. 2). Specifically, prior to presenting participants with a given Stroop stimulus (the *target*) we cued participants with the amount of monetary reward they would receive if successful on that trial ($0.10 vs. $1.00) and whether success would be determined by their performance (being fast and accurate; *high efficacy*) or whether it would instead be determined independently of their performance (based on a weighted coin flip; *low efficacy*). Using an adaptive yoking procedure, we held expected reward constant across efficacy levels, while also varying reward and efficacy independently of task difficulty (i.e., levels of congruency).

Participants performed this task in an experimental session that measured only behavior (Study 1; N=21) or one that additionally measured EEG activity and pupillometry (Study 2; N=44). Studies 1-2 had the same trial structure but differed slightly in the design of the incentive cues, overall number of trials, and within-trial timing, and were run at different study sites (see Methods). The predictions for Study 2 were preregistered based on findings from Study 1 (osf.io/35akg).

Performance improves with increasing expected reward and efficacy. We predicted that reward and efficacy would together incentivize greater control allocation. Given that participants needed to be fast and accurate to perform well on our task. we expected to find that participants would be faster to respond correctly when they expected control to be more rewarding and more efficacious . Replicating previous findings[27], across both studies we found that reaction times on accurate trials (i.e., accurate RTs) were faster for high compared to low reward trials (Study 1: b

= -9.81, p = .002; Study 2: b = -5.03, p = .004). Critically, accurate RTs were *also* faster for high compared with low efficacy trials (Study 1: b = -14.855, p <.001; Study 2: b = -5.89, p = .016). We further found reward-efficacy interactions in the predicted direction – with the speeding effect of reward being enhanced on high efficacy trials – but this interaction was only significant in Study 2 (Study 1: b = -9.75, p = .116; Study 2: b = -9.23, p = .009; cf. Fig. 3).

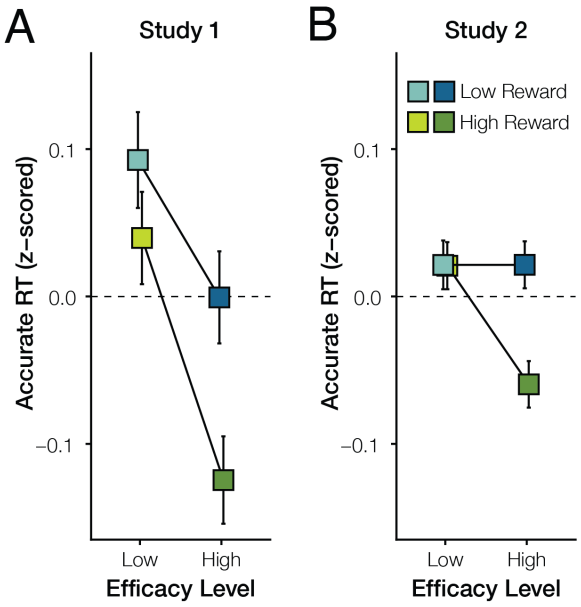

Figure 3. Reward and efficacy improve performance. Participants in both Study 1 (**A**) and Study 2 (**B**) were fastest to give a correct response when both reward and efficacy were high, suggesting that these variables jointly determined control allocation. Error bars represent within-subject standard error of the mean.

Additional analyses confirmed that these performance improvements were not driven by speed-accuracy tradeoffs. Whereas participants were faster when reward or efficacy was high, they were not less accurate (Tables S1-S2). In fact, their accuracies tended to improve when reward or efficacy were high, though only the effect of efficacy on accuracy in Study 2 was significant (b = 0.08, p = .033, Table S2). Together, the faster RTs and more accurate responses suggest that the effects of reward and efficacy on response speed reflected increased control allocation rather than speed-accuracy tradeoffs (lowering of response thresholds; .e., increased impulsivity).

All of the analyses above control for the effects of task difficulty (response congruence) and trial history effects on performance, which in both studies manifested as worse performance (slower and less accurate responding) with increasing response incongruence, and improved performance (faster and more accurate responding) over time (practice effects) (Table S1-S2, Fig. S1). The effects of reward and efficacy on performance did not significantly differ between the two studies (Table S3).

Reward and efficacy levels are reflected in neural signatures of cue evaluation and control allocation. Our behavioral results suggest that participants adjust their mental effort investment (allocation of cognitive control) based on the expected rewards and the degree to which control is perceived to be efficacious; that is, they invest more effort when the expected reward and efficacy are high. To examine the neural and temporal dynamics associated with the processing of these two incentive components, we focused on two well-characterized event-related potentials (ERPs): the P3b, which peaks around 250-550 ms following cue onset and is typically associated with cue evaluation[27]; the CNV, which emerges about 500 ms prior to Stroop target onset and is typically associated with preparatory attention or proactive control[27,28,30,32,36,37].

We found that reward and efficacy modulated both of these ERPs (Table 1, Fig. 4). Replicating past work[27], cues signaling higher rewards were associated with significantly larger amplitudes of both P3b ($b = 0.34$, $p < .001$) and CNV ($b = -0.28$, $p = .001$). Importantly, holding reward constant, cues signaling high rather than low efficacy were likewise associated with significantly larger amplitudes of P3b ($b = 0.44$, $p < .001$) and CNV ($b = -0.30$, $p = .008$).

Crucially, only the CNV tracked the interaction of reward and efficacy ($b = -0.35$, $p = .046$), reflecting the finding that the effect of reward on CNV amplitude was enhanced when

efficacy was high. We did not find a significant reward-efficacy interaction with the P3b (b = -0.01, p = .947). Thus, although reward and efficacy independently modulated the P3b and CNV (i.e., main effects of reward and efficacy on both ERPs), only the CNV reflected their integration (i.e., reward-efficacy interaction). This pattern of results is consistent with our prediction that reward and efficacy are initially evaluated separately (reflected in the P3b), but are subsequently integrated to determine EVC and thereby allocate control (reflected in the CNV).

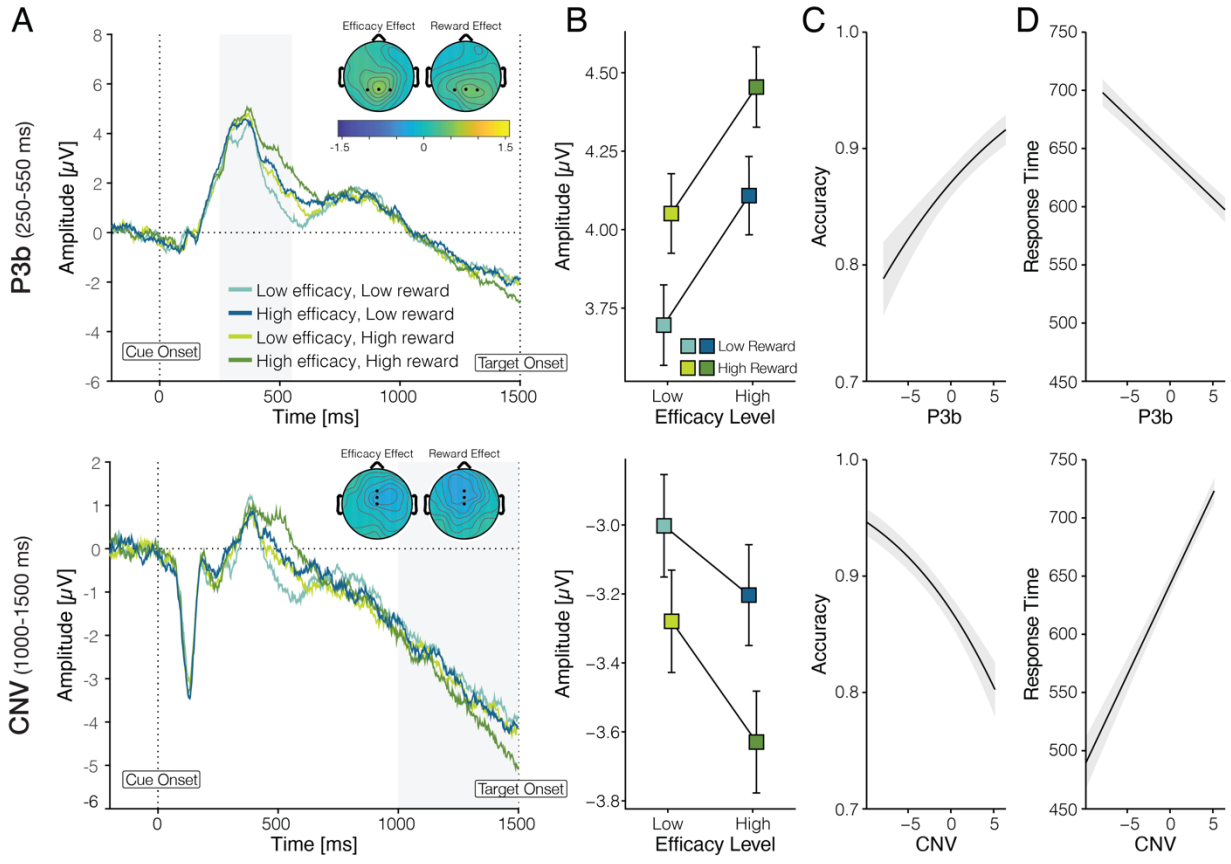

Figure 4. P3b and CNV amplitudes track increases in expected reward and efficacy. A. ERP averages for each incentive condition, separately for the P3b (**top**) and CNV (**bottom**). Shaded areas indicate time windows used for quantification. Topographies show fixed-effect contrasts for reward and efficacy, respectively. **B.** Average ERP amplitudes within the relevant time windows show that the magnitude of the (positive-going) P3b and the (negative-going) CNV increase with greater reward and efficacy. The CNV but not the P3b demonstrated an interaction between reward and efficacy, suggesting an interaction between these that is reflected in behavior (cf. Fig. 3). **C-D.** Fixed effects of ERP amplitude on accuracy (**C**) and accurate RT (**D**) show that increased P3b and CNV predicted better performance on the upcoming trial. Shaded error bars represent standard error of the mean.

Table 1. *Effects of Reward and Efficacy on Neural Signals*

	P3b			**CNV**		
Predictors	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*
(Intercept)	0.26	-0.27 – 0.78	0.339	-0.16	-0.63 – 0.31	0.507
Efficacy	0.44	0.24 – 0.65	**<0.001**	-0.30	-0.53 – -0.08	**0.008**
Reward	0.34	0.16 – 0.51	**<0.001**	-0.28	-0.45 – -0.11	**0.001**
Trial	-0.54	-0.62 – -0.46	**<0.001**	-0.03	-0.12 – 0.05	0.461
P3b Baseline	0.00	-0.01 – 0.01	0.950			
Efficacy:Reward	-0.01	-0.33 – 0.31	0.948	-0.35	-0.69 – -0.01	**0.046**
P3b				0.16	0.14 – 0.17	**<0.001**
CNV Baseline				-0.02	-0.03 – -0.00	**0.005**
Observations	22580			22580		

Note: Statistically significant p-values (< 0.05) are displayed in bold.

Neural signatures of incentive processing predict effort investment. We have shown that reward and efficacy affect behavioral performance (accurate RT) and neural activity during initial cue evaluation (P3b) and subsequent control allocation (CNV), suggesting that these neural signals reflect the transformation of incentives into effort allocation. To test this hypothesis more directly, we included single-trial P3b and CNV amplitude (normalized within-subject) as regressors in our models of accurate RT and accuracy, to test whether variability in these two neural signals explained trial-by-trial variability in task performance (Table 2). We found that both P3b and CNV were associated with better Stroop task performance on the upcoming trial: Larger ERP magnitudes were associated with an increased probability of responding correctly (P3b; b = 0.08, p <.001, CNV: b = -0.10, p <.001), and also with faster accurate RTs (P3b: b = -7.04, p <.001, CNV: b = 15.58, p <.001). Crucially, the CNV's relationship with accurate RT was significantly stronger than the P3b's (X^2 = 18.51, p < .001), providing evidence consistent with our prediction that the CNV plays a more important role in allocating control than the P3b, and with our observation that CNV's relationship with reward

and efficacy more closely resembles that found for accurate RT (i.e., both demonstrating an interaction of the two variables; compare Fig. 3 and Fig. 4B). However, CNV and P3b did not differ reliably in their association with accuracy ($X^2 = 0.78$, $p = .378$). Together, these findings suggest that the P3b and CNV index the transformation of incentive processing into effort investment, a process that entails integration of the reward and efficacy expected on a given trial.

Table 2. *Effects of Neural Signals on Behavioral Performance*

	Accuracy			**Accurate RT**		
Predictors	*Log-Odds*	*CI*	*p*	*Estimates*	*CI*	*p*
(Intercept)	1.87	1.65 – 2.08	**<0.001**	647.66	630.60 – 664.71	**<0.001**
Efficacy	0.08	0.00 – 0.15	**0.044**	-4.00	-8.84 – 0.85	0.106
Reward	0.03	-0.05 – 0.10	0.468	-3.25	-6.85 – 0.34	0.076
P3b	0.08	0.04 – 0.11	**<0.001**	-7.04	-8.87 – -5.21	**<0.001**
CNV	-0.10	-0.14 – -0.06	**<0.001**	15.58	12.02 – 19.15	**<0.001**
Congruency (i-n)	0.65	0.44 – 0.85	**<0.001**	-64.00	-68.85 – -59.14	**<0.001**
Congruency (n-c)	0.34	0.18 – 0.49	**<0.001**	-15.86	-20.27 – -11.45	**<0.001**
Baseline	0.02	-0.02 – 0.07	0.326	2.23	0.05 – 4.42	**0.045**
Trial	0.04	0.00 – 0.08	**0.036**	-10.77	-12.58 – -8.95	**<0.001**
Efficacy:Reward				-9.96	-17.14 – -2.78	**0.007**
Observations	22580			18999		

Note: Statistically significant p-values (< 0.05) are displayed in bold. Congruency (n-i) refers to the comparison between incongruent and neutral Stroop stimulus; Congruency (c-n) refers to the comparison between neutral and congruent Stroop stimulus.

Opposing effects of expected reward and efficacy on pupil responses. As predicted, pupil diameter was larger when expected reward was higher (Fig. 5). This effect was statistically significant from 40 ms following cue onset and lasted throughout the trial (adjusted ps < .044). Contrary to our predictions, however, pupil diameter was *smaller* when efficacy was higher and this effect lasted from 220 ms to 2870 ms following cue onset (adjusted ps < .049). We did not find a significant interaction between reward and efficacy on pupil diameter at any time point within the 5000 ms window we tested (adjusted ps > .062). Although the opposing effects of

reward and efficacy on pupil responses might be difficult to reconcile with the behavioral and EEG results (i.e., performance and ERP magnitudes both scaled positively with reward and efficacy), they nevertheless provide evidence against the idea that our behavioral and EEG results merely reflect changes in arousal. If this alternative explanation were true, pupil diameter—a reliable index of arousal[39-41]—should have increased when either reward or efficacy were high.

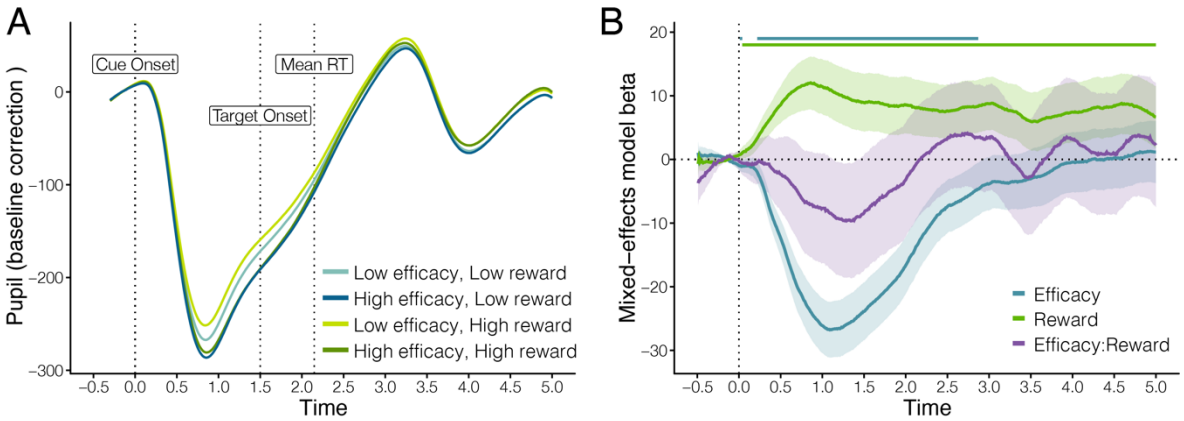

Figure 5. Effects of reward and efficacy on cue-related pupillary responses. **A.** Following an initial cue-related constriction due to the pupillary light reflex, the pupil dilates and pupil responses then begin to diverge depending on levels of expected reward and efficacy. **B.** Time-course of beta coefficients show that reward and efficacy had opposing influences on pupil diameter, with reward positively associated and efficacy negatively associated with pupil diameter. We did not find a significant interaction between these two variables. The horizontal bars at the top of the plot show time points (in 10 ms steps) that survived false-discovery rate (FDR) correction ($p < .05$). Shaded regions reflect 95% confidence intervals of the parameter estimates.

Influences of incentives on EEG signatures of response and feedback monitoring.

While the focus of our study was on measures of incentive processing and proactive control allocation, we performed additional exploratory analyses to examine whether different levels of reward and/or efficacy modulated ERPs associated with the monitoring of response accuracy and subsequent feedback (e.g. in support of reactive control). When examining the error-related negativity (ERN) – a negative deflection in response-locked activity for errors relative to correct

responses [42,43] (though see [44] for ERN elicited by partial errors on correct trials)– we did not find main effects of reward or efficacy (ps > .444) but did find a significant interaction (b = 1.52, p = .001; Fig. S2A; Table S4), whereby ERN amplitude on error trials was greatest (i.e., most negative) on trials with low reward and low efficacy. While this pattern of activity was not predicted, it is notable that it mirrors the pattern we observed for error RTs (Fig. S2), where low-reward/low-efficacy trials were associated with the fastest errors (consistent with effort minimization by, e.g., responding with any key; b = 29.08, p = .001; Table S6). Speculatively, these findings may reflect a shift from more effortful proactive to less effortful reactive control as the expected value of proactive control decreased[45]. Thus, participants may have given these responses more impulsively, and then evaluated those responses immediately after they were executed (see Supplementary Results and Discussion).

We found a different pattern of results when examining the feedback-related negativity (FRN), which typically indexes the difference in feedback-locked activity for trials that resulted in negative compared to positive feedback[46]. Consistent with previous findings[47-52], we found a reliable effect of receipt vs omission of reward on FRN amplitude (b = 0.80, p <.001), and this was enhanced for high reward trials (b = 0.81, p = .007; Table S7). However, in addition to this, we found that effects of reward receipt vs omission on FRN amplitude were reduced for trials with high efficacy compared to those with low efficacy (b = -0.83, p = .007; Table S7). This efficacy-related FRN finding might reflect the fact that, under conditions of low efficacy, reward outcomes are less predictable, thus weakening predictions about forthcoming reward (see Supplementary Discussion).

Discussion

Cognitive control is critical but also costly. People must therefore choose what type and how much control is worth exerting at a given time. Whereas previous studies have highlighted the critical role of expected rewards when making these decisions, our studies highlight a further determinant of control value that is equally critical: how efficacious one perceives their efforts to be (i.e., how much does intensifying control increase their chances of obtaining their reward). Across two studies, we showed that participants were sensitive to both expected reward and efficacy when determining how much control to allocate, and therefore performed best when expecting higher levels of reward and efficacy. Study 2 further demonstrated that both incentive components increase distinct event-related potentials, separately related to cue evaluation and proactive control, providing markers of different stages of incentive processing (evaluation vs. allocation of control). Collectively, these findings lend support to our theory that participants integrate information relevant to determining the Expected Value of Control (EVC), and adjust their control allocation accordingly.

Previous research has shown that people often expend more effort on a task when it promises greater reward[7-16], but this effort expenditure has its limits. If obtaining that reward also requires greater effort (i.e., if higher reward is also associated with greater difficulty), the individual may decide not to invest the effort[53]. Similarly, if difficulty remains constant, but reward becomes less contingent on effort (i.e., efficacy decreases), the individual may again decide to divest their efforts[33,34,54,55]. The EVC theory can account for all of these phenomena, and predicts that expected reward and efficacy will jointly determine how mental effort is allocated (in the form of cognitive control), and that the underlying evaluation process will unfold from initial cue evaluation to eventual control allocation. Specifically, the theory predicts

that these incentive components will be processed sequentially over multiple stages that include initial evaluation of each component, their integration, control allocation, and execution of the allocated control. Our behavioral and neural findings validate the predictions of this theory: When participants expected their control to have greater reward and efficacy, we saw increased neural activity within consecutive ERPs associated with incentive evaluation (P3b) and control allocation (CNV), followed by increases in control (reflected in improved performance).

Our EEG results extend and clarify previous findings. First, a previous study found (like us) that the cue-locked P3b is sensitive to the reward expected on an upcoming trial but not the expected difficulty of the task to be performed on that trial[27]. We varied expected efficacy while holding expected difficulty constant, and show that this is sufficient to generate comparable increases in the cue-locked P3b as variability in expected reward. The difference between our finding and the null result previously observed for task difficulty may be accounted for by the fact that efficacy (like reward) has a monotonic relationship with motivational salience, whereas difficulty does not.

Second, our results extend previous studies that linked the CNV with preparatory attention and proactive control[56-58]. CNV amplitudes scale with a cue's informativeness about an upcoming task[28,36,37,59,60], temporal expectations about an upcoming target[61-63], and an individual's predicted confidence in succeeding on the upcoming trial[64]. Critically, CNV amplitudes also scale with the expected reward for and difficulty of successfully performing an upcoming task[27,30,32,38], suggesting that this component reflects adjustments to proactive control in response to motivationally relevant variables. Here, we extend this body of work by showing that the CNV not only varies with expected efficacy (when isolated from expected difficulty) but that, unlike the P3b, it reflects an interaction between reward and efficacy (i.e., the expected

payoff for control; Fig. 1), and predicts trial-to-trial variability in performance, suggesting that it may index the allocation and/or execution of control based on an evaluation of EVC.

With that said, we note that P3b amplitude was also associated with variability in performance, though to a somewhat lesser degree. It is therefore possible that control allocation was already being determined at this early stage of the trial but, in conjunction with past findings[65], it is perhaps equally or more likely that the P3b indexed the initial evaluation of the motivational relevance of cued incentives. Consistent with this alternate interpretation, we found that the amplitude of the P3b (but not CNV) decreased over the course of the session, potentially reflecting decreased attentiveness to the cues.

Our remaining findings provide evidence against alternative interpretations of these neural results, for instance that they reflect increased arousal or overall engagement throughout the trial. Pupil diameter, an index of arousal[40,41,66], was larger when participants were expecting higher reward but also when expecting lower efficacy. Although this pattern was not predicted in advance, these findings are consistent with evidence that pupil responses more closely track arousal than proactive control[39]. In contrast, the magnitudes of the P3b and CNV increased uniformly with reward and efficacy, suggesting that these two ERPs reflect processes related to proactive control rather than changes in arousal.

Our response- and feedback-related results further suggest that reward and efficacy *specifically* increased proactive control, but not reactive control (performance monitoring; [67]) or overall engagement. Unlike the P3b and CNV, indices of performance monitoring (the ERN and FRN) were not enhanced with greater reward and efficacy, suggesting that these incentive conditions were not simply amplifying the motivational salience of errors and reward outcomes. Rather than reflecting motivational influences on control, the unique patterns of ERN and FRN

amplitudes we observed across conditions may instead provide insight into how participants formed and updated expectations of performance and outcomes across these different conditions[68] (see Supplemental Discussion).

Our study builds on past approaches to studying interactions between motivation and cognitive control[9,12,16,38] by examining changes in effort allocation in response to two incentive components that are predicted to jointly increase one's motivation. Thus, unlike studies that only vary the expected reward for an upcoming task, our behavioral and neural findings cannot be accounted for by general increases in drive, vigor, or arousal. Further, unlike studies that vary the expected difficulty of an upcoming task, resulting in non-monotonic allocation of effort (the classic inverted U-shaped function of effort by difficulty; [69,70,71]) the incentive components we varied should only engender monotonic increases in effort. The monotonic relationship between these incentive components and the value of control (EVC) can in fact account for the nonlinear effect of difficulty on effort allocation: at very high levels of difficulty, a given level of control becomes less and less efficacious. Our study therefore provides the most direct insight yet into the mechanisms and influences of EVC per se, rather than only some of its components.

One interesting feature of our results is that participants engaged in some reasonably high level of effort even when their efforts were completely inefficacious (0% efficacy). There are several plausible explanations for this, including an intrinsic bias towards accuracy (or against error commission)[72] and potential switch costs associated with the interleaved trial structure[73]. For instance, switch costs associated with control adjustments may discourage a significant drop in control following a high efficacy trial. An even more intriguing possibility is that experiences in the real world drive participants to have strong priors that their efforts are generally efficacious (and practice allocating control within a certain range of expected efficacies), making

366 it difficult for them to adjust all the way to the expectation that reward is completely unrelated to
367 their performance on a task.

368 Individual differences in expectations of efficacy may also play a significant role in
369 determining one's motivation to engage in individual tasks or effortful behavior at large[19,21,74-76].
370 Forms of amotivation, like apathy and anhedonia, are common across a variety of psychiatric and
371 neurological disorders, and most likely reflect deficits in the process of evaluating potential
372 incentive components, determining the overall EVC of candidate control signals, specifying the
373 EVC-maximizing control allocation, and/or executing this control. Thus, to understand what
374 drives adaptive versus suboptimal control, we need to find new and better ways to assess what
375 drives these key processing stages underlying motivated effort. By highlighting the crucial role
376 efficacy plays in determining whether control is worthwhile, and identifying candidate neural
377 signatures of the process by which this is evaluated and integrated into decisions about control
378 allocation, our studies pave the way towards this goal.

Method

Study 1

Participants. 21 individuals participated in Study 1 (age: M = 21.14, SD = 5.15; 17 female). Participants gave informed consent and received partial course credits and cash ($5 to $10, depending on their performance and task contingencies) for completing the study. The study was approved by the university's institutional review board.

Design and Procedure. We used a within-subject 2 (reward: high, low) x 2 (efficacy: high, low) design. On high and low reward Stroop trials, participants saw cues that informed them that they would receive $1.00 and $0.10 respectively on the upcoming trial (Fig. 2). Reward levels were crossed with efficacy. On high efficacy trials, whether participants were rewarded depended entirely on their performance (i.e., fast and accurate responses were always rewarded—100% performance-reward contingency). On low efficacy trials, rewards were not contingent on participants' performance; instead, rewards were sampled from a rolling window (size = 10) of reward rate in high efficacy trials to match reward rates across efficacy levels. This approach parallels and builds on recent work examining the influence of performance contingency in the domain of motor control (where individuals simply needed to respond quickly [54,55]), but importantly our task required participants engage cognitive control in order to be fast while also overcoming a prepotent bias to respond based on the color word.

Participants first completed three practice blocks. In the first practice block (80 trials), participants learned the key-color mappings by indicating whether the stimulus XXXXX was displayed in red, yellow, green, or blue (using D, F, J, K keys; counterbalanced across participants). In the second practice block (16 trials), participants learned to associate cues with different reward and efficacy levels (Fig. 2). Finally, participants completed a practice block (64

trials) that resembled the actual task. Then, participants completed the main task. On an individual trial, cues were presented for 1500 ms, followed by a 250 ms fixation cross, followed by a target. To increase task difficulty, the response deadline for each trial was 750 ms but reaction times were recorded as long as a response was made within 1000 ms after Stroop stimulus onset. Immediately after a response was made, feedback was presented for 750 ms. If a response was made before 1000 ms, the remaining time was added to the inter-trial-interval, in which a fixation cross was displayed for 500 ms to 1000 ms. The main task consisted of four blocks of 75 trials each (except for the first 14 participants, who completed 80 trials per block).

After completing the task, participants completed questionnaires. At the end of the experiment, ten trials were randomly chosen and participants received a bonus according to the summed outcomes of those trials.

Study 2

Participants. Before data collection, we conducted a sensitivity analysis, which indicated that a sample size of N = 50 will provide 80% statistical power to detect effect sizes of d = 0.3 or larger. We preregistered our sample size, task design, and analysis plan (osf.io/35akg) and recruited 53 undergraduate students (age M = 20.18, SD = 2.30; 15 male; 38 female). We excluded from all analyses 9 participants who performed poorly on the Stroop task (i.e., below 60% accuracy on high efficacy trials), leaving 44 participants in the final sample. Technical issues also prevented us from recording clean pupil data from 7 participants in this final sample, leaving 37 participants in the pupil analyses. Participants gave informed consent and received partial course credits and cash ($5 to $10, depending on their performance and task contingencies) for completing the study. The study was approved by the university's institutional review board.

Design and Procedure. The behavioral paradigm and procedures were similar to those in Study 1. In addition, we recorded EEG and pupillary responses and changed the following task parameters: No fixation cross was presented during the cue-target interval to avoid inducing visual evoked potentials that will influence the amplitude of the CNV; we added a post-response blank screen (800 ms) to dissociate response evaluation and feedback processing; participants performed eight blocks of 75 trials each.

EEG Recording and Preprocessing. EEG data were recorded from 32 Ag/AgCl electrodes embedded in a stretched Lycra cap (Electro-Cap International, Eaton, OH) at a sampling rate of 512 Hz. Impedances were kept below 5 kΩ during recording. Vertical electrooculography (VEOG) was recorded from two electrodes placed above and below the right eye, respectively. Signals were amplified using ANT TMSi Refa8 device (Advanced Neuro Technology, Enschede, The Netherlands), grounded to the forehead, and referenced online to the average of all electrodes. Offline, the EEG data were re-referenced to the average of electrodes placed on the two earlobes. During preprocessing, continuous data were high-pass filtered at 0.1 Hz (12 dB/oct, zero phase-shift Butterworth filter) and decomposed into independent components using the infomax independent component analysis algorithm implemented in EEGLAB [77]. We inspected the independent components and used the ICLabel EEGLAB extension [78] to help identify and remove blink and noise components.

Pre-processed EEG data were epoched relative to the onset of four events: cue (-200 to 1500 ms), stimulus (-200 to 800 ms), response (-200 to 800 ms), and feedback (-200 to 800 ms). All epochs were baseline-corrected using the mean amplitude before event onset (-200 to 0 ms), and single-trial baseline activity was included as covariates in the statistical models [79]. Epochs containing artifacts, with amplitudes exceeding ±150 μV or gradients larger than 50 μV, were

excluded from further analysis. We focused our analyses on these event-related potentials: cue-locked P3b (250 to 550 ms, averaged across Pz, P3 and P4), cue-locked early and late contingent negative variations (CNV; early: 700 to 1000 ms; late: 1000 to 1500 ms, each averaged across Fz, FCz, and Cz), stimulus-locked N2 (100 ms time-window centered on the peak at ~ 318 ms determined in the grand average ERP), response-locked correct- and error-related negativities (CRN/ERN; 0 to 100 ms), and feedback-locked feedback-related negativity (FRN; quantified peak-to-peak at FCz as the difference between the negative peak between 250 to 350 ms and the positive peak in the preceding 100 ms from the detected peak). For each ERP (except the FRN that was quantified peak-to-peak), we averaged the amplitudes within the specified time window separately for each epoch and exported these single-trial values for further analyses in R. All data analyses were performed using custom MATLAB scripts using EEGLAB functions (cf.[80]).

Pupillometry Recording and Preprocessing. Pupil data were recorded using the EyeLink 1000 Desktop Mount eye tracker (SR Research, Mississauga, Ontario, CA). The EyeLink system was configured using a 35-mm lens, 5-point gaze location calibration, monocular right-eye sampling at a rate of 500 Hz, and centroid fitting for pupil area recordings. All data processing was performed using custom MATLAB and R scripts. Blink artifacts detected using the EyeLink blink detection algorithm were removed using linear interpolation from -200 ms prior to and post-blink onset.

Continuous pupil data were downsampled to 100 Hz and epoched relative to the onset of four events (-1000 to 5000 ms around event onset): cue, stimulus, response, and feedback. All epochs were baseline-corrected to the mean amplitude prior to cue onset (-300 to 0 ms). Single-trial baseline activity (-300 to 0 ms) relative to stimulus, response, and feedback onset were also included as covariates in the statistical models.

Analysis

Classical frequentist statistical analyses were performed in R. The design was entirely within-subjects; unless stated otherwise, all estimates and statistics were obtained by fitting mixed-effects (multilevel) single-trial regression models (two-levels: all factors and neurophysiological responses for each condition were nested within participants) with random intercepts and slopes (unstructured covariance matrix) using the R package lme4 [81]. Random effects were modeled as supported by the data, determined using singular value decomposition, to avoid overparameterization and model degeneration [82,83]. All continuous regressors were mean-centered. Two-tailed probability values and degrees of freedom associated with each statistic was determined using the Satterthwaite approximation implemented in lmerTest.

Behavioral. In both samples, accuracy was analyzed using generalized linear mixed effects models with a binomial link function. The predictors or regressors were reward, efficacy, their interaction, congruency, and trial number. Accurate RTs were modeled using linear mixed effects models and the same predictors. For trial-by-trial predictions of performance with ERPs, the behavioral models were extended by including P3 and CNV amplitudes z-scored within participants as predictors.

EEG. Full linear mixed effects models for all ERPs included reward, efficacy, and their interactions, as well as trial as predictors. For each ERP, we regressed out the baseline activity at the same electrode sites [79]. In the analyses of CNV, we further controlled for variation in the preceding P3b amplitude. For ERN analyses, we included as predictors target congruency, response accuracy, and interactions with incentives. For FRN analyses, we included as predictors the outcome (whether trials were rewarded or not), and interactions with incentives.

Pupillometry. For each event (cue, target, response, feedback), we fitted a mixed-effects model to model trial-by-trial pupil responses at every timepoint from -1000 to 5000 ms around event onset and controlled for error rates with false discovery rate correction [84]. We used this data-driven mass univariate approach because pupil responses are more protracted than EEG neural responses and it can be difficult to visually determine the delayed temporal dynamics of experimental effects. Moreover, unlike ERPs that are similar across tasks, pupil responses vary significantly across tasks. Predictors in the cue-locked models were reward, efficacy, reward-efficacy interaction, and trial number; predictors in the stimulus-locked models were reward, efficacy, congruency, all interactions between reward, efficacy, and congruency, trial number, and pre-stimulus baseline; predictors in the response-locked models were reward, efficacy, accuracy, all interactions between reward, efficacy, and accuracy, trial number, pre-response baseline, and RT; predictors in the feedback-locked models were reward, efficacy, feedback, all interactions between reward, efficacy, and feedback, trial number, and pre-feedback baseline. For each event, we plotted the raw pupil time-series alongside the corresponding mixed-effects model beta coefficients time-series. To avoid visual clutter, we visualized only effects pertinent to our hypotheses.

Acknowledgments

The authors are grateful to Elizabeth Cory and Aravinth Jebanesan for assistance in data collection.

Author Contributions

The authors conceived the study jointly. HL collected the data. RF and HL analyzed the data and wrote the manuscript. All authors edited the manuscript.

Competing Interests

The authors declare no competing interests.

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