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Title

Neural precursors of deliberate and arbitrary decisions in the study of voluntary action

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Abstract

The readiness potential (RP)—a key ERP correlate of upcoming action—is known to precede subjects' reports of their decision to move. Some view this as evidence against a causal role for consciousness in human decision-making and thus against free-will. Yet those studies focused on arbitrary decisions—purposeless, unreasoned, and without consequences. It remains unknown to what degree the RP generalizes to deliberate, more ecological decisions. We directly compared deliberate and arbitrary decision-making during a $1000-donation task to non-profit organizations. While we found the expected RPs for arbitrary decisions, they were strikingly absent for deliberate ones. Our results and drift-diffusion model are congruent with the RP representing accumulation of noisy, random fluctuations that drive arbitrary—but not deliberate—decisions. They further point to different neural mechanisms underlying deliberate and arbitrary decisions, challenging the generalizability of studies that argue for no causal role for consciousness in decision-making to real-life decisions.

Significance Statement

The extent of human free will has been debated for millennia. Previous studies demonstrated that neural precursors of action—especially the readiness potential—precede subjects' reports of deciding to move. Some viewed this as evidence against free-will. However, these experiments focused on arbitrary decisions—e.g., randomly raising the left or right hand. We directly compared deliberate (actual $1000 donations to NPOs) and arbitrary decisions, and found readiness potentials before arbitrary decisions, but—critically—not before deliberate decisions. This supports the interpretation of readiness potentials as byproducts of accumulation of random fluctuations in arbitrary but not deliberate decisions and points to different neural mechanisms underlying deliberate and arbitrary choice. Hence, it challenges the generalizability of previous results from arbitrary to deliberate decisions.

MAIN TEXT

Introduction

Humans typically experience freely selecting between alternative courses of action, say, when ordering a particular item off a restaurant menu. Yet a series of human studies using electroencephalography (EEG) (Haggard & Eimer, 1999; Libet, Gleason, Wright, & Pearl, 1983; Salvaris & Haggard, 2014), fMRI (Bode & Haynes, 2009; Bode et al., 2011; Soon, Brass, Heinze, & Haynes, 2008; Soon, He, Bode, & Haynes, 2013), intracranial (Perez et al., 2015), and single-cell recordings (Fried, Mukamel, & Kreiman, 2011) challenged the veridicality of this common experience. These studies found neural correlates of decision processes hundreds of milliseconds and even seconds prior to the moment that subjects reported having consciously decided.

The seminal research that launched this series of studies was conducted by Benjamin Libet and colleagues (Libet et al., 1983). There, the authors showed that the readiness potential (RP)—a ramp-up in EEG negativity before movement onset, thought to originate from the presupplementary motor area (pre-SMA)—begins before subjects report a conscious decision to act. Libet and colleagues took the RP to be a marker for an unconscious decision to act (Libet et al., 1983; Soon et al., 2008) that, once it begins, ballistically leads to action (Shibasaki & Hallett, 2006). Under that interpretation, the fact that RP onset precedes the report of the onset of the conscious decision to act was taken as evidence that decisions about actions are made unconsciously. And thus the subjective human experience of freely and consciously deciding to act is but an illusion (Harris, 2012; Libet et al., 1983; Wegner, 2002). This finding has been at the center of the free-will debate in neuroscience for almost four decades, captivating scholars from many disciplines in and outside of academia (C. Frith, Blakemore, & Wolpert, 2000; C. D. Frith & Haggard, 2018; Haggard, 2008; Jeannerod, 2006; Lau, Rogers, Haggard, & Passingham, 2004; Mele, 2006; Wegner, 2002).

Critically, in the above studies, subjects were told to arbitrarily move their right hand or flex their right wrist; or they were instructed to arbitrarily move either the right or left hand (Haggard, 2008; Hallett, 2016; Roskies, 2010). Thus, their decisions when and which hand to move were always unreasoned, purposeless, and bereft of any real consequence. This stands in sharp contrast to many real-life decisions that are deliberate—i.e., reasoned, purposeful, and bearing consequences (Ullmann-Margalit & Morgenbesser, 1977): which clothes to wear, what route to take to work, as well as more formative decisions about life partners, career choices, and so on.

Deliberate decisions have been widely studied in the field of neuroeconomics (Kable & Glimcher, 2009; Sanfey, Loewenstein, McClure, & Cohen, 2006) and in perceptual tasks (Gold & Shadlen, 2007). Yet, interestingly, little has been done in that field to assess the relation between decision-related activity, subjects' conscious experience of deciding, and the neural activity instantaneously contributing to this experience. Though some studies compared, for example, internally driven and externally cued decisions (Thut et al., 2000; Wisniewski, Goschke, & Haynes, 2016), or stimulus-based and intention-based actions (Waszak et al., 2005), these were typically arbitrary decisions and actions with no real implications. Therefore, the results of these studies provide no direct evidence about potential differences between arbitrary and deliberate decisions.

Such direct comparisons are critical for the free will debate, because it is deliberate, rather than arbitrary, decisions that are at the center of philosophical arguments about free will and moral responsibility (Breitmeyer, 1985; Maoz & Yaffe, 2015; Roskies, 2010). Deliberate decisions

typically involve more conscious and lengthy deliberation and might thus be more tightly bound to conscious processes than arbitrary ones. Consequently, if the RP is a marker for unconscious decisions, while deliberate decisions are driven more by conscious than by unconscious processes, then the RP might be substantially diminished, or even absent, for deliberate decisions.

Another reason that the RP might be completely absent during deliberate decisions has to do with a recent computational model (Schurger, Sitt, & Dehaene, 2012). This model claims that the RP—which has been deemed a preparatory signal with a causal link to the upcoming movement—actually reflects an artifact that results from a combination of (i) biased sampling stemming from the methodology of calculating this component and (ii) autocorrelation (or smoothness) in the EEG signal. The RP is calculated by aligning EEG activity (typically in electrode C_z) to movement onset, then segmenting a certain time duration around each movement onset, and finally averaging across all movements. Hence, we only look for an RP before movement onset, which results in biased sampling (as 'movement absent' is not probed). Put differently, we search for and generally find a ramp up in EEG negativity in C_z before movement onset. But we do not search for movement onset every time there is a ramp up in EEG negativity on C_z. What is more, as EEG is autocorrelated, ramps up or down are to be expected (unlike, say, for white-noise activity).

Schurger and colleagues demonstrated that RPs can be modeled using a mechanistic, stochastic, autocorrelated, drift-diffusion process that integrates noise to a bound (or threshold; see Model section in Methods for details). In the model, it is only the threshold crossing that reflects decision onset and directly leads to action. And thus the beginning of (what is in hindsight and on average) the ramp up toward the threshold is certainly not the onset of the decision that ballistically leads to the threshold crossing and hence to movement onset (Schurger et al., 2012). This interpretation of the RP thus rather takes the sting out of the Libet argument against free will, as the latter was based on interpreting the RP of an unconscious decision to act. Importantly for our purposes, within the framework of the model, this artificial accumulation of stochastic fluctuations toward a threshold is expected to occur for arbitrary decisions, but not for deliberate ones. Unlike arbitrary decisions, deliberate decisions are generally not driven by random fluctuations. Rather, it is the values of the decision alternatives that mainly drive the decision and ultimately lead to action. Therefore, if the RP indeed reflects the artificial accumulation of stochastic fluctuations, as the model suggests, a key prediction of the model is that no RP will be found for deliberate decisions (see more below).

Thus, demonstrating the absence of an RP in deliberate decisions challenges the interpretation of the RP as a general index of internal, unconscious decision-making; if this interpretation were correct, such a marker should be found for all decision types. What is more, and importantly, it questions the generalizability of any studies focused on arbitrary decisions to everyday, ecological, deliberate decisions. In particular, it challenges RP-based claims relating to moral responsibility (Haggard, 2008; Libet, 1985; Roskies, 2010), as moral responsibility can be ascribed only to deliberate decisions.

Here, we tested this hypothesis and directly compared the neural precursors of deliberate and arbitrary decisions—and in particular the RP—on the same subjects, in an EEG experiment. Our experiment utilized a donation-preference paradigm, in which a pair of non-profit organizations (NPOs) were presented in each trial. In deliberate-decision trials, subjects chose to which NPO they would like to donate $1000. In arbitrary-decision trials, both NPOs received an equal donation of $500, irrespective of subjects' key presses (Fig. 1). In both conditions, subjects were instructed to report their decisions as soon as they made them, and their hands were placed on the response keys, to make sure they could do so as quickly as

possible. Notably, while the visual inputs and motor outputs were identical between deliberate and arbitrary decisions, the decisions' meaning for the subjects was radically different: in deliberate blocks, the decisions were meaningful and consequential—reminiscent of important, real-life decisions—while in arbitrary blocks, the decisions were meaningless and bereft of consequences—mimicking previous studies of volition.

Results

Behavioral Results

Subjects' reaction times (RTs) were analyzed using a 2-way ANOVA along decision type (arbitrary/deliberate) and difficulty (easy/hard). This was carried out on log-transformed data (raw RTs violated the normality assumption; $W=0.94$, $p=0.001$). As expected, subjects were substantially slower for deliberate ($M=2.33$, $SD=0.51$) than for arbitrary ($M=0.99$, $SD=0.32$) decisions (Fig. 2, left; $F(1,17)=114.87$, $p<0.0001$ for the main effect of decision type). A main effect of decision difficulty was also found ($F(1,17)=21.54$, $p<0.0005$), with difficult decisions ($M=1.77$, $SD=0.40$) being slower than easy ones ($M=1.56$, $SD=0.28$). Importantly, subjects were significantly slower for hard ($M=2.52$, $SD=0.62$) vs. easy ($M=2.13$, $SD=0.44$) decisions in the deliberate case ($t(17)=4.78$, $p=0.0002$), yet not for the arbitrary case ($M=1.00$, $SD=0.34$; $M=0.98$, $SD=0.32$, for hard and easy arbitrary decisions, respectively; $t(17)=1.01$, $p=0.33$; $F(1,17)=20.85$, $p<0.0005$ for the interaction between decision type and decision difficulty). This validates our experimental manipulation and further demonstrates that, in deliberate decisions, subjects were making meaningful decisions, affected by the difference in the values of the two NPOs, while for arbitrary decisions they were not. What is more, the roughly equal RTs between easy and hard arbitrary decisions provide evidence inconsistent with concerns that subjects were deliberating during arbitrary decisions.

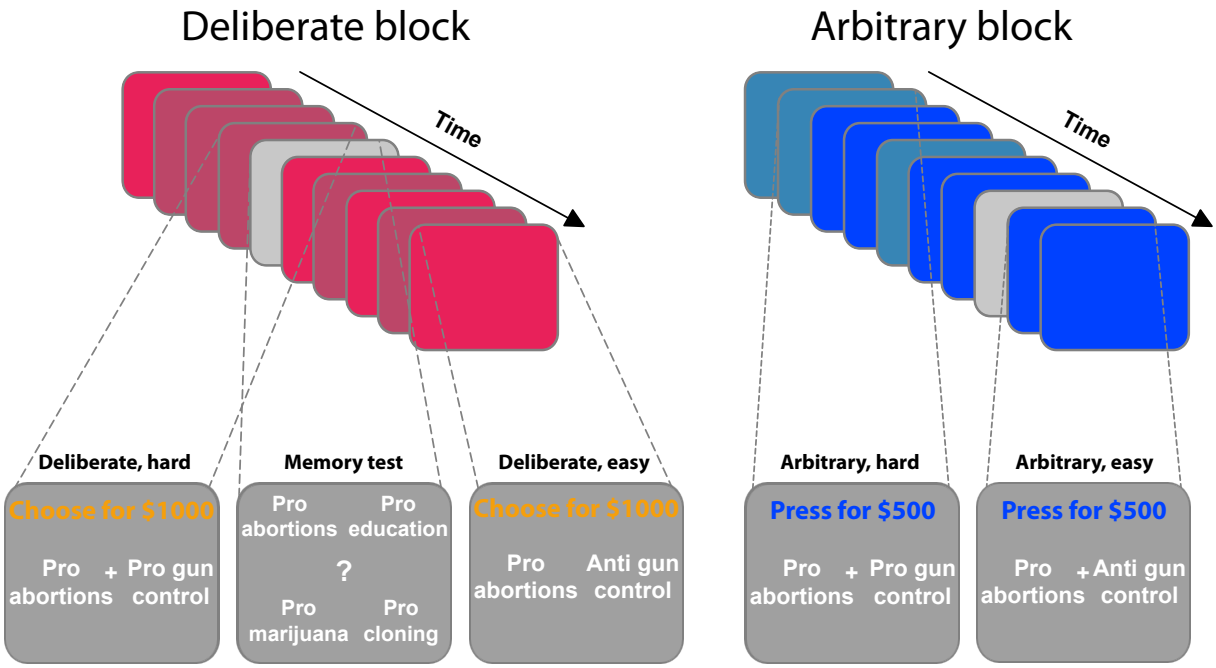

Figure 1: Experimental paradigm. The experiment included deliberate (red, left panel) and arbitrary (blue, right panel) blocks, each containing nine trials. In each trial, two causes—

reflecting NPO names—were presented, and subjects were asked to either choose to which NPO they would like to donate (deliberate), or to simply press either right or left, as both NPOs would receive an equal donation (arbitrary). They were specifically instructed to respond as soon as they reached a decision, in both conditions. Within each block, some of the trials were easy (lighter colors) decisions, where the subject's preferences for the two NPOs substantially differed (based on a previous rating session), and some were hard decisions (darker colors), where the preferences were more similar; easy and hard trials were randomly intermixed within each block. To make sure subjects were paying attention to the NPO names, even in arbitrary trials, and to better equate the cognitive load between deliberate and arbitrary trials, memory tests (in light grey) were randomly introduced. There, subjects were asked to determine which of four NPO names appeared in the immediately previous trial. For a full list of NPOs and causes see Supplementary Table 1.

The consistency between subjects' choices throughout the main experiment and the NPO ratings they gave prior to the main experimental session was also analyzed using a 2-way ANOVA (see Methods). As expected, subjects were highly consistent with their own, previous ratings when making deliberate decisions (M=0.91, SD=0.04), but not when making arbitrary ones (M=0.52, SD=0.04; Fig. 2, right; $F(1,17)=946.55$, $p<0.0001$, $BF=2.32*10^{29}$) for the main effect of decision type. A main effect of decision difficulty was also found ($F(1,17)=57.39$, $p<0.0001$, though BF=1.57), with hard decisions evoking less consistent scores (M=0.66, SD=0.05) than easy ones (M=0.76, SD=0.03). Again, decision type and decision difficulty interacted ($F(1,17)=25.96$, $p<0.0001$, BF=477.47): subjects were much more consistent with their choices in easy (M=0.99, SD=0.02) vs. hard (M=0.83, SD=0,64) deliberate decisions ($t(17)=11.15$, $p<0.0001$, $BF=3.68*10^{6}$), than they were in easy (M=0.54, SD=0.07) vs. hard (M=0.49, SD=0.05) arbitrary decisions ($t(17)=2.50$, $p=0.023$, BF=2.69). Nevertheless, though subjects were around chance (i.e., 0.5) in their consistency in arbitrary decisions (ranging between 0.39 and 0.64), it seems that some subjects were slightly influenced by their preferences in easy-arbitrary decisions trials, resulting in the significant difference between hard-arbitrary and easy-arbitrary decisions above, though the Bayes factor was inconclusive. Finally, no differences were found between subjects' tendency to press the right vs. left key in the different conditions (both main effects and interaction: $F<1$).

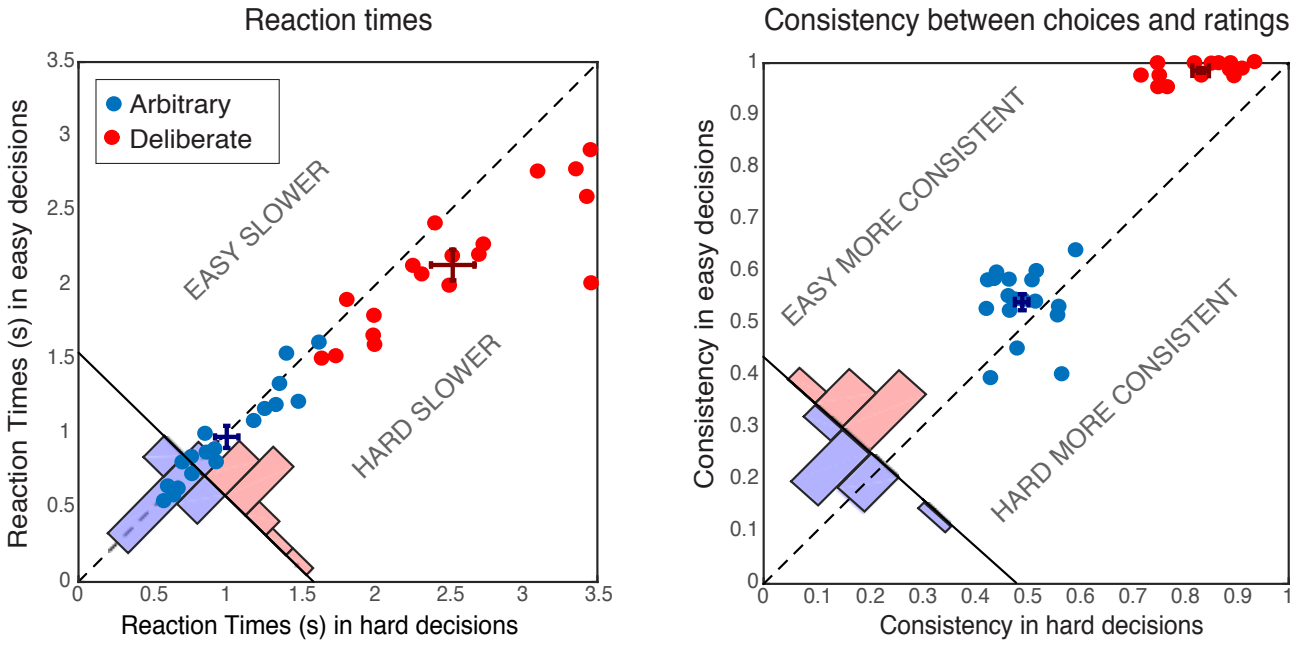

Figure 2: Behavioral results. Reaction Times (RTs; left) and Consistency Grades (CG; right) in arbitrary (blue) and deliberate (red) decisions. Each dot represents the average RT/CG for easy and hard decisions for an individual subject (hard decisions: x-coordinate; easy decisions: y-coordinate). Group means and SEs are represented by dark red and dark blue crosses. The red and blue histograms at the bottom-left corner of each plot sum the number of red and blue dots with respect to the solid diagonal line. The dashed diagonal line represents equal RT/CG for easy and hard decisions; data points below that diagonal indicate longer RTs or higher CGs for hard decisions. In both measures, arbitrary decisions are more centered around the diagonal than deliberate decisions, showing no or substantially reduced differences between easy and hard decisions.

EEG Results: Readiness Potential (RP)

The RP is generally held to index unconscious readiness for upcoming movement (Haggard, 2008; Kornhuber & Deecke, 1990; Libet et al., 1983; Shibasaki & Hallett, 2006); although more recently, alternative interpretations of the RP have been suggested (Miller, Shepherdson, & Trevena, 2011; Schmidt, Jo, Wittmann, & Hinterberger, 2016; Schurger et al., 2012; Trevena & Miller, 2010; Verleger, Haake, Baur, & Śmigasiewicz, 2016). It has nevertheless been the standard component studied in EEG versions of the Libet paradigm (Haggard, 2008; Haggard & Eimer, 1999; Hallett, 2007; Libet, 1985; Libet et al., 1983; Libet, Wright, & Gleason, 1982; Miller et al., 2011; Schurger et al., 2012; Shibasaki & Hallett, 2006; Trevena & Miller, 2010). As is common, we measured the RP over electrode C_z in the different conditions by averaging the activity across trials in the 2 s prior to subjects' movement.

Focusing on the last 500 ms before movement onset for our statistical tests, we found a clear RP in arbitrary decisions, yet RP amplitude was not significantly different from 0 in deliberate decisions (Fig. 3A; $F(1,17)=11.86$, $p=0.003$, BF=309.21 for the main effect of decision type; in t-tests against 0 for this averaged activity in the different conditions, corrected for multiple comparisons, an effect was only found for arbitrary decisions (hard: $t(17)=5.09$, $p=0.0001$, BF=307.38; easy: $t(17)=5.75$, $p<0.0001$, BF=1015.84) and not for deliberate ones; the Bayes factor—while trending in the right direction—indicated inconclusive evidence (hard: $t(17)=1.24$, $p>0.5$, BF=0.47; easy: $t(17)=1.84$, $p=0.34$, BF=0.97). Our original baseline was stimulus locked (see Methods). And we hypothesized that the inconclusive Bayes factor for deliberate trials had to do with a constant, slow, negative drift that our model predicted for deliberate trials (see below) rather than reflecting a typical RP. As the RTs for deliberate trials were longer than for arbitrary ones, this trend might have become more pronounced for those trials. To test this, we switched the baseline period to -1000 ms to -500 ms relative to *movement* onset (i.e., a baseline that immediately preceded our time of interest window). Under this analysis, we found moderate evidence that deliberate decisions (pooled across decision difficulty) are not different from 0 (BF=0.332), supporting the claim that the RP during the last 500 ms before response onset was completely absent (BF for similarly pooled arbitrary decisions was $5.07 \cdot 10^4$).

In an effort to further test for continuous time regions where the RP is different from 0 for deliberate and arbitrary trials, we ran a cluster-based nonparametric permutation analysis (Maris & Oostenveld, 2007) for all four conditions against 0. Using the default parameters (see Methods), we found a prolonged cluster (~1.2s) of activation that reliably differed from 0 in both arbitrary conditions (designated by horizontal blue-shaded lines above the x axis in Fig. 3A). The same analysis revealed no clusters of activity differing from zero in either of the deliberate conditions.

In a similar manner, regressing voltage against time for the last 1000 ms before response onset, the downward trend was significant for arbitrary decisions (Fig. 3B; $p<0.0001$, BF$>10^{25}$ for

both easy and hard conditions) but not for deliberate decisions, with the Bayes factor indicating conclusive evidence for no effect (hard: $p>0.5$, BF=0.09; easy: $p=0.35$, BF=0.31; all Bonferroni corrected for multiple comparisons). Notably, this pattern of results was also manifested for single-subject analysis (Fig. 4; 14 of the 18 subjects had significant downward slopes for arbitrary decisions—i.e., $p<0.05$, Bonferroni corrected for multiple comparisons—when regressing voltage against time for every trial over the last 1000 ms before response onset; but only 5 of the 18 subjects had significant downward slopes for the same regression analysis for deliberate decisions; see Methods. In addition, the average slopes for deliberate and arbitrary decisions were -0.28±0.25 and -1.9±0.32 (mean±SE), respectively, a significant difference: $t(17)=4.55$, $p<0.001$, BF=380.02). The regression analysis complements the averaged amplitude analysis above, and further demonstrates that the choice of baseline cannot explain our results. This is because the slopes of linear regressions are, by construction, independent of baseline.

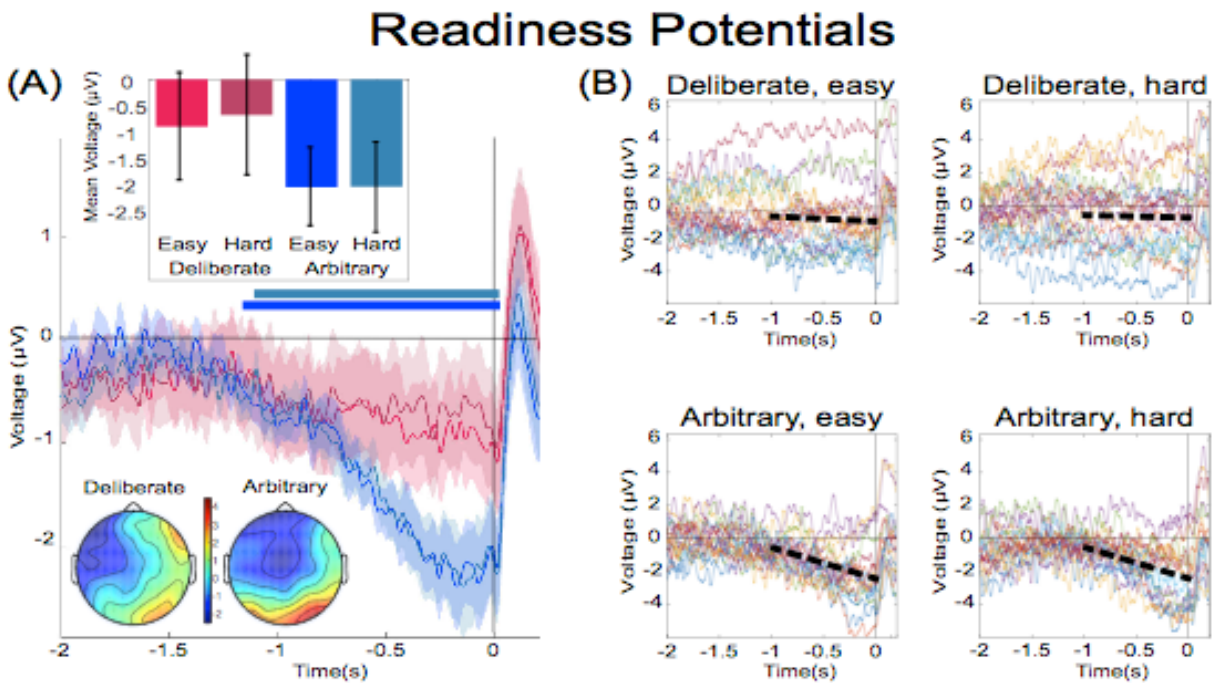

Figure 3: The readiness potentials for deliberate and arbitrary decisions. (A) Mean and SE of the readiness potential (RP; across subjects) in deliberate (red shades) and arbitrary (blue shades) easy and hard decisions in electrode C_z, as well as scalp distributions. Zero refers to time of right/left movement, or response, made by the subject. Notably, the RP significantly differs from zero and displays a typical scalp distribution for arbitrary decisions only. Similarly, temporal clusters where activity was significantly different from 0 were found for arbitrary decisions only (horizontal blue lines above the x axis). Scalp distributions depict the average activity between -0.5 and 0 s, across subjects. The inset bar plots show the mean amplitude of the RP, with 95% confidence intervals, over the same time window. Response-locked potentials with an expanded timecourse, and stimulus-locked potentials are given in Fig. 6B and 6A, respectively. The same (response-locked) potentials as here, but with a *movement-locked baseline* of -1 to -0.5 s (same as in our Bayesian analysis), are given in Fig. 6C. **(B)** Individual subjects' C_z activity in the four conditions (n=18). The linear-regression line for voltage against time over the last 1000 ms before response onset is designated by a dashed, black line. The lines have slopes significantly different from 0 for arbitrary decisions only. Note that the waveforms converge to an RP only in arbitrary decisions.

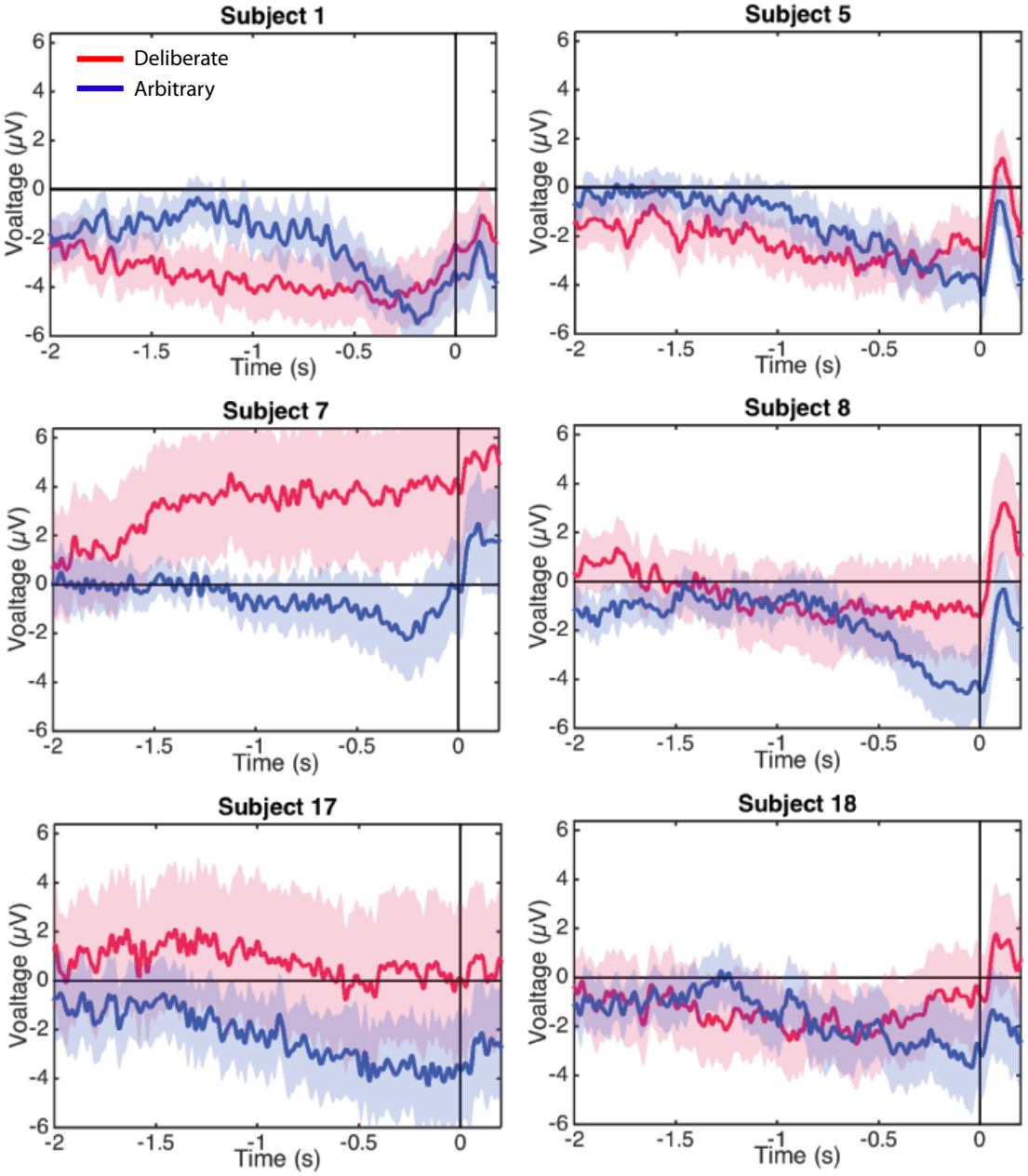

Figure 4: Individual-subjects RPs. Six examples of for individual subjects' RPs for deliberate decisions (in red) and arbitrary ones (in blue) pooled across decision difficulty.

Control analyses

We further tested whether differences in reaction time between the conditions, eye movements, filtering, and subjects' consistency scores might explain our effect. We also tested whether the RPs might reflect some stimulus-locked potentials or be due to baseline considerations.

Differences in reaction times (RT) between conditions, including stimulus-locked potentials and baselines, do not drive the effect

RTs in deliberate decisions were typically more than twice as long as RTs in arbitrary decisions. We therefore wanted to rule out the possibility that the absence of RP in deliberate decisions stemmed from the difference in RTs between the conditions. We carried out six analyses for this purpose. First, we ran a median split analysis—dividing the subjects into two

groups based on their RTs: lower (faster) and higher (slower) than the median, for deliberate and arbitrary trials, respectively. We then ran the same analysis using only the faster subjects in the deliberate condition (M=1.91 s, SD=0.25) and the slower subjects in the arbitrary condition (M=1.25 s, SD=0.23). If RT length affects RP amplitudes, we would expect the RP amplitudes to be more similar between these two groups. However, though there were only half the data points, a similar pattern of results to those over the whole dataset was observed (Fig. 5A; compare to Fig. 3A). Deliberate and arbitrary decisions were still reliably different ($F(1,17)=5.22$, $p=0.03$), with significant RPs found in arbitrary (easy: $t(8)=4.57$, $p=0.0018$; hard: $t(8)=4.09$, $p=0.0035$), but not deliberate (easy: $t(8)=1.92$, $p=0.09$; hard: $t(8)=0.63$, $p=0.54$) decisions. In addition, the RPs for arbitrary decisions were not significantly different between the subjects with above-median RTs and the entire population for the easy or hard conditions (easy: $t(25)=0.14$, $p>0.5$; hard: $t(25)=0.56$, $p>0.5$). Similarly, the RPs for deliberate decisions were not significantly different between the subjects with below-median RTs and the entire population for the easy or hard conditions (easy: $t(25)=-0.34$, $p>0.5$; hard: $t(25)=0.17$, $p>0.5$). This suggest that RTs do not reliably affect C_z activation for deliberate or arbitrary decisions in our results.

Second, we regressed the difference between RPs in deliberate and arbitrary decisions (averaged over the last 500 ms before response onset) against the difference between the RTs in these two conditions for each subject (Fig. 5B). Again, if RT length affects RP amplitudes, we would expect differences between RTs in deliberate and arbitrary conditions to correlate with differences between RPs in the two conditions. But no correlation was found between the two measures ($r=0.28$, $t(16)=0.86$, $p=0.4$). We further tried regressing the RP differences on RT differences. The regression did not produce any reliable relation between RT and RP differences (regression line: $y = 0.54$ [CI -0.8, 1.89] x - 0.95 [CI -2.75, 0.85]; the R^2 was very low, at 0.05 (as expected from the r value above), and, as the confidence intervals suggest, the slope was not significantly different from 0, $F(1,16)=0.74$, $p=0.4$).

While the results of the above analyses suggested that our effects do not stem from differences between the RTs in deliberate and arbitrary decisions, the average RTs for fast deliberate subjects were still 660 ms slower than for slow arbitrary subjects. In addition, we had only half of the subjects in each condition due to the median split, raising the concern that some of our null results might have been underpowered. We also wanted to look at the effect of cross-trial variations within subjects and not just cross-subjects ones. We therefore ran a third, within-subjects analysis. We combined the two decision difficulties (easy and hard) for each decision type (arbitrary and deliberate) for greater statistical power. And then we took the faster (below-median RT) deliberate trials and slower (above-median RT) arbitrary trials for each subject separately. So, this time we had 17 subjects (again, one was removed) and better powered results. Here, fast deliberate arbitrary trials (M=1.63 s, SD=0.25) were just 230 ms slower than slow arbitrary decisions (M=1.40 s, SD=0.45), on average. This cut the difference between fast deliberate and slow arbitrary by about 2/3 from the between-subjects analysis. We then computed the RPs for just these fast deliberate and slow arbitrary trials within each subject (Fig. 5C). Visually, the pattern there is the same as the main analysis (Fig. 3A). What is more, deliberate and arbitrary decisions remained reliably different ($t(16)=3.36$, $p=0.004$). Arbitrary trials were again different from 0 ($t(16)=-4.40$, $p=0.0005$), while deliberate trials were not ($t(16)=-1.54$, $p=0.14$).

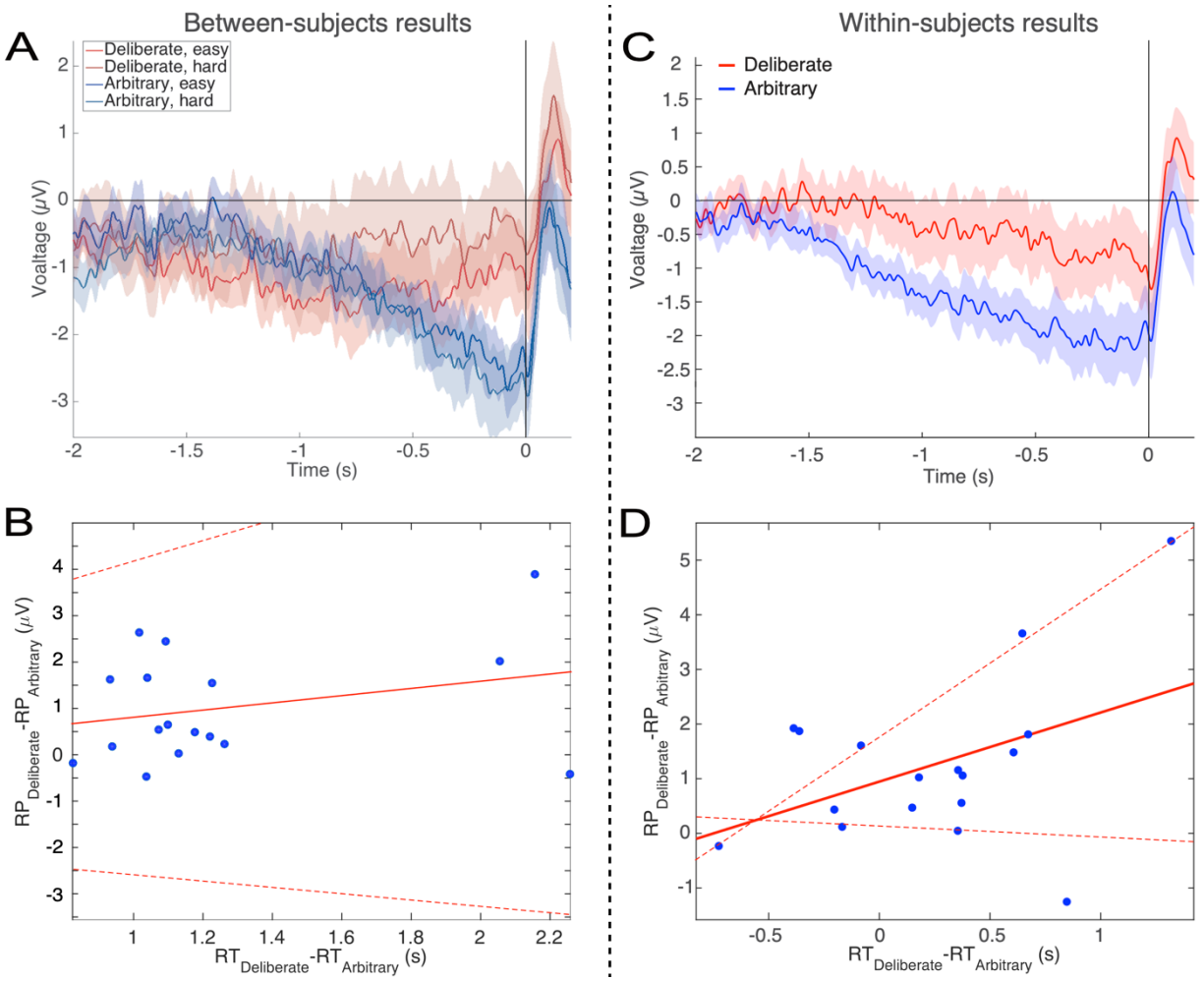

Figure 5: Relations between RTs and RPs between subjects (A&B) and within subjects (C&D). (A) The subjects with above-median RTs for arbitrary decisions (in blue) and below-median RTs for deliberate decisions (in red), show the same activity pattern that was found in the main analysis (compare Fig. 3A). **(B)** A regression of the difference between the RPs versus the difference between the RTs for deliberate and arbitrary decisions for each subject. The equation of the regression line (solid red) is y = 0.54 [CI -0.8, 1.89] x - 0.95 [CI -2.75, 0.85] (confidence intervals: dashed red lines). The R^2 is 0.05. One subject, #7, had an RT difference between deliberate and arbitrary decisions that was more than 6 interquartile ranges (IQRs) away from the median difference across all subjects. That same subject's RT difference was also more than 5 IQRs higher than the 75th percentile across all subjects. That subject was therefore designated an outlier and removed only from this regression analysis. **(C)** For each subject separately, we computed the RP using only the faster (below-median RT) deliberate trials and slower (above-median RT) arbitrary trials. The pattern is again the same as the one found for the main analysis. **(D)** We computed the same regression between the RP differences and the RT differences as in B, but this time the median split was within subjects. The equation of the regression line is y = 1.27 [CI -0.2, 2.73] x - 0.95 [CI 0.14, 1.76]. The R^2 is 0.18.

We further regressed the within-subject differences between RPs in fast deliberate and slow arbitrary decisions (defined as above) against the differences between the corresponding RTs for each subject to ascertain that such a correlation would not exist for trials that are closer together. We again found no reliable relation between the two differences (Fig. 5D; regression line: y = 1.27 [CI -0.2, 2.73] x - 0.95 [CI 0.14, 1.76]; R^2=0.18).

Yet another concern that could relate to the RT differences among the conditions is that the RP in arbitrary blocks might actually be some potential evoked by the stimuli (i.e., the

presentations of the two causes), specifically in arbitrary blocks, where the RTs are shorter (and thus stimuli-evoked effects could still affect the decision). In particular, a stimulus-evoked potential might just happen to bear some similarity to the RP when plotted locked to response onset. To test this explanation, we ran a fifth analysis, plotting the potentials in all conditions, locked to the onset of the stimulus (Fig. 6A). We also plotted the response-locked potentials across an expanded timecourse for comparison (Fig. 6B). If the RP-like shape we see in Figs. 3A and 6B is due to a stimulus-locked potential, we would expect to see the following before the 4 mean response onset times (indicated by vertical lines at 0.98 and 1.00, 2.13, and 2.52 s for arbitrary easy, arbitrary hard, deliberate easy, and deliberate hard, respectively) in the stimulus-locked plot (Fig. 6A): Consistent potentials, which precede the mean response times, that would further be of a similar shape and magnitude to the RPs found in the decision-locked analysis in the arbitrary condition (though potentially more smeared for stimulus locking). We thus calculated a stimulus-locked version of our ERPs, using the same baseline (Fig. 6A). As the comparison between Fig. 6A and 6B clearly shows, no such consistent potentials were found before the 4 response times, nor were these potentials similar to the RP in either shape or magnitude (their magnitudes are at the most around 1μV, while the RP magnitudes we found are around 2.5 μV; Figs. 3A, 6B). This analysis thus suggests that it is unlikely that a stimulus-locked potential drives the RP we found.

Notably, the stimulus-locked alignment did imply that the arbitrary easy condition evoked a stronger activity in roughly the last 0.5 s before stimulus onset. However, this prestimulus activity cannot explain the response-locked RP, as it was found *only in arbitrary easy trials* and not in arbitrary hard trials. At the same time, the response-locked RP did not differ between these conditions. What is more, easy and hard trials were randomly interspersed within deliberate and arbitrary blocks, and the subject discovered the trial difficulty only at stimulus onset. Thus, there could not have been differential preparatory activity that varies with decision difficulty. This divergence in one condition only is accordingly not likely to reflect any preparatory RP activity.

One more concern is that the differences in RTs may affect the results in the following manner: Because the main baseline period we used thus far was 1 to 0.5 s before stimulus onset, the duration from the baseline to the decision varied widely between the conditions. To make sure this difference in temporal distance between the baseline period and the response to which the ERPs were locked did not drive our results, we recalculated the potentials for all conditions with a *response-locked* baseline of -1 to -0.5 s (Fig. 6C; the same baseline we used for the Bayesian analysis above). The rationale behind this choice of baseline was to have the time that elapsed from baseline to response onset be the same across all conditions. As is evident in Fig. 6C, the results for this new baseline were very similar to those for the stimulus-locked baseline we used before. Focusing again on the -0.5 to 0 s range before response onset for our statistical tests, we found a clear RP in arbitrary decisions, yet RP amplitude was not significantly different from 0 in deliberate decisions (Fig. 6C; ANOVA $F(1,17)=12.09$, $p=0.003$ for the main effect of decision type; in t-tests against 0, corrected for multiple comparisons, an effect was only found for arbitrary decisions (hard: $t(17)=4.13$, $p=0.0007$; easy: $t(17)=4.72$, $p=0.0002$) and not for deliberate ones (hard: $t(17)=0.38$, $p>0.5$; easy: $t(17)=1.13$, $p=0.27$). This supports the notion that the choice of baseline does not strongly affect our main results. Taken together, the results of the six analyses above provide strong evidence against the claim that the differences in RPs stem from or are affected by the differences in RTs between the conditions.

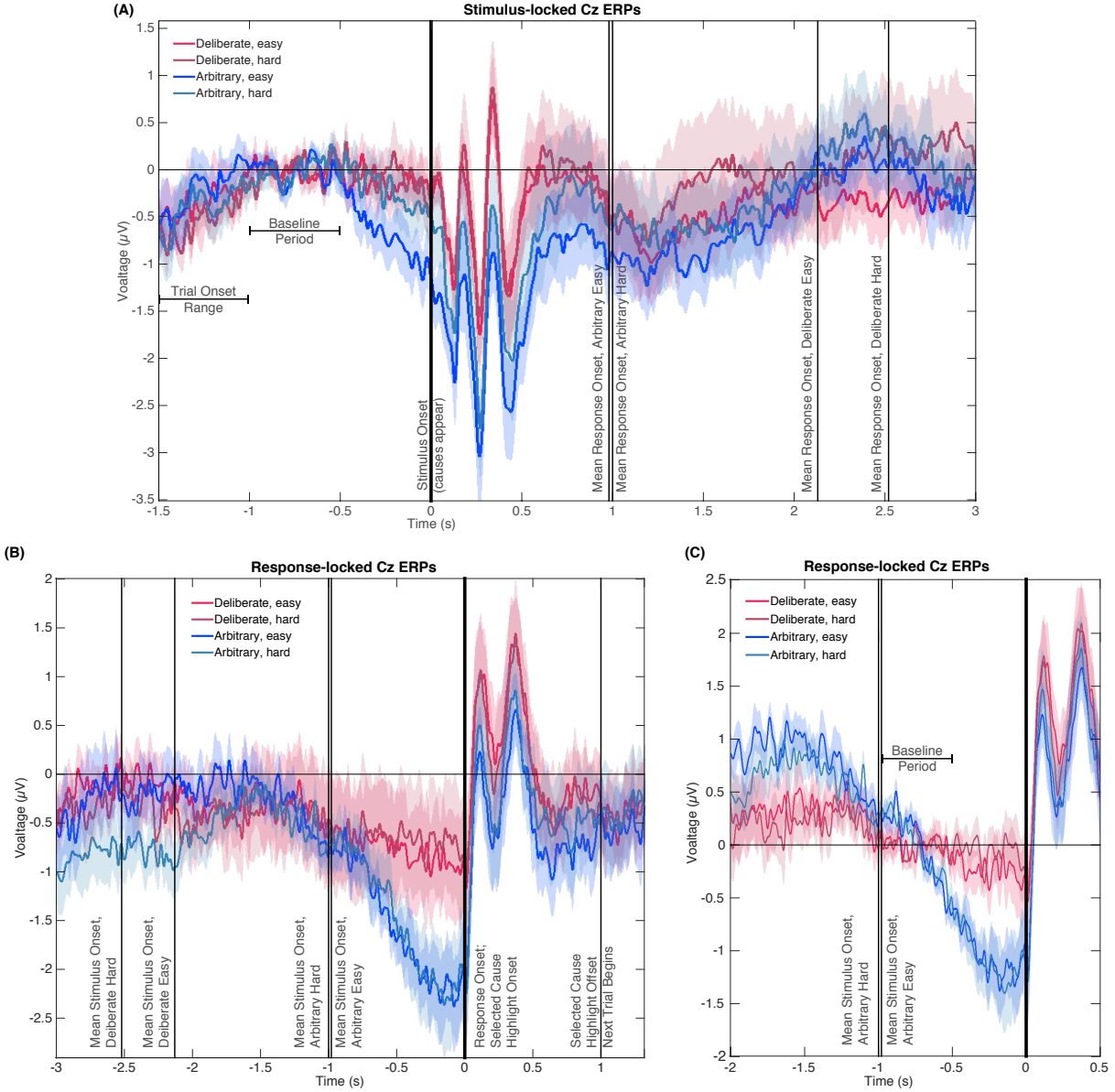

Figure 6: Stimulus- and response-locked Cz-electrode ERPs with different baselines and overlaid events. (A) Stimulus-locked waveforms including the trial onset range, baseline period, and mean reaction times for all four experimental conditions. **(B)** Response-locked waveforms with mean stimulus onsets for all four conditions as well as the offset of the highlighting of the selected cause and the start of the next trial. **(C)** Same potentials and timeline as Fig. 3A, but with a *response-locked* baseline of -1 to -0.5 s—the same baseline used for our Bayesian analysis.

Eye movements do not affect the results

Though ICA was used to remove blink artifacts and saccades (see Methods), we wanted to make sure our results do not stem from differential eye movement patterns between the conditions. We therefore computed a saccade-count metric (SC; see Methods) for each trial for all subjects. Focusing again on the last 500 ms before response onset, we computed mean (±s.e.m.) SC values of 1.65±0.07 and 1.67±0.06 saccades for easy and hard deliberate decisions, respectively, and 1.69±0.07 and 1.73±0.07 saccades for easy and hard arbitrary decisions, respectively. We found no reliable differences between the number of saccades during deliberate and arbitrary trials ($F(1,17)=2.56$, $p=0.13$ for main effect of decision type).

We further investigated potential effects of saccades by running a median-split analysis—dividing the trials for each subject into two groups based on their SC score: lower and higher than the median, for deliberate and arbitrary trials, respectively. We then ran the same analysis using only the trials with more saccades in the deliberate condition (SC was 2.02±0.07 and 2.04±0.07 for easy and hard, respectively) and those with less saccades for the arbitrary condition (SC was 1.33±0.07 and 1.31±0.08 for easy and hard, respectively). If the number of saccades affects RP amplitudes, we would expect that the differences in RPs between arbitrary and deliberate trials will diminish, or even reverse (as now we had more saccades in the deliberate condition). However, though there were only half the data points for each subject in each condition, a similar pattern of results to those over the whole dataset was observed: Deliberate and arbitrary decisions were still reliably different within the median-split RPs ($F(1,17)=16.70$, $p<0.001$), with significant RPs found in arbitrary (easy: $t(17)=4.79$, $p=0.002$; hard: $t(17)=5.77$, $p<0.001$), but not deliberate (easy: $t(17)=0.90$, $p=0.38$; hard: $t(17)=0.30$, $p>0.5$) decisions. In addition, we compared the RP data across all the trials with the median-split RP data above. No significant differences were found for arbitrary decisions (easy: $t(17)=1.02$, $p=0.32$; hard: $t(17)=0.75$, $p=0.46$) or for deliberate decisions (easy: $t(17)=1.63$, $p=0.12$; hard: $t(17)=1.47$, $p=0.16$). Taken together, the analyses above provide strong evidence against the involvement of eye movements in our results.

Testing alternative explanations

We took a closer look at subjects' behavior in the easy arbitrary condition, where some subjects had a consistency score that was further above 0.5 (chance) than others. It seems like those subjects had a greater difficulty ignoring their preferences, despite the instructions to do so. We therefore wanted to test to what extent the RP of those subjects was similar to the RPs of the other subjects. Focusing on the 8 subjects that had a consistency score above 0.55 ($M=0.59$, $SD=0.03$) and comparing their RPs to those of the 10 other subjects (consistency $M=0.50$, $SD=0.06$) in easy arbitrary trials, we found no reliable differences ($t(16)=0.94$, $p=0.36$). This is not surprising, as the mean consistency score of these subjects—though higher than chance—was still far below their consistency score for easy deliberate decisions ($M=0.99$, $SD=0.02$).

High-pass filter cutoff frequency does not affect the results

Finally, another alternative explanation might rely on our selection of high-pass filter cutoff frequency, which was 0.1 Hz. Though this frequency was used in some studies of the RP (e.g., Lew, Chavarriaga, Silvoni, & Millán, 2012; MacKinnon, Allen, Shiratori, & Rogers, 2013), others opted for lower cutoff frequencies (e.g., Haggard & Eimer, 1999). Arguably, a higher cutoff frequency for the high-pass filter might reduce the chances to find the RP, which is a low-frequency component. And this might have affected the RP for deliberate decisions more than the RP for arbitrary ones, given the slower RTs there. To examine this possible confound, we reanalyzed the data using a 0.01 high-pass filter. This reduced the number of usable trials for each subject, as it allowed lower-frequency trends to remain in the data. Given that our focus was on arbitrary vs. deliberate decisions (with decision difficulty serving mostly to validate the manipulation), we collapsed the trials across decision difficulty, and only tested RP amplitudes in arbitrary vs. deliberate decisions against each other and against zero. In line with our original results, a difference was found between RP amplitude in the two conditions ($t(13)=2.29$, $p=0.039$), with RP in the arbitrary condition differing from zero ($t(13)=-5.71$, $p<0.0001$), as opposed to the deliberate condition, where it did not ($t(13)=-0.76$, $p=0.462$). This provides evidence against the claim that our results are due to our choice of high-pass filter.

EEG Results: Lateralized Readiness Potential (LRP)

The LRP, which reflects activation processes within the motor cortex for action preparation after action selection (Eimer, 1998; Masaki, Wild-wall, Sangals, & Sommer, 2004), was measured by subtracting the difference potentials (C3-C4) in right-hand response trials from this difference in left-hand responses trials and averaging the activity over the same time window (Eimer, 1998; Haggard & Eimer, 1999). In this purely motor component, no difference was found between the two decision types and conclusive evidence against an effect of decision type was further found (Fig. 7; all Fs<0.35; BF=0.299). Our analysis of EOG channels suggests that some of that LRP might be driven by eye movements (we repeated the LRP computation on the EOG channels instead of C3 and C4). However, the shape of the eye-movement-induced LRP is very different from the LRP we calculated from C3 and C4. Also, the differences that we found between conditions in the EOG LRP are not reflected in the C3/C4 LRP. So, while our LRP might be boosted by eye movements, it is not strictly driven by these eye movements.

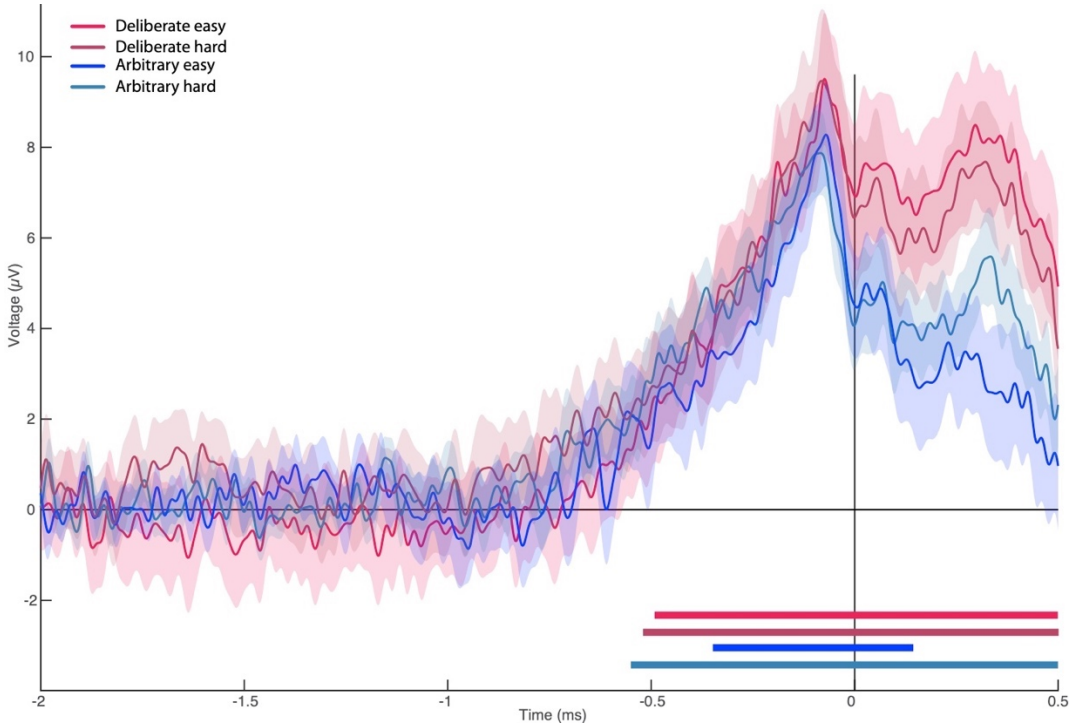

Figure 7: Lateralized readiness potential. The lateralized readiness potential (LRP) for deliberate and arbitrary, easy and hard decisions. No difference was found between the conditions (ANOVA all Fs<0.35). Temporal clusters where the activity for each condition was independently found to be significantly different from 0 are designated by horizontal thick lines at the bottom of the figure (with their colors matching the legend).

Modeling

The main finding of this study—the absent (or at least strongly diminished) RP in deliberate decisions, suggesting different neural underpinnings of arbitrary and deliberate decisions—is in line with a recent study using a drift-diffusion model (DDM) to investigate the RP (Schurger et al., 2012). There, the RP was modeled as an accumulation of white noise (that results in autocorrelated noise) up to a hard threshold. When activity crosses that threshold, it designates decision-onset leading to movement. The model focuses on the activity leading up to the threshold crossing, when that activity is time-locked to the onset of the threshold crossing (corresponding to movement-locked epochs in EEG). Averaging across many threshold crossings, this autocorrelation activity resembles an RP (Schurger et al., 2012). Hence, according to this model, the threshold crossing leading to response onset is largely determined by spontaneous, subthreshold, stochastic fluctuations of the neural activity. This interpretation

of the RP challenges its traditional understanding as stemming from specific, unconscious preparation for, or ballistic-like initiation of, movement (Shibasaki & Hallett, 2006). Instead, Schurger and colleagues claimed, the RP is not a cognitive component of motor preparation; it is an artifact of accumulating autocorrelated noise to a hard threshold and then looking at signals only around threshold crossings.

We wanted to investigate whether our results could be accommodated within the general framework of the Schurger model, though with the deliberate and arbitrary decisions mediated by two different mechanisms. The first mechanism is involved in value assessment and drives deliberate decisions. It may be subserved by brain regions like the Ventromedial Prefrontal Cortex; VMPFC, (Ramnani & Owen, 2004; Wallis, 2007). But, for the sake of the model, we will remain agnostic about the exact location associated with deliberate decisions and refer to this region as *Region X*. A second mechanism, possibly at the (pre-)SMA, was held to generate arbitrary decisions driven by random, noise fluctuations.

Accordingly, we expanded the model developed by Schurger et al. (2012) in two manners. First, we defined two DDM processes—one devoted to value-assessment (in Region X) and the other to noise-generation (in SMA; see Fig. 8A and Methods). Both of them were run during both decision types, yet the former determined the result of deliberate trials, and the latter determined the results of arbitrary trials. Second, Schurger and colleagues modeled only *when* subjects would move and not *what* (which hand) subjects would move. We wanted to account for the fact that, in our experiment, subjects not only decided when to move, but also what to move (either to indicate which NPO they prefer in the deliberate condition, or to generate a meaningless right/left movement in the arbitrary condition). We modeled this by defining two types of movement. One was moving the hand corresponding to the location of the NPO that was rated higher in the first, rating part of the experiment (the *congruent* option; see Methods). The other was moving the hand corresponding to the location of the lower-rated NPO (the *incongruent* option). We used the race-to-threshold framework to model the decision process between this pair of leaky, stochastic accumulators, or DDMs. One DDM simulated the process that leads to selecting the congruent option, and the other simulated the process that leads to selecting the incongruent option (see again Fig. 8A). (We preferred the race-to-threshold model over a classic DDM with two opposing thresholds because we think it is biologically more plausible (de Lafuente, Jazayeri, & Shadlen, 2015) and because it is easier to see how a ramp-up-like RP might be generated from such a model without requiring a vertical flip of the activity accumulating toward one of the thresholds in each model run.) Hence, in each model run, the two DDMs in Region X and the two in the SMA ran in parallel; the first one to cross the threshold (in Region X for deliberate decisions and in the SMA for arbitrary ones) determined decision onset and outcome. Thus, if the DDM corresponding to the congruent (incongruent) option reached the threshold first, the trial ended with selecting the congruent (incongruent) option. For deliberate decisions, the congruent cause had a higher value than the incongruent cause and, accordingly, the DDM associated with the congruent option had a higher drift rate than that of the DDM associated with the incongruent option. For arbitrary decisions, the values of the decision alternatives mattered little and this was reflected in the small differences among the drift rates and in other model parameters (Table 1).

Therefore, within this framework, C_z-electrode activity (above the SMA) should mainly reflect the SMA component (Note that we suggest that noise generation might be a key function of the SMA and other brain regions underneath the C_z electrode, *at least during this specific task*. When subjects make arbitrary decisions, these might be based on some symmetry-breaking mechanism, which is driven by random fluctuations that are here simulated as noise. Thus, we neither claim nor think that noise generation is the main purpose or function of these brain regions in general.) And so, finding that the model-predicted EEG activity resembles the actual

EEG pattern we found would imply that our findings are compatible with an account by which the RP represents an artifactual accumulation of stochastic, autocorrelated noise, rather than indexing a genuine marker of an unconscious decision ballistically leading to action.

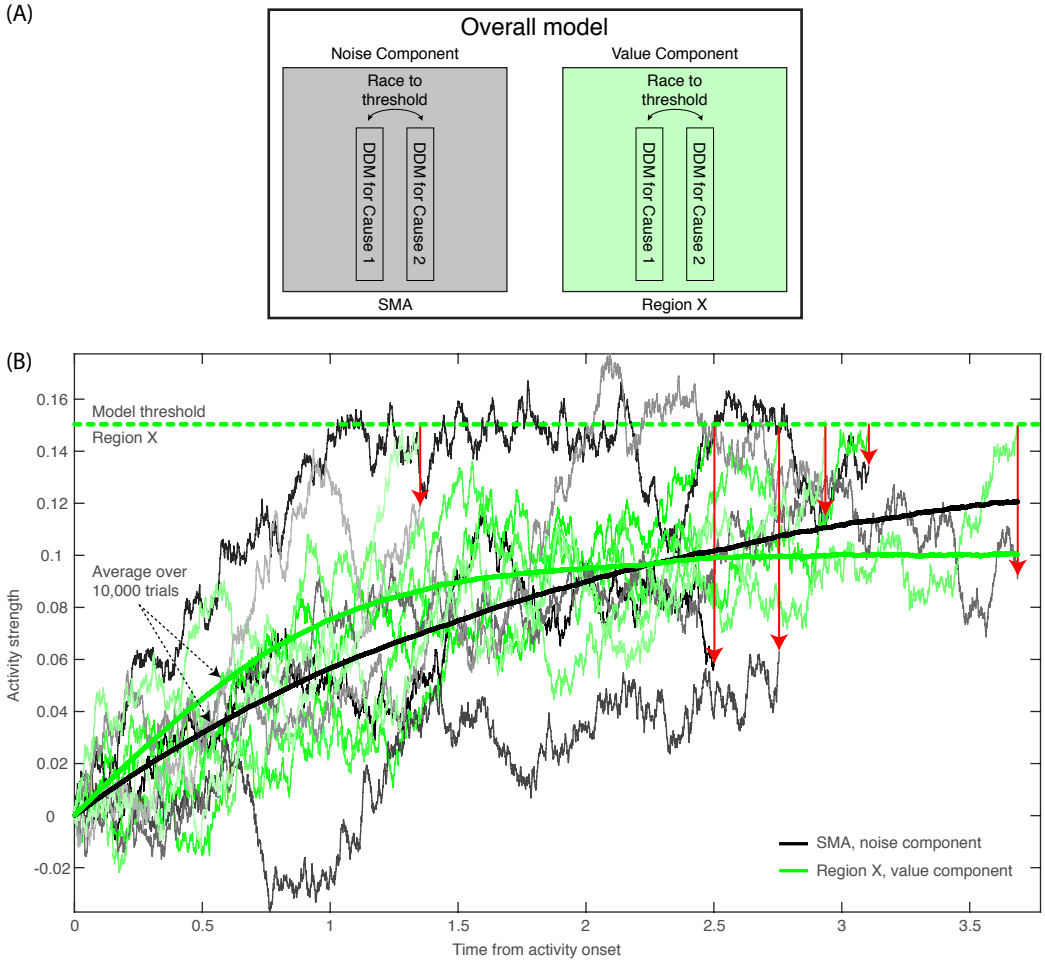

Figure 8: Model description and model runs in the SMA and in Region X. (A) A block diagram of the model, with its noise (SMA) and value (Region X) components, each instantiated as a race to threshold between a pair of DDMs (or causes—one congruent with the ratings in the first part of the experiment, the other incongruent). **(B)** A few runs of the model in the deliberate condition, in Region X (green colors), depicting the DDM for the congruent option. As is apparent, the DDM stops when the value-based component reaches threshold. Red arrows point from the Region X DDM trace at threshold to the corresponding time in the trace of the SMA (black and gray colors). The SMA traces integrate without a threshold (as the decision outcome is solely determined by the value component in Region X). The thick green and black lines depict average Region X and SMA activity, respectively, over 10,000 model runs locked to *stimulus onset*. Hence, we do not expect to find an RP in either brain region. (For decision-locked activity see Fig. 9B).

The critical prediction of our model for our purposes relates to what happens during deliberate decisions in the SMA (C_z electrode). For ease of explanation, and because decision difficulty had no consistent effect on the EEG data, we focus the discussion below on easy decisions (though the same holds for hard decisions). According to our model, the race-to-threshold pair of DDMs that would determine deliberate decisions and trigger the ensuing action is the value-assessment one in Region X. Hence, when the first DDM of the Region X pair would reach the threshold, the decision would be made and movement would ensue. At the same time and in contrast, the SMA pair would not integrate toward a decision (Fig. 8B). We modeled this by not including any decision threshold in the SMA in deliberate decisions (i.e., the threshold was

set to infinity, letting the DDM accumulate forever). (The corresponding magnitudes of the drift-rate and other parameters are detailed in the Methods and Table 1.) So, when Region X activity reaches the threshold, the SMA (supposedly recorded using electrode C_z) will have happened to accumulate to some random level (Fig. 8B). This entails that, when we align such SMA activity to decision (or movement) onset, we will find just a simple, weak linear trend in the SMA. This trend is the one depicted in red in Fig. 9B for the deliberate easy and hard conditions (here model activity was flipped vertically—from increasing above the *x* axis to decreasing below it—as in Schurger et al., 2012). In arbitrary decisions, on the other hand, the SMA pair, from which we record, is also the one that determines the outcome. Hence, motion ensues whenever one of the DDMs crosses the threshold. Thus, when its activity is inspected with respect to movement onset, it forms the RP-like shape of Fig. 9B (in blue), in line with the model by Schurger and colleagues (2012).

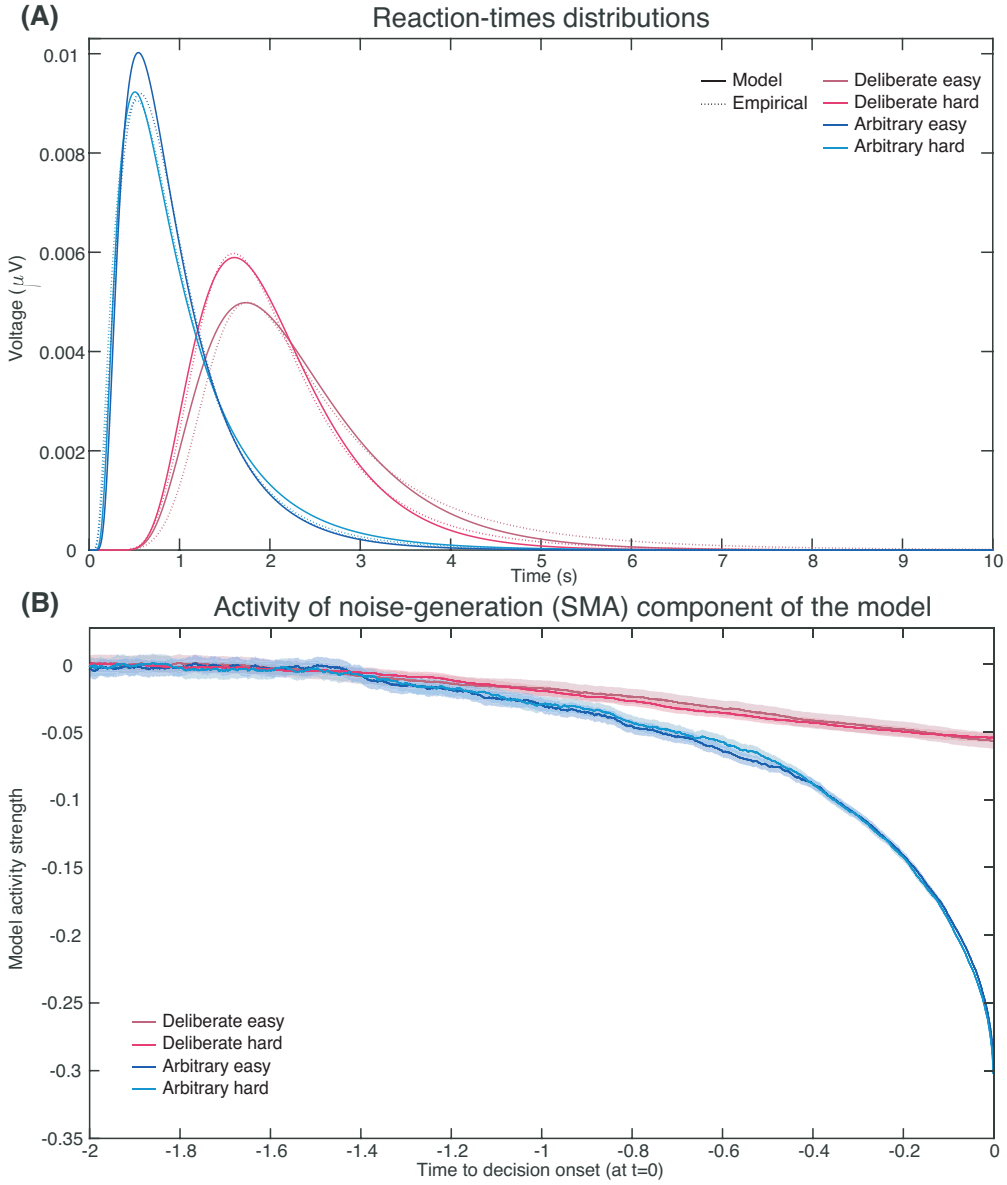

Figure 9: Empirical and model RTs and model prediction for Cz activity. (A) The model (solid) and empirical (dashed) distributions of subjects' RTs (fitted with a gamma function to the cumulative distribution, see Methods) across the four decision types. **(B)** The model's prediction for the ERP activity in its noise component (Fig. 8A) in the SMA (electrode C_z), locked to decision onset (at t = 0 s), across all four decision types.

Akin to the Schurger model, we simultaneously fit our DDMs to the complete distribution of our empirical reaction-times (RTs; Fig. 9A) and to the empirical consistency scores (the proportions of congruent decisions; see Methods). The models' fit to the empirical RTs and consistencies were good (RT & consistency errors for deliberate easy and hard were 0.054 & 0.004 and 0.108 & 0.008 respectively; for arbitrary easy and hard they were 0.053 & 0.002, and 0.055 & 0.003, respectively; Fig. 9A) The averages of these empirical RT distributions were 2.13, 2.52, 0.98 and 1.00 s and the empirical consistency scores were 0.99, 0.83, 0.54 and 0.49 for deliberate easy, deliberate hard, arbitrary easy, and arbitrary hard, respectively.

Once we found the models with the best fit (see Methods for details), we used those to predict the resulting ERP patterns in the SMA—i.e., those we would expect to record in C_z. The ERP that the model predicted was the mean between the congruent and incongruent activities, as both would be picked up by C_z. The result was an RP-like activity for arbitrary decisions, but only a very slight slope for deliberate decisions (Fig. 9B; both activities were flipped vertically, as in Schurger's model). This was in line with our empirical results (compare Fig. 3A).

Note that the Schurger model aims to account for neural activity leading up to the decision to move, but no further (Schurger et al., 2012). Similarly, we expect our DDM to fit Cz neural data only up to around -0.1 to -0.2 s (100 to 200 ms before empirical response onset). What is more, we model Region X activity here using a DDM for simplicity. But we would get a similar result—SMA RP-like activity for arbitrary decision and only a trend for deliberate ones—for other models of decision making, as long as the onset of deliberate decisions would remain statistically independent from threshold crossing in the DDMs of the SMA. Further, we make no claims that ours is the only, or even optimal, model that explains our results. Rather, by specifically extending the Schurger model, our goal was to show how that interpretation of the RP could also be applied to our more-complex paradigm. (We refer the reader to work by Schurger and colleagues (Schurger, 2018; Schurger et al., 2012) for more detailed discussions about the model, its comparison to other models, and the relation to conscious-decision onset).

Discussion

Since the publication of Libet's seminal work—which claimed that neural precursors of action, in the form of the RP, precede subjects' reports of having consciously decided to act (Libet et al., 1983)—a vigorous discussion has been raging among neuroscientists, philosophers, and other scholars about the meaning of the RP for the debate on free will (recent collections include (Mele, 2015; Pockett, Banks, & Gallagher, 2009; Sinnott-Armstrong & Nadel, 2011)). Some claim that the RP is simply a marker for an unconscious decision to act and thus its onset at least reflects an intention to move and ballistically leads to movement (Libet et al., 1983; Soon et al., 2008). Under this interpretation, the onset of the RP before the reported onset of the conscious decision to move effectively removes conscious will from the causal chain leading to action (Haggard, 2005, 2008; Libet, 1985; Wegner, 2002). Others do not agree (Breitmeyer, 1985; Mele, 2009; Nahmias, Shepard, & Reuter, 2014; Roskies, 2010). But, regardless, the RP lies at the heart of much of this debate (Kornhuber & Deecke, 1990; Libet et al., 1983).

Notably, the RP and similar findings showing neural activations preceding the conscious decision to act have typically been based on arbitrary decisions (Haggard & Eimer, 1999; Lau et al., 2004; Libet, 1985; Libet et al., 1983; Sirigu et al., 2004; Soon et al., 2008; Soon et al., 2013). This, among other reasons, rested on the notion that for an action to be completely free, it should not be determined in any way by external factors (Libet, 1985)—which is the case for arbitrary, but not deliberate, decisions (for the latter, each decision alternative is associated with a value, and the values the of alternatives typically guide one's decision). But this notion

641 of freedom faces several obstacles. First, most discussions of free will focus on deliberate
642 decisions, asking when and whether these are free (Frankfurt, 1971; Hobbes, 1994; Wolf,
643 1990). This might be because everyday decisions to which we associate freedom of will—like
644 choosing a more expensive but more environmentally friendly car, helping a friend instead of
645 studying more for a test, donating to charity, and so on—are generally deliberate, in the sense
646 of being reasoned, purposeful, and bearing consequences (although see Deutschländer, Pauen,
647 and Haynes (2017)). In particular, the free will debate is often considered in the context of
648 moral responsibility (e.g., was the decision to harm another person free or not) (Fischer, 1999;
649 Haggard, 2008; Maoz & Yaffe, 2015; Roskies, 2012; Sinnott-Armstrong, 2014; Strawson,
650 1994), and free will is even sometimes defined as the capacity that allows one to be morally
651 responsible (Mele, 2006, 2009). In contrast, it seems meaningless to assign blame or praise to
652 arbitrary decisions. Thus, though the scientific operationalization of free will has typically
653 focused on arbitrary decisions, the common interpretations of these studies—in neuroscience
654 and across the free will debate—have often alluded to deliberate decisions. This is based on the
655 implicit assumption that the RP studies capture the same, or a sufficiently similar, process as
656 that which occurs in deliberate decisions. And so, inferences from RP results on arbitrary
657 decisions can be made to deliberate decisions.

658 However, here we show that this assumption may not be justified, as the neural precursors of
659 arbitrary decisions, at least in the form of the RP, do not generalize to meaningful, deliberate
660 decisions (Breitmeyer, 1985; Roskies, 2010). For *arbitrary decisions*, we replicated the
661 waveform found in previous studies, where the subjects moved endogenously, at a time of their
662 choice with no external cues (Shibasaki & Hallett, 2006). The RP was also similarly recorded
663 in the C_z electrode and with a typical scalp topography. However, the RP was altogether
664 absent—or at least substantially diminished—for *deliberate decisions*; it showed neither the
665 expected slope over time nor the expected scalp topography.

666 Null-hypothesis significance testing (NHST) suggested that the null hypothesis—i.e., that there
667 is no RP—can be rejected for arbitrary decisions but cannot be rejected for deliberate ones. A
668 cluster-based nonparametric permutation analysis—to locate temporal windows where EEG
669 activity is reliably different from 0—found prolonged activity of this type during the 1.2 s
670 before movement onset for easy and hard arbitrary decisions, but no such activity was found
671 for either type of deliberate decisions. A Bayesian analysis found clear evidence for an RP in
672 arbitrary decisions and an inconclusive trend toward no RP in deliberate decisions. Changing
673 the baseline to make it equally distant from arbitrary and deliberate decisions did provide
674 evidence for the absence of an RP in deliberate decisions, while still finding clear evidence for
675 an RP in arbitrary decisions. Further, baseline-invariant trend analysis showed that there is no
676 trend during the RP time window for deliberate decisions (here Bayesian analysis suggested
677 moderate to strong evidence against a trend) while there existed a reliable trend for arbitrary
678 decisions (and extremely strong evidence for an effect in the Bayesian framework). Thus,
679 taken together, there is overwhelming evidence for an RP in arbitrary decisions (in all six
680 different analyses that we conducted—NHST and Bayesian). But, in contrast, we found no
681 evidence for the existence of an RP in deliberate decisions (in all six analyses) and, at the same
682 time, there was evidence against RP existence in such decisions (in five of the six analyses,
683 with the single, remaining analysis providing only inconclusive evidence for an absence of an
684 RP). Therefore, when the above analyses are taken together, we think that the most plausible
685 interpretation of our results is that the RP is absent in deliberate decisions.

686 Nevertheless, even if one takes our results to imply that the RP is only strongly diminished in
687 deliberate compared to arbitrary decisions, this provides evidence against drawing strong
688 conclusions regarding the free-will debate from the Libet and follow-up results. The
689 assumption in the Libet-type studies is that the RP simply reflects motor preparation (Haggard,

690 2019; Haggard & Eimer, 1999; Libet, 1985; Libet et al., 1983; Shibasaki & Hallett, 2006) and
691 in that it lives up to its name. However, in our paradigm, both the sensory inputs and the motor
692 outputs were the same between arbitrary and deliberate trials. Thus, motor preparation is
693 expected in both conditions and the RP should have been found in both. Accordingly, any
694 consistent difference in the RP between the decision types suggests—to the very least—that it
695 is a more complex signal than Libet and colleagues had assumed. For one, it shows that it is
696 influenced by cognitive state and that it cannot be regarded as a genuine index of a voluntary
697 decision, be it arbitrary or deliberate. Further, our model predicted an RP in arbitrary decisions
698 but only a slow trend in movement-locked ERP during deliberate decisions that is in the
699 direction as the RP, *but is not an RP*. Hence, a signal that resembles a strongly diminished RP
700 but is in fact just slow trend in the same direction is congruent with our model.

701 Interestingly, while the RP was present in arbitrary decisions but absent in deliberate ones, the
702 LRP—a long-standing, more-motor ERP component, which began much later than the RP——
703 was indistinguishable between the different decision types. This provides evidence that, at the
704 motor level, the neural representation of the deliberate and arbitrary decisions that our subjects
705 made may have been indistinguishable, as was our intention when designing the task.

706 Our findings and the model thus suggest that two different neural mechanisms may be involved
707 in arbitrary and deliberate decisions. Earlier literature demonstrated that deliberate, reasoned
708 decision-making—which was mostly studied in the field of neuroeconomics (Kable &
709 Glimcher, 2009) or using perceptual decisions (Gold & Shadlen, 2007)—elicited activity in the
710 prefrontal cortex (PFC; mainly the dorsolateral (DLPFC) part (Sanfey, Rilling, Aronson,
711 Nystrom, & Cohen, 2003; Wallis & Miller, 2003) and ventromedial (VMPFC)
712 part/orbitofrontal cortex (OFC) (Ramnani & Owen, 2004; Wallis, 2007) and the anterior
713 cingulate cortex (ACC) (Bush, Luu, & Posner, 2000; Carter et al., 1998). Arbitrary,
714 meaningless decisions, in contrast, were mainly probed using variants of the Libet paradigm,
715 showing activations in the Supplementary Motor Area (SMA), alongside other frontal areas
716 like the medial frontal cortex (Brass & Haggard, 2008; Krieghoff, Waszak, Prinz, & Brass,
717 2011) or the frontopolar cortex, as well as the posterior cingulate cortex (Fried et al., 2011;
718 Soon et al., 2008) (though see Hughes, Schütz-Bosbach, and Waszak (2011), which suggests
719 that a common mechanism may underlie both decision types). Possibly then, arbitrary and
720 deliberate decisions may differ not only with respect to the RP, but be subserved by different
721 underlying neural circuits, which makes generalization from one class of decisions to the other
722 more difficult. Deliberate decisions are associated with more lateralized and central neural
723 activity while arbitrary ones are associated with more medial and frontal ones. This appears to
724 align with the different brain regions associated with the two decision types above, as also
725 evidenced by the differences we found between the scalp distributions of arbitrary and
726 deliberate decisions (Fig. 3A). Further studies are needed to explore this potential divergence
727 in the neural regions between the two decision types.

728 Therefore, at the very least, our results support the claim that the previous findings regarding
729 the RP should be confined to arbitrary decisions and do not generalize to deliberate ones. What
730 is more, if the ubiquitous RP does not generalize, it cannot simply be assumed that other
731 markers will. Hence, such differences clearly challenge the generalizability of previous studies
732 focusing on arbitrary decisions to deliberate ones, regardless of whether they were based on the
733 RP or not. In other words, our results put the onus on attempts to generalize markers of
734 upcoming action from arbitrary to deliberate decisions; it is on them now to demonstrate that
735 those markers do indeed generalize. And, given the extent of the claims made and conclusions
736 derived based on the RP in the neuroscience of free will (see again Mele, 2015; Pockett,
737 Banks, & Gallagher, 2009; Sinnott-Armstrong & Nadel, 2011), our findings call for a re-
738 examination of some of the basic tenents of the field.

It should be noted that our study does not provide positive evidence that consciousness is more involved in deliberate decisions than in arbitrary ones; such a strong claim requires further evidence, perhaps from future research. But our results highlight the need for such research. Under some (strong) assumptions, the onset of the RP before the onset of reported intentions to move may point to there being no role for consciousness in arbitrary decisions. But, even if such conclusions can be reached, they cannot be safely extended to deliberate decisions.

To be clear, and following the above, we do not claim that the RP captures all unconscious processes that precede conscious awareness. However, some have suggested that the RP represents unconscious motor-preparatory activity before any kind of decision (e.g., Libet, 1985). But our results provide evidence against that claim, as we do not find an RP before deliberate decisions, which also entail motor preparation. What is more, in deliberate decisions in particular, it is likely that there are neural precursors of upcoming actions—possibly involving the above neural circuits as well as circuits that represents values—which are unrelated to the RP (the lack of such precursors is not merely implausible; it implies dualism: Mudrik & Maoz, 2014; Wood, 1985).

Note also that we did not attempt to clock subjects' conscious decision to move. Rather, we instructed them to hold their hands above the relevant keyboard keys and press their selected key as soon as they made up their mind. This was to keep the decisions in this task more ecological and because we think that the key method of measuring decision onset (using some type of clock to measure Libet's W-time) is highly problematic (see Methods). But, even more importantly, clock monitoring was demonstrated to have an effect on RP size (Miller et al., 2011), so it could potentially confound our results (Maoz et al., 2015).

Some might also claim that unconscious decision-making could explain our results, suggesting that in arbitrary decisions subjects engage in unconscious deliberation or in actively inhibiting their urge to follow their preference as well as in free choice, while in deliberate decisions only deliberation is required. But this interpretation is unlikely because the longer RTs in deliberate decisions suggest, if anything, that more complex mental processes (conscious or unconscious) took place before deliberate and not arbitrary decisions. In addition, these interpretations should impede our chances of finding the RP in arbitrary trials (as the design diverges from the original Libet task), yet the RP was present, rendering them less plausible.

Aside from highlighting the neural differences between arbitrary and deliberate decisions, this study also challenges a common interpretation of the function of the RP. If the RP is not present before deliberate action, it does not seem to be a necessary link in the general causal chain leading to action. Schurger et al. (2012) suggested that the RP reflects the accumulation of autocorrelated, stochastic fluctuations in neural activity that lead to action, following a threshold crossing, when humans arbitrarily decide to move. According to that model, the shape of the RP results from the manner in which it is computed from autocorrelated EEG: averaged over trials that are locked to response onset (that directly follows the threshold crossing). Our results and our model are in line with that interpretation and expand it to decisions that include both when and which hand to move. They suggest that the RP represents the accumulation of noisy, random fluctuations that drive arbitrary decisions, Whereas deliberate decisions are mainly driven by the values associated with the decision alternatives (Maoz et al., 2013).

Our drift-diffusion model was based on the assumption that every decision can be driven by a component reflecting the values of the decision alternatives (i.e., subjects' support for the two NPOs we presented) or by another component representing noise—random fluctuations in neural activity. The value component plays little to no role in arbitrary decisions, so action

786 selection and timing depend on when the accumulation of noise crosses the decision threshold
787 for the congruent and incongruent decision alternatives. In deliberate decisions, in contrast, the
788 value component drives the decisions, while the noise component plays little to no role. Thus,
789 in arbitrary decisions, action onset closely tracks threshold crossings of the noise (or SMA)
790 component. But, in deliberate decisions, the noise component reaches a random level and is
791 then stopped; so, the value component drives the decision. Hence, as we record from the SMA
792 (the noise component)—locking the ERP to response onset and averaging over trials to obtain
793 the RP leads to a slight slope for deliberate decisions but to the expected RP shape in arbitrary
794 decisions. This provides evidence that the RP does not reflect subconscious movement
795 preparation. Rather, it is induced by threshold crossing of stochastic fluctuations in arbitrary
796 decisions, which do not drive deliberate decisions; accordingly, the RP is not found in
797 deliberate decisions. Our model therefore challenges RP-based claims against free will in both
798 arbitrary and deliberate decisions. Further studies of the causal role of consciousness in
799 deliberate versus arbitrary decisions are required to test this claim.

800 Nevertheless, two possible, alternative explanations of our results can be raised. First, one
801 could claim that—in the deliberate condition only—the NPO names act as a cue, thereby
802 creating a stimulus-response mapping and in that turning what we term internal, deliberate
803 decisions into no more than simple responses to external stimuli. Under this account, if the
804 preferred NPO is on the right, it is immediately interpreted as "press right". It would therefore
805 follow that subjects are actually not making decisions in deliberate trials, which in turn is
806 reflected by the absence of the RP in those trials. However, the reaction time and consistency
807 results that we obtained for deliberate trials provide evidence against this interpretation. We
808 found longer reaction times for hard-deliberate decisions than for easy-deliberate ones (2.52
809 versus 2.13 s, on average, respectively; Fig. 2 left) and higher consistencies with the initial
810 ratings for easy-deliberate decisions than for hard-deliberate decisions (0.99 versus 0.83, on
811 average, respectively; Fig. 2 right). If the NPO names acted as mere cues, we would have
812 expected no differences between reaction times or consistencies for easy- and hard-deliberate
813 decisions. In addition, there were 50 different causes in the first part of the experiment. So, it is
814 highly unlikely that subjects could memorize all 1225 pairwise preferences among these causes
815 and simply transform any decision between a pair of causes into a stimulus instructing to press
816 left or right.

817 Another alternative interpretation of our results is that subjects engage in (unconscious)
818 deliberation also during arbitrary decisions (Tusche, Bode, & Haynes, 2010), as they are trying
819 to find a way to break the symmetry between the two possible actions. If so, the RP in the
820 arbitrary decisions might actually reflect the extra effort in those types of decisions, which is
821 not found in deliberate decisions. However, this interpretation entails a longer reaction time for
822 arbitrary than for deliberate decisions, because of the heavier cognitive load, which is the
823 opposite of what we found (Fig. 2A).

824 In conclusion, our study suggests that RPs do not precede deliberate decisions (or at the very
825 least are strongly diminished before such decisions). In addition, it suggests that RPs represent
826 an artificial accumulation of random fluctuations rather than serving as a genuine marker of an
827 unconscious decision to initiate voluntary movement. Hence, our results challenge RP-based
828 claims of Libet and follow-up literature against free will in arbitrary decisions and much more
829 so the generalization of these claims to deliberate decisions. The neural differences we found
830 between arbitrary and deliberate decisions as well as our model further put the onus on any
831 study trying to draw conclusions about the free-will debate from arbitrary decisions to
832 demonstrate that these conclusions generalize to deliberate ones. This motivates future
833 investigations into other precursors of action besides the RP using EEG, fMRI, or other
834 techniques. It also highlights that it would be of particular interest to find the neural activity

835 that precedes deliberate decisions as well as neural activity, which is not motor activity, that is
836 common to both deliberate and arbitrary decisions.

837 **Materials and Methods**

838 *Subjects*

839 Twenty healthy subjects participated in the study. They were California Institute of
840 Technology (Caltech) students as well as members of the Pasadena community. All subjects
841 had reported normal or corrected-to-normal sight and no psychiatric or neurological history.
842 They volunteered to participate in the study for payment ($20 per hour). Subjects were
843 prescreened to include only participants who were socially involved and active in the
844 community (based on the strength of their support of social causes, past volunteer work, past
845 donations to social causes, and tendency to vote). The data from 18 subjects was analyzed; two
846 subjects were excluded from our analysis (see *Sample size and exclusion criteria* below). The
847 experiment was approved by Caltech's Institutional Review Board (14-0432; Neural markers
848 of deliberate and random decisions), and informed consent was obtained from all participants
849 after the experimental procedures were explained to them.

850 *Sample size and exclusion criteria*

851 We ran a power analysis based on the findings of Haggard and Eimer (1999). Their RP in a
852 free left/right-choice task had a mean of 5.293 μV and standard deviation of 2.267 μV. Data
853 from a pilot study we ran before this experiment suggested that we might obtain smaller RP
854 values in our task (they referenced to the tip of the nose and we to the average of all channels,
855 which typically results in a smaller RP). Therefore, we conservatively estimated the magnitude
856 of our RP as half of that of Haggard & Eimer, 2.647 μV, while keeping the standard deviation
857 the same at 2.267 μV. Our power analysis therefore suggested that we would need at least 16
858 subjects to reliably find a difference between an RP and a null RP (0 μV) at a p-value of 0.05
859 and power of 0.99. This number agreed with our pilot study, where we found that a sample size
860 of at least 16 subjects resulted in a clear, averaged RP. Following the above reasoning, we
861 decided beforehand to collect 20 subjects for this study, taking into account that some could be
862 excluded as they would not meet the following predefined inclusion criteria: at least 30 trials
863 per experimental condition remaining after artifact rejection; and averaged RTs (across
864 conditions) that deviated by less than 3 standard deviations from the group mean.

865 Subjects were informed about the overall number of subjects that would participate in the
866 experiment when the NPO lottery was explained to them (see below). So, we had to finalize
867 the overall number of subjects who would participate in the study—but not necessarily the
868 overall number of subjects whose data would be part of the analysis—before the experiment
869 began. After completing data collection, we ran only the EEG preprocessing and behavioral-
870 data analysis to test each subject against the exclusion criteria. This was done before we looked
871 at the data with respect to our hypothesis or research question. Two subjects did not meet the
872 inclusion criteria: the data of one subject (#18) suffered from poor signal quality, resulting in
873 less than 30 trials remaining after artifact rejection; another subject (#12) had RTs longer than
874 3 standard deviations from the mean. All analyses were thus run on the 18 remaining subjects.

875 *Stimuli and apparatus*

876 Subjects sat in a dimly lit room. The stimuli were presented on a 21" Viewsonic G225f (20"
877 viewable) CRT monitor with a 60-Hz refresh rate and a 1024×768 resolution using
878 Psychtoolbox version 3 and Mathworks Matlab 2014b (Brainard, 1997; Pelli, 1997). They

appeared with a gray background (RGB values: [128, 128,128]). The screen was located 60 cm away from subjects' eyes. Stimuli included names of 50 real, non-profit organizations (NPOs). Twenty organizations were consensual (e.g., the Cancer Research Institute, or the Hunger project), and thirty were more controversial: we chose 15 causes that were widely debated (e.g., pro/anti guns, pro/anti abortions), and selected one NPO that supported each of the two sides of the debate. This was done to achieve variability in subjects' willingness to donate to the different NPOs. In the main part of the experiment, succinct descriptions of the causes (e.g., pro-marijuana legalization, pro-child protection; for a full list of NPOs and causes see Supplementary Table 1) were presented in black Comic Sans MS.

Study Design

The objective of this study was to compare ERPs elicited by arbitrary and deliberate decision-making, and in particular the RP. We further manipulated decision difficulty to validate our manipulation of decisions type: we introduced hard and easy decisions which corresponded to small and large differences between subjects' preferences for the pairs of presented NPOs, respectively. We reasoned that if the manipulation of decision type (arbitrary vs. deliberate) was effective, there would be behavioral differences between easy and hard decisions for deliberate choices but not for arbitrary choices (because differences in preferences should not influence subjects' arbitrary decisions). Our 2 x 2 design was therefore decision type (arbitrary vs. deliberate) by decision difficulty (easy vs. hard). Each condition included 90 trials, separated into 10 blocks of 9 trials each, resulting in a total of 360 trials and 40 blocks. Blocks of different decision types were randomly intermixed. Decision difficulty was randomly counterbalanced across trials within each block.

Experimental Procedure

In the first part of the experiment, subjects were presented with each of the 50 NPOs and the causes with which the NPOs were associated separately (see Supplementary Table 1). They were instructed to rate how much they would like to support that NPO with a $1000 donation on a scale of 1 ("I would not like to support this NPO at all) to 7 ("I would very much like to support this NPO"). No time pressure was put on the subjects, and they were given access to the website of each NPO to give them the opportunity to learn more about the NPO and the cause it supports.

After the subjects finished rating all NPOs, the main experiment began. In each block of the experiment, subjects made either deliberate or arbitrary decisions. Two succinct cause descriptions, representing two actual NPOs, were presented in each trial (Fig. 1). In deliberate blocks, subjects were instructed to choose the NPO to which they would like to donate $1000 by pressing the <Q> or <P> key on the keyboard, using their left and right index finger, for the NPO on the left or right, respectively, as soon as they decided. Subjects were informed that at the end of each block one of the NPOs they chose would be randomly selected to advance to a lottery. Then, at the end of the experiment, the lottery will take place and the winning NPO will receive a $20 donation. In addition, that NPO will advance to the final, inter-subject lottery, where one subject's NPO will be picked randomly for a $1000 donation. It was stressed that the donations were real and that no deception was used in the experiment. To persuade the subjects that the donations were real, we presented a signed commitment to donate the money, and promised to send them the donation receipts after the experiment. Thus, subjects knew that in deliberate trials, every choice they made was not hypothetical, and could potentially lead to an actual $1020 donation to their chosen NPO.

Arbitrary trials were identical to deliberate trials except for the following crucial differences. Subjects were told that, at the end of each block, the pair of NPOs in one randomly selected trial would advance to the lottery together. And, if that pair wins the lottery, both NPOs would receive $10 (each). Further, the NPO pair that would win the inter-subject lottery would receive a $500 donation each. Hence it was stressed to the subjects that there was no reason for them to prefer one NPO over the other in arbitrary blocks, as both NPOs would receive the same donation regardless of their button press. Subjects were told to therefore simply press either <Q> or <P> as soon as they decided to do so.

Thus, while subjects' decisions in the deliberate blocks were meaningful and consequential, their decisions in the arbitrary blocks had no impact on the final donations that were made. In these trials, subjects were further urged not to let their preferred NPO dictate their response. Importantly, despite the difference in decision type between deliberate and arbitrary blocks, the instructions for carrying out the decisions were identical: Subjects were instructed to report their decisions as soon as they made them in both conditions. They were further asked to place their left and right index fingers on the response keys, so they could respond as quickly as possible. Note that we did not ask subjects to report their "W-time" (time of consciously reaching a decision), because this measure was shown to rely on neural processes occurring after response onset (Lau, Rogers, & Passingham, 2007) and to potentially be backward inferred from movement time (Banks & Isham, 2009). Even more importantly, clock monitoring was demonstrated to have an effect on RP size (Miller et al., 2011), so it could potentially confound our results (Maoz et al., 2015).

Decision difficulty (Easy/Hard) was manipulated throughout the experiment, randomly intermixed within each block. Decision difficulty was determined based on the rating difference between the two presented NPOs. NPO pairs with 1 or at least 4 rating-point difference were designated hard or easy, respectively. Based on each subject's ratings, we created a list of NPO pairs, half of each were easy choices and the other half hard choices.

Each block started with an instruction written either in dark orange (Deliberate: "In this block choose the cause to which you want to donate $1000") or in blue (Arbitrary: "In this block both causes may each get a $500 donation regardless of the choice") on a gray background that was used throughout the experiment. Short-hand instructions appeared at the top of the screen throughout the block in the same colors as that block's initial instructions; Deliberate: "Choose for $1000" or Arbitrary: "Press for $500 each" (Fig. 1).

Each trial started with the gray screen that was blank except for a centered, black fixation cross. The fixation screen was on for a duration drawn from a uniform distribution between 1 and 1.5 s. Then, the two causes appeared on the left and right side of the fixation cross (left/right assignments were randomly counterbalanced) and remained on the screen until the subjects reported their decisions with a key press—<Q> or <P> on the keyboard for the cause on the left or right, respectively. The cause corresponding to the pressed button then turned white for 1 s, and a new trial started immediately. If subjects did not respond within 20 s, they received an error message and were informed that, if this trial would be selected for the lottery, no NPO would receive a donation. However, this did not happen for any subject on any trial.

To assess the consistency of subjects' decisions during the main experiment with their ratings in the first part of the experiment, subjects' choices were coded in the following way: each binary choice in the main experiment was given a consistency grade of 1, if subjects chose the NPO that was rated higher in the rating session, and 0 if not. Then an averaged consistency grade for each subject was calculated as the mean consistency grade over all the choices. Thus,

970 a consistency grade of 1 indicates perfect consistency with one's ratings across all trials, 0 is
971 perfect inconsistency, and 0.5 is chance performance.

972 We wanted to make sure subjects were carefully reading and remembering the causes also
973 during the arbitrary trials. This was part of an effort to better equate—as much as possible,
974 given the inherent difference between the conditions—memory load, attention, and other
975 cognitive aspects between deliberate and arbitrary decisions—except those aspects directly
976 associated with the decision type, which was the focus of our investigation. We therefore
977 randomly interspersed 36 memory catch-trials throughout the experiment (thus more than one
978 catch trial could occur per block). On such trials, four succinct descriptions of causes were
979 presented, and subjects had to select the one that appeared in the previous trial. A correct or
980 incorrect response added or subtracted 50 cents from their total, respectively. (Subjects were
981 informed that, if they reached a negative balance, no money will be deducted off their payment
982 for participation in the experiment.) Thus, subjects could earn $18 more for the experiment, if
983 they answered all memory test questions correctly. Subjects typically did well on these
984 memory questions, on average erring in 2.5 out of 36 memory catch trials (7% error) and
985 gaining additional $16.75 (SD=3.19). Subjects' error rates in the memory task did not differ
986 significantly between the experimental conditions (2-way ANOVA; decision type:
987 $F(1,17)=2.51$, $p=0.13$; decision difficulty: $F(1,17)=2.62$, $p=0.12$; interaction: $F(1,17)=0.84$,
988 $p=0.37$).

989 *ERP recording methods*

990 The EEG was recorded using an Active 2 system (BioSemi, the Netherlands) from 64
991 electrodes distributed based on the extended 10–20 system and connected to a cap, and seven
992 external electrodes. Four of the external electrodes recorded the EOG: two located at the outer
993 canthi of the right and left eyes and two above and below the center of the right eye. Two
994 external electrodes were located on the mastoids, and one electrode was placed on the tip of the
995 nose. All electrodes were referenced during recording to a common-mode signal (CMS)
996 electrode between POz and PO3. The EEG was continuously sampled at 512 Hz and stored for
997 offline analysis.

998 *ERP analysis*

999 ERP analysis was conducted using the "Brain Vision Analyzer" software (Brain Products,
1000 Germany) and in-house Mathworks Matlab scripts. Data from all channels were referenced
1001 offline to the average of all channels (excluding external electrodes), which is known to result
1002 in a reduced-amplitude RP (because the RP is such a spatially diffuse signal). The data were
1003 then digitally high-pass filtered at 0.1 Hz using a Finite Impulse Response (FIR) filter to
1004 remove slow drifts. A notch filter at 59-61 Hz was applied to the data to remove 60-Hz
1005 electrical noise. The signal was then cleaned of blink and saccade artifacts using Independent
1006 Component Analysis (ICA) (Junghofer, Elbert, Tucker, & Rockstroh, 2000). Signal artifacts
1007 were detected as amplitudes exceeding ±100 μV, differences beyond 100 μV within a 200 ms
1008 interval, or activity below 0.5 μV for over 100 ms (the last condition was never found).
1009 Sections of EEG data that included such artifacts in any channel were removed (150 ms before
1010 and after the artifact). We further excluded single trials in which subjects pressed a button that
1011 was not one of the two designated response buttons (<Q> or <P>) as well as trials where
1012 subjects' RTs were less than 200 ms, more than 10s, or more than 3 standard deviations away
1013 from that subject's mean in that condition (mean number of excluded trials =7.17, SD=2.46,
1014 which are 1.99% of the trials). Overall, the average number of included trials in each
1015 experimental cell was 70.38 trials with a range of 36-86 out of 90 trials per condition. Channels
1016 that consistently had artifacts were replaced using interpolation (0-6 channels, 1.95 channels

per subject, on average). No significant differences were found in the number of excluded trials across conditions (2-way ANOVA; decision type: $F(1,17)=3.31$, $p=0.09$; decision difficulty: $F(1,17)=1.83$, $p=0.19$; interaction: $F(1,17)=0.42$, $p=0.53$).

The EEG was segmented by locking the waveforms to subjects' movement onset, starting 2s prior to the movement and ending 0.2s afterwards, with the segments averaged separately for each decision type (Deliberate/Arbitrary x Easy/Hard) and decision content (right/left hand). The baseline period was defined as the time window between -1000 ms and -500 ms prior to *stimulus* onset, that is, the onset of the causes screen, rather than prior to movement onset. In addition to the main baseline, we tested another baseline—from -1000 ms to -500 ms relative to *movement* onset—to investigate whether the baseline period influenced our main results (see Results). Furthermore, we segmented the EEG based on *stimulus* onset, using the same baseline, for stimulus-locked analysis (again, see Results).

To assess potential effects of eye movements during the experiment, we defined the radial eye signal as the average over all 4 EOG channels, when band-pass filtered to between 30 and 100 Hz. We then defined a saccade as any signal that was more than 2.5 standardized IQRs away from the median of the radial signal for more than 2 ms. Two consecutive saccades had to be at least 50 ms apart. The saccade count (SC) was the number of saccades during the last 500 ms before response onset (Keren, Yuval-Greenberg, & Deouell, 2010) (see also (Croft & Barry, 2000; Elbert, Lutzenberger, Rockstroh, & Birbaumer, 1985; Shan, Moster, & Roemer, 1995)).

Statistical Analysis

EEG differences greater than expected by chance were assessed using two-way ANOVAs with decision type (deliberate, arbitrary) and decision difficulty (easy, hard), using IBM SPSS statistics, version 24. For both RP and LRP signals, the mean amplitude from 500 ms before to button-press onset were used for the ANOVAs. Greenhouse–Geisser correction was never required as sphericity was never violated (Picton et al., 2000).

Trend analysis on all subjects' data was carried out by regressing the voltage for every subject against time for the last 1000 ms before response onset using first-order polynomial linear regression (see Results). We first added a 25 Hz low-pass filter for anti-aliasing and then used every 10th time sample for the regression (i.e., the 1st, 11th, 21st, 31st samples, and so on) to conform with the individual-subject analysis (see below). For the individual-subject analysis, the voltage on all trials was regressed against time in the same manner (i.e., for the last 1000 ms before response onset and using first-order polynomial linear regression). As individual-trial data is much noisier than the mean over all trials in each subject, we opted for standard robust-regression using iteratively reweighted least squares (implemented using the *robustfit()* function in Mathworks Matlab). The iterative robust-regression procedure is time consuming. So, we used every 10th time sample instead of every sample to make the procedure's run time manageable. Also, as EEG signals have a 1/f power spectrum, taking every 10th sample further better conforms with the assumption of i.i.d. noise in linear regression.

Furthermore, we conducted Bayesian analyses of our main results. This allowed us to assess the strength of the evidence for or against the existence of an effect, and specifically test whether null results stem from genuine absence of an effect or from insufficient or underpowered data. Specifically, the Bayes factor allowed us to compare the probability of observing the data given H_0 (i.e., no RP in deliberate decisions) against the probability of observing the data given H_1 (i.e., RP exists in deliberate decisions). We followed the convention that $BF < 0.33$ implies substantial evidence for lack of an effect (that is, the data is at least three times more likely to be observed given H_0 than given H_1), $0.33 < BF < 3$ suggests

insensitivity of the data, and BF > 3 denotes substantial evidence for the presence of an effect (H_1) (Jeffreys, 1998). Bayesian analysis was carried out using JASP (ver. 0.8; default settings).

In addition to the above, we used the cluster-based nonparametric method developed by Maris and Oostenveld to find continuous temporal windows where EEG activity was reliably different from 0 (Maris & Oostenveld, 2007). We used an in-house implementation of the method in Mathworks Matlab with a threshold of 2 on the *t* statistic and with a significance level of $p = 0.05$.

Model and Simulations

All simulations were performed using Mathworks Matlab 2018b. The model was devised off the one proposed by Schurger et al. (2012). Like them, we built a drift-diffusion model (DDM) (Ratcliff, 1978; Usher & McClelland, 2001), which included a leaky stochastic accumulator (with a threshold on its output) and a time-locking/epoching procedure. The original model amounted to iterative numerical integration of the differential equation

$$\delta x_i = (I - kx_i)\Delta t + c\xi_i\sqrt{\Delta t} \tag{1}$$

where I is the drift rate, k is the leak (exponential decay in x), ξ is Gaussian noise, and c is a noise-scaling factor. Δt is the discrete time step used in the simulation (we used $\Delta t = 0.001$). The model integrates x_i over its iterations until it crosses a threshold, which represents a decision having been made.

In such drift-diffusion models, for a given k and c, the values of I and the threshold together determine how quickly a decision will be reached, on average. If we further fix the threshold, a higher drift rate, I, represents a faster decision, on average. The drift rate alone can thus be viewed as a constant "urgency to respond" (using the original Schurger term) that is inherent in the demand characteristics of the task, evidenced by the fact that no subject took more than 20 s to make a decision on any trial. The leak term, k, ensures that the model would not be too linear; i.e., it prevented the drift rate from setting up a linear trajectory for the accumulator toward the threshold. Also, k has a negative sign and is multiplied by x_i. So, kx_i acts against the drift induced by I and gets stronger as x_i grows. Hence, due to the leak term, doubling the height of the threshold could make the accumulator rarely reach the threshold instead of reaching it in roughly twice the amount of time (up to the noise term).

When comparing the model's activity for the SMA and Region X, we needed to know how to set the drift rates for the DDM in the two regions for deliberate decisions. We made the assumption that the ratio between the drift rate in Region X and in the SMA during deliberate decisions would be the same as the ratio between the average empirical activity in the SMA and in the rest of the brain during arbitrary decisions. Our EEG data suggested that this ratio (calculated as activity in C_z divided by the mean activity in the rest of the electrodes) is 1.45. Hence, we set the drift rates in the SMA to be 1.45 times smaller than those of Region X for deliberate decisions (see Table 1 for all parameter values).

Our model differed from Schurger's in two main ways. First, it accounted for both arbitrary and deliberate decisions and was thus built to fit our paradigm and empirical results. We devised a model that was composed of two distinct components (Fig. 8A), each a race to threshold between 2 DDMs based on Eq. (1) (see below), but with different parameter values for each DDM (Table 1). The first component accumulated activity that drove arbitrary

1104 decisions (i.e., stochastic fluctuations (Schurger et al., 2012)). Such model activation reflects
1105 neural activity that might be recorded over the SMA by the C_z electrode. We term this
1106 component of the model the *Noise* or *SMA* component. The second component of the model
1107 reflects brain activity that drives deliberate decisions, based on the values that subjects
1108 associated with the decision alternatives. We term this second component the *Value* or *Region*
1109 *X* component. Our model relied on its noise component to reflect arbitrary decisions and on its
1110 value component to reflect deliberate decisions.

1111 A second difference between our model and Schurger and colleagues' is that theirs modeled
1112 only the decision *when* to move (during arbitrary decisions), as those were the only decisions
1113 that their subjects faced. But our subjects decided both *when* and *which hand* to move. So, we
1114 had to extend the Schurger model in that respect as well. We did this using a race-to-threshold
1115 mechanism between the decision alternatives. In our empirical paradigm, the difference in
1116 rating of the two causes was either 1 (for hard decisions) or 4-6 (for easy decisions; see
1117 "Experimental Procedure" in Methods), so there was always an alternative that was ranked
1118 higher than the other. Choosing the higher- or lower-ranked alternative was termed a congruent
1119 or incongruent choice with respect to the initial ratings, respectively. Hence, we modeled each
1120 decision the subjects made as a race to threshold between the DDMs for the congruent and
1121 incongruent alternatives in the noise component (for arbitrary decisions) or value component
1122 (for deliberate ones).

1123 We found the reaction time (RT) distribution and consistency score for each subject (as
1124 detailed above). The model's RT was defined as the overall time that it took from the onset of
1125 the simulation until the first threshold crossing in the race-to-threshold pair (between the
1126 congruent and incongruent DDM; for $\Delta t = 0.001$ s). To determine the RT error, we ran a
1127 similar procedure to Schurger et al. (2012). We computed the empirical, cumulative RT
1128 distribution for each subject and fit a gamma function to it. We then averaged those gamma-
1129 fitted distributions across all subjects and designated that the empirical distribution. Then, we
1130 computed the RT for the model for each parameter set from 1000 model runs and fitted a
1131 gamma function to that too. Finally, we computed the ratio of the intersection of the two
1132 cumulative distributions and their union; and the RT error was defined as 1 minus that ratio.
1133 The consistency error was computed as the absolute difference between the empirical and
1134 model consistencies. Finally, the overall error was defined as mean of these two errors.

1135 To find the parameter values that minimize this overall error, we ran an exhaustive grid search
1136 over all the parameters. To do this, we fixed the threshold at 0.3 and found the values of the
1137 congruent and incongruent drift rates as well as the leak and the noise scaling factors that, at
1138 the same time, best fit the empirical distribution of the RTs and the consistency scores (with
1139 equal weights) for (easy, hard) x (deliberate, arbitrary) decisions (Table 1) in the manner
1140 described below. We had 4 parameters to fit: the congruent drift rate ($I_{congruent}$), the incongruent
1141 drift rate ($I_{incongruent}$), the leak (k), and the noise-scaling factor (c). We first explored the 4D
1142 space of those 4 parameters to test how smooth it was and to find the range in which to run the
1143 exhaustive grid search. We found that $I_{congruent}$ was minimized between 0.05 and 0.4 and that
1144 $I_{incongruent}$ was minimized somewhere between the value of the $I_{congruent}$ and 5 times smaller than
1145 that. Then k was found to lie between 0.2 and 0.55, and c between 0.01 and 0.3. Based on our
1146 tests and specifically the smoothness of the search space, we found that we could divide each
1147 parameter range into 5 equal parts. So, we then tested the model in each of the 5 x 5 x 5 x5 (=
1148 625) entries in the grid by running the model 1000 times and computing its error by comparing
1149 its RT distribution and consistency rate to the empirical ones. Once we found the minimum
1150 point in that 4D grid, we zoomed in on the range between the grid value to the left of the entry
1151 were the minimum error was found and the one to the right of that minimum. (We chose the
1152 initial range such that the minimum error was never achieved on the smallest or largest point

for all parameters.) We continued this process of finding the minimum and zooming in until each parameter reached a range that was smaller than 0.025 or the error was less than 0.025.

Once we found the parameters that minimized the mismatch between the empirical and model RTs and consistencies for each decision type, we ran the model with those parameters 1000 times again. For each run of the model, we identified the first threshold crossing point (for the congruent or incongruent DDM), which we termed that model the "winning" DDN. We then extracted the activity in both the winning and losing DDM in the last 2 s (2000 steps) before the crossing in each. And we averaged across the activity of the two DDM (because an electrode over the SMA or Region X would be expected to pick up signals from both DDMs). If the first crossing was earlier than sample no. 2,000 by $n > 0$ samples, we padded the beginning of the epoch with n null values (NaN or "not-a-number" in Matlab). Finally, we averaged across all 1000 model runs to calculate the model's RP. Note that NaN values did not contribute to the average across simulated trials, so the simulated average RP became noisier at earlier time points in the epoch. Hence, our model was similarly limited to the Schurger model in its inability to account for activity earlier than the beginning of the trial (see Results).

Table 1: Values of the model's parameters across decision types and decision difficulties. Values of the drift-rate parameter, I, for the congruent and incongruent options; for the leak rate, k; and for the noise scaling factor, c. We fixed the threshold at the value of 0.3. For deliberate decisions, drift-rate values in SMA were 1.45 times smaller than in this table for each entry.

Decision Type	Decision Difficulty	$I_{congruent}$	$I_{incongruent}$	k	c
Deliberate	Easy	0.23	0.06	0.52	0.08
	Hard	0.15	0.07	0.21	0.09
Arbitrary	Easy	0.24	0.21	0.53	0.22
	Hard	0.22	0.20	0.54	0.23

References

Banks, W. P., & Isham, E. A. (2009). We infer rather than perceive the moment we decided to act. *Psychological Science, 20*(1), 17.

Bode, S., & Haynes, J. (2009). Decoding sequential stages of task preparation in the human brain. *Neuroimage, 45*(2), 606-613.

Bode, S., He, A., Soon, C., Trampel, R., Turner, R., & Haynes, J. (2011). Tracking the unconscious generation of free decisions using uitra-high field fMRI. *PLoS One, 6*(6), e21612.

Brainard, D. H. (1997). The psychophysics toolbox. *Spatial vision, 10*, 433-436.

Brass, M., & Haggard, P. (2008). The what, when, whether model of intentional action. *The Neuroscientist, 14*(4), 319.

Breitmeyer, B. G. (1985). Problems with the psychophysics of intention. *Behavioral and brain sciences, 8*(04), 539-540.

Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influences in anterior cingulate cortex. *Trends in Cognitive Sciences, 4*(6), 215-222.

Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D., & Cohen, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science, 280*(5364), 747-749.

Croft, R. J., & Barry, R. J. (2000). Removal of ocular artifact from the EEG: a review. *Neurophysiologie Clinique/Clinical Neurophysiology, 30*(1), 5-19.

de Lafuente, V., Jazayeri, M., & Shadlen, M. N. (2015). Representation of accumulating evidence for a decision in two parietal areas. *Journal of Neuroscience, 35*(10), 4306-4318.

Deutschländer, R., Pauen, M., & Haynes, J.-D. (2017). Probing folk-psychology: Do Libet-style experiments reflect folk intuitions about free action? *Consciousness and cognition, 48*, 232-245.

Eimer, M. (1998). The lateralized readiness potential as an on-line measure of central response activation processes. *Behavior Research Methods, Instruments, & Computers, 30*(1), 146-156.

Elbert, T., Lutzenberger, W., Rockstroh, B., & Birbaumer, N. (1985). Removal of ocular artifacts from the EEG—a biophysical approach to the EOG. *Electroencephalography and Clinical Neurophysiology, 60*(5), 455-463.

Fischer, J. M. (1999). Recent work on moral responsibility. *Ethics, 110*(1), 93-139.

Frankfurt, H. G. (1971). Freedom of the Will and the Concept of a Person. *The Journal of Philosophy, 68*(1), 5-20.

Fried, I., Mukamel, R., & Kreiman, G. (2011). Internally generated preactivation of single neurons in human medial frontal cortex predicts volition. *Neuron, 69*, 548-562.

Frith, C., Blakemore, S., & Wolpert, D. (2000). Abnormalities in the awareness and control of action. *Philosophical Transactions of the Royal Society B: Biological Sciences, 355*(1404), 1771.

Frith, C. D., & Haggard, P. (2018). Volition and the Brain–Revisiting a Classic Experimental Study. *TRENDS in Neurosciences, 41*(7), 405-407.

Gold, J., & Shadlen, M. (2007). The neural basis of decision making.

Haggard, P. (2005). Conscious intention and motor cognition. *Trends in Cognitive Sciences, 9*(6), 290-295.

Haggard, P. (2008). Human volition: towards a neuroscience of will. *Nature Reviews Neuroscience, 9*(12), 934-946.

Haggard, P. (2019). The neurocognitive bases of human volition. *Annual Review of Psychology, 70*, 9-28.

Haggard, P., & Eimer, M. (1999). On the relation between brain potentials and the awareness of voluntary movements. *Experimental Brain Research, 126*(1), 128-133.

Hallett, M. (2007). Volitional control of movement: The physiology of free will. *Clinical Neurophysiology, 118*(6), 1179-1192.

Hallett, M. (2016). Physiology of free will. *Annals of neurology, 80*(1), 5-12.

Harris, S. (2012). *Free will*. New York, NY: Simon & Schuster, Inc.

Hobbes, T. (1994). *Leviathan: with selected variants from the Latin edition of 1668*: Hackett Publishing Company.

Hughes, G., Schütz-Bosbach, S., & Waszak, F. (2011). One action system or two? Evidence for common central preparatory mechanisms in voluntary and stimulus-driven actions. *Journal of Neuroscience, 31*(46), 16692-16699.

Jeannerod, M. (2006). *Motor cognition: What actions tell the self*: Oxford University Press.

Jeffreys, H. (1998). *The theory of probability*: OUP Oxford.

Junghofer, M., Elbert, T., Tucker, D., & Rockstroh, B. (2000). Statistical control of artifacts in dense array EEG/MEG studies. *Psychophysiology, 37*(04), 523-532.

Kable, J. W., & Glimcher, P. W. (2009). The neurobiology of decision: consensus and controversy. *Neuron, 63*(6), 733-745.

Keren, A. S., Yuval-Greenberg, S., & Deouell, L. Y. (2010). Saccadic spike potentials in gamma-band EEG: characterization, detection and suppression. *Neuroimage, 49*(3), 2248-2263.

Kornhuber, H., & Deecke, L. (1990). Readiness for movement—the Bereitschafts potential-story. *Current Contents Life Sciences, 33*(4), 14.

Krieghoff, V., Waszak, F., Prinz, W., & Brass, M. (2011). Neural and behavioral correlates of intentional actions. *Neuropsychologia, 49*(5), 767-776.
Lau, H., Rogers, R., Haggard, P., & Passingham, R. (2004). Attention to intention. *Science, 303*(5661), 1208.
Lau, H., Rogers, R., & Passingham, R. (2007). Manipulating the experienced onset of intention after action execution. *Journal of cognitive neuroscience, 19*(1), 81-90.
Lee, M., & Wagenmakers, E. (2013). Bayesian data analysis for cognitive science: A practical course. In: New York, NY: Cambridge University Press.
Lew, E., Chavarriaga, R., Silvoni, S., & Millán, J. d. R. (2012). Detection of self-paced reaching movement intention from EEG signals. *Frontiers in Neuroengineering, 5*, 13.
Libet, B. (1985). Unconscious cerebral initiative and the role of conscious will in voluntary action. *Behavioral and brain sciences, 8*, 529-539.
Libet, B., Gleason, C., Wright, E., & Pearl, D. (1983). Time of conscious intention to act in relation to onset of cerebral activity (readiness-potential): The unconscious initiation of a freely voluntary act. *Brain, 106*(3), 623.
Libet, B., Wright, E., & Gleason, C. (1982). Readiness-potentials preceding unrestricted 'spontaneous' vs. pre-planned voluntary acts. *Electroencephalography and Clinical Neurophysiology, 54*(3), 322-335.
MacKinnon, C. D., Allen, D. P., Shiratori, T., & Rogers, M. W. (2013). Early and unintentional release of planned motor actions during motor cortical preparation. *PLoS One, 8*(5), e63417.
Maoz, U., Mudrik, L., Rivlin, R., Ross, I., Mamelak, A., & Yaffe, G. (2015). On reporting the onset of the intention to move. In A. R. Mele (Ed.), *Surrounding Free Will: Philosophy: Psychology, Neuroscience*: Oxford University Press.
Maoz, U., Rutishauser, U., Kim, S., Cai, X., Lee, D., & Koch, C. (2013). Predeliberation activity in prefrontal cortex and striatum and the prediction of subsequent value judgment. *Frontiers in neuroscience, 7*, 225.
Maoz, U., & Yaffe, G. (2015). What does recent neuroscience tell us about criminal responsibility? *Journal of Law and the Biosciences, 3*(1), 120-139.
Maris, E., & Oostenveld, R. (2007). Nonparametric statistical testing of EEG-and MEG-data. *Journal of neuroscience methods, 164*(1), 177-190.
Masaki, H., Wild-wall, N., Sangals, J., & Sommer, W. (2004). The functional locus of the lateralized readiness potential. *Psychophysiology, 41*(2), 220-230.
Mele, A. (2006). *Free will and luck*: Oxford University Press.
Mele, A. (2009). *Effective intentions: the power of conscious will*: Oxford University Press, USA.
Mele, A. (2015). *Surrounding free will: philosophy, psychology, neuroscience*: Oxford University Press.
Miller, J., Shepherdson, P., & Trevena, J. (2011). Effects of Clock Monitoring on Electroencephalographic Activity. *Psychological Science, 22*(1), 103-109.
Mudrik, L., & Maoz, U. (2014). “Me & My Brain”: Exposing Neuroscience's Closet Dualism. *Journal of cognitive neuroscience, 27*(2), 211-221.
Nahmias, E., Shepard, J., & Reuter, S. (2014). It’s OK if ‘my brain made me do it’: People’s intuitions about free will and neuroscientific prediction. *Cognition, 133*(2), 502-516.
Pelli, D. G. (1997). The VideoToolbox software for visual psychophysics: Transforming numbers into movies. *Spatial vision, 10*(4), 437-442.
Perez, O., Mukamel, R., Tankus, A., Rosenblatt, J. D., Yeshurun, Y., & Fried, I. (2015). Preconscious prediction of a driver's decision using intracranial recordings. *Journal of cognitive neuroscience*.
Picton, T., Bentin, S., Berg, P., Donchin, E., Hillyard, S., Johnson, R., . . . Rugg, M. (2000). Guidelines for using human event-related potentials to study cognition: recording standards and publication criteria. *Psychophysiology, 37*(02), 127-152.

Pockett, S., Banks, W. P., & Gallagher, S. (2009). *Does consciousness cause behavior?* : Mit Press.
Ramnani, N., & Owen, A. M. (2004). Anterior prefrontal cortex: insights into function from anatomy and neuroimaging. *Nature Reviews Neuroscience, 5*(3), 184-194.
Ratcliff, R. (1978). A theory of memory retrieval. *Psychological Review, 85*(2), 59-108.
Roskies, A. (2010). How Does Neuroscience Affect Our Conception of Volition? *Annual Review of Neuroscience, 33*, 109-130.
Roskies, A. (2012). How does the neuroscience of decision making bear on our understanding of moral responsibility and free will? *Current Opinion in Neurobiology, 22*(6), 1022-1026.
Salvaris, M., & Haggard, P. (2014). Decoding Intention at Sensorimotor Timescales. *PLoS One, 9*(2), e85100.
Sanfey, A., Loewenstein, G., McClure, S., & Cohen, J. (2006). Neuroeconomics: cross-currents in research on decision-making. *Trends in Cognitive Sciences, 10*(3), 108-116.
Sanfey, A., Rilling, J., Aronson, J., Nystrom, L., & Cohen, J. (2003). The neural basis of economic decision-making in the ultimatum game. *Science, 300*(5626), 1755-1758.
Schmidt, S., Jo, H.-G., Wittmann, M., & Hinterberger, T. (2016). 'Catching the waves'–slow cortical potentials as moderator of voluntary action. *Neuroscience & Biobehavioral Reviews, 68*, 639-650.
Schurger, A. (2018). Specific Relationship between the Shape of the Readiness Potential, Subjective Decision Time, and Waiting Time Predicted by an Accumulator Model with Temporally Autocorrelated Input Noise. *eNeuro, 5*(1), ENEURO. 0302-0317.2018.
Schurger, A., Sitt, J. D., & Dehaene, S. (2012). An accumulator model for spontaneous neural activity prior to self-initiated movement. *Proceedings of the National Academy of Sciences, 109*(42), E2904-E2913.
Shan, Y., Moster, M. L., & Roemer, R. A. (1995). The effects of time point alignment on the amplitude of averaged orbital presaccadic spike potential (SP). *Electroencephalography and Clinical Neurophysiology, 95*(6), 475-477.
Shibasaki, H., & Hallett, M. (2006). What is the Bereitschaftspotential? *Clinical Neurophysiology, 117*(11), 2341-2356.
Sinnott-Armstrong, W. (2014). *Moral Psychology: Free Will and Moral Responsibility* (Vol. 4): Mit Press.
Sinnott-Armstrong, W., & Nadel, L. (Eds.). (2011). *Conscious will and responsibility: A tribute to Benjamin Libet*: Oxford University Press.
Sirigu, A., Daprati, E., Ciancia, S., Giraux, P., Nighoghossian, N., Posada, A., & Haggard, P. (2004). Altered awareness of voluntary action after damage to the parietal cortex. *Nature Neuroscience, 7*(1), 80-84.
Soon, C., Brass, M., Heinze, H., & Haynes, J. (2008). Unconscious determinants of free decisions in the human brain. *Nature Neuroscience, 11*(5), 543-545.
Soon, C., He, A., Bode, S., & Haynes, J. (2013). Predicting free choices for abstract intentions. *Proceedings of the National Academy of Sciences, 110*(15), 6217-6222.
Strawson, G. (1994). The impossibility of moral responsibility. *Philosophical Studies, 75*(1), 5-24.
Thut, G., Hauert, C.-A., Viviani, P., Morand, S., Spinelli, L., Blanke, O., . . . Michel, C. (2000). Internally driven vs. externally cued movement selection: a study on the timing of brain activity. *Cognitive Brain Research, 9*(3), 261-269.
Trevena, J., & Miller, J. (2010). Brain preparation before a voluntary action: Evidence against unconscious movement initiation. *Consciousness and cognition, 19*(1), 447-456.
Tusche, A., Bode, S., & Haynes, J. (2010). Neural responses to unattended products predict later consumer choices. *Journal of Neuroscience, 30*(23), 8024-8031.
Ullmann-Margalit, E., & Morgenbesser, S. (1977). Picking and choosing. *Social Research, 44*(4), 757-783.

Usher, M., & McClelland, J. L. (2001). The time course of perceptual choice: the leaky, competing accumulator model. *Psychological Review, 108*(3), 550.
Verleger, R., Haake, M., Baur, A., & Śmigasiewicz, K. (2016). Time to Move Again: Does the Bereitschaftspotential Covary with Demands on Internal Timing? *Frontiers in human neuroscience, 10*.
Wallis, J. (2007). Orbitofrontal cortex and its contribution to decision-making. *Annu. Rev. Neurosci., 30*, 31-56.
Wallis, J., & Miller, E. (2003). Neuronal activity in primate dorsolateral and orbital prefrontal cortex during performance of a reward preference task. *Eur J Neurosci, 18*(7), 2069-2081. doi:2922 [pii]
Waszak, F., Wascher, E., Keller, P., Koch, I., Aschersleben, G., Rosenbaum, D. A., & Prinz, W. (2005). Intention-based and stimulus-based mechanisms in action selection. *Experimental Brain Research, 162*(3), 346-356.
Wegner, D. (2002). *The illusion of conscious will*: MIT Press.
Wisniewski, D., Goschke, T., & Haynes, J.-D. (2016). Similar coding of freely chosen and externally cued intentions in a fronto-parietal network. *Neuroimage, 134*, 450-458.
Wolf, S. (1990). *Freedom within reason*: Oxford University Press.
Wood, C. C. (1985). Pardon, your dualism is showing. *Behavioral and brain sciences, 8*(04), 557-558.

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1396 Supplementary Data

1397 **Supplementary Table 1: NPO names and causes acronyms**

NPO	Cause	NPO website
Consensual NPOs		
American Society on Aging	Pro Quality of Life for the Elderly	http://asaging.org/
Conservation Fund	Pro Environment protection	http://www.conservationfund.org/
Bill & Melinda Gates Foundation	Pro Education	http://www.gatesfoundation.org/
Global Fund for Women	Pro Women's Rights	https://www.globalfundforwomen.org/
The Hunger Project	Pro Hunger Relief	https://www.thp.org/
Oxfam International	Pro Poverty & Disaster Relief	http://www.oxfam.org/
World Wild Life Fund (WWF)	Pro Species Conservation	http://worldwildlife.org/
Cancer Research Institute	Pro Cancer Research	http://www.cancerresearch.org/
Habitat for Humanity	Pro Housing for All	http://www.habitat.org/
Reading is Fundamental	Pro Advancement of Literacy	http://www.rif.org/
International Institute for Conservation of Historic and Artistic Works	Pro Culture & Arts Preservation	https://www.iiconservation.org/
Big Brothers and Big Sisters of America	Pro Youth Development	http://www.bbbs.org/site/c.9iILI3NGKhK6F/ b.5962335/k.BE16/Home.htm
United Nations Children's Fund (UNICEF)	Pro Child Protection	http://www.unicef.org/
Doctors without Borders (Medecins sans frontieres)	Pro Disaster Medical Care	http://www.msf.org/
Soldiers' Angels	Pro Veterans & Military	http://www.soldiersangels.org/heroes/index.php
Disability Rights International	Pro Disabilities Rights	http://www.disabilityrightsintl.org/

National Crime Prevention Council (NCPC)	Pro Crime Prevention	http://www.ncpc.org/
Amnesty International	Pro Human Rights	https://www.amnesty.org/
Peace Corps	Pro Peace & Development	http://www.peacecorps.gov/
World Health Organization	Pro World Health	http://www.who.int/en/
Controversial NPOs		
Planned Parenthood	Pro Abortion & Family Planning	http://www.plannedparenthood.org/
Pro-Life Alliance	Anti Abortion & Family Planning	http://www.prolifealliance.com/
Human Rights Campaign	Pro LBGTQ Rights	http://www.hrc.org/
National Organization for Marriage	Anti LBGTQ Rights	https://www.nationformarriage.org/
Stem for Life Foundation	Pro Stem Cell Research	http://www.stemforlife.org/
Christian Dental & Medical Association	Anti Stem Cell Research	http://www.cmda.org/
Greenpeace	Pro Action Against Climate Change	http://www.greenpeace.org/international/en/
Global Climate Scam	Anti Action Against Climate Change	http://www.globalclimatescam.com/
National Association for Gun Rights	Pro Gun Rights	http://www.nationalgunrights.org/
Coalition to Stop Gun Violence	Pro Gun Control	http://csgv.org/
American Gas Association	Pro Fracking for Natural Gas	http://www.aga.org/Pages/default.aspx
Americans Against Fracking	Anti Fracking for Natural Gas	http://www.americansagainstfracking.org/
StandWithUs (Israel)	Pro Israel	http://www.standwithus.com/
Palestinian Centre for Human Rights	Pro Palestine	http://www.pchrgaza.org/portal/en/
National Organization for the Reform of Marijuana Laws	Pro Marijuana Legalization	http://norml.org/
Citizens Against Legalizing Marijuana	Anti Marijuana Legalization	http://www.calmca.org/
Understanding Animal Research	Pro Scientific Experiments on Animals	http://www.understandinganimalresearch.org.uk/

International Association Against Painful Experiments on Animals	Anti Scientific Experiments on Animals	http://www.iaapea.com/
Federation for American Immigration Reform	Pro Immigration Reform	http://www.fairus.org/
American Immigration Control	Anti Immigration Reform	http://www.immigrationcontrol.com/
Human Cloning Foundation	Pro Human Cloning	http://www.humancloning.org/
Americans to Ban Cloning	Anti Human Cloning	http://www.cloninginformation.org/
Americans United for Separation of Church and State	Pro Separation of Church & State	https://www.au.org/
Christian Coalition of America	Anti Separation of Church & State	http://www.cc.org/
Death with Dignity National Center	Pro Euthanasia (Assisted Suicide)	http://www.deathwithdignity.org/
Euthanasia Prevention Coalition	Anti Euthanasia (Assisted Suicide)	http://www.epcc.ca/
The Alliance for Better Foods	Pro Genetically Modified Foods	http://www.betterfoods.org/
Non-GMO Project	Anti Genetically Modified Foods	http://www.nongmoproject.org/
Answers in Genesis	Pro Creationism Teaching	https://answersingenesis.org
National Center for Science Education	Pro Evolution Teaching	http://ncse.com/

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