Published in final edited form as:

Int J Psychophysiol. 2013 March ; 87(3): 254–261. doi:10.1016/j.ijpsycho.2012.08.010.

The Influence of Risky and Conservative Mental Sets on Cerebral Activations of Cognitive Control

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Abstract

Mental set is known to influence cognitive functioning. Risk-seeking and risk-averse mental sets alter cerebral responses to conflicting events. Here, building on our previous imaging work of the stop signal task, we introduced a “reward uncertainty” condition to elicit changes in participants' mental sets and examined how individual differences altered the neural responses to salient events. Approximately half of 27 adult participants – the Conservatives – became more risk-averse in the “reward” as compared to the “standard” condition, by slowing down in go reaction time. We hypothesized that stop errors were more salient for these participants, as compared to the other subjects – the Riskys – who speeded up in go reaction time in the reward as compared to standard condition. With statistical parametric mapping, we showed greater activation of the retrosplenial cortex, somatosensory cortex, pre-supplementary motor area, and thalamus during stop error, in contrast to stop success trials, in the Conservatives as compared to Riskys. These results provided evidence that mental set influences cerebral activations during stop signal performance and extended the potential utility of the stop signal task in elucidating the contextual effects on cognitive control.

Keywords

Stop-signal task; Reward uncertainty; Risk taking; Performance monitoring; Conflict resolution; Retrosplenial cortex

1. Introduction

Cognitive control enables effective learning and production of goal-directed actions. Most actions we produce are goal-directed, in that we care whether we produce them as intended or not. However, despite our desire to do so, certain mental sets may hinder the flexibility of thought required to optimize performance. A mental set is the tendency to respond to a new problem in the manner used to respond to an old problem (Gerrig & Zimbardo, 2002). This study explores whether individuals may alter their mental sets in response to a contextual

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manipulation and how risky and conservative mental sets may differentially affect cognitive control.

To illustrate, imagine you are driving in a hurry on your way to work. Approaching a green traffic light from a distance, you are uncertain when the light will turn yellow, and speed up slightly to reduce your time to the intersection. Based on your previous experiences, you slow down while you are at a critical decision making distance, say between 20 and 5 feet before the intersection. Then, if the light has not turned yellow, you are reasonably certain you will make it before the light turns red, so you continue through at a regular speed. Next, imagine driving under the influence of a risky mental set. You may choose to maintain your increased speed longer, making it more likely that you will reach the intersection before the light turns yellow, but knowing if the light turns yellow while you are at a critical decision making distance, you have forced yourself to choose between either coming to a screeching stop, or continuing despite the increased risk that the light will turn red before you are through the intersection. Finally, imagine driving under the influence of a conservative mental set. You are still behind schedule on your way to work, but having misplaced and driving without your license, you want to avoid being pulled over even more than usual. Exhibiting more cautious behavior, you do not speed up on your approach to the intersection and slow as you get closer in order to gracefully stop if the light turns yellow.

Driving under a risky or conservative mental set influences the cognitive and emotional conflicts attributed to these different “stop signal” scenarios. At times when you are more concerned about arriving on time, a decision to continue through a nearly red light is associated with less conflict and negative affect than deciding to stop and wait for the next green light. In contrast, when you are more concerned about complying with traffic laws, a decision to stop at a nearly red light would not be associated with much conflict and negative affect compared to a decision to continue through it. That is, stopping and driving through the red light is each more salient under a risky and conservative mental set, respectively. The experiment we present here provides support for the hypothesis that risky and conservative mental sets alter cerebral activations related to “stop success” and “stop error” during cognitive control.

The stop signal task (SST) is a psychophysical paradigm used to evaluate cognitive control and localize brain regions underlying related processes (Fig. 1; Li et al., 2006; Li et al., 2008a; Li et al., 2008b; Logan & Cowan, 1984). In the SST, participants respond to a “go” signal in go trials, which occur frequently. Occasionally, in stop trials, an additional “stop” signal instructs participants to withhold their response. Because the go response is prepotentiated and participants are required to respond both quickly and accurately, conflicts occur to a greater degree during stop trials.

Individuals' mental sets during the SST vary from moment to moment based on cumulative trial experience; for instance, individuals tend to slow down in go trials after they encounter an error (Ide & Li, 2011a; Li et al., 2008a). Mental sets are also amenable to task instructions, affective cues, and other general, contextual influences (Lupien et al., 2007; Pessoa, 2009; Eysenk, 2010; Padmala and Pessoa, 2010; Pessoa, in press; Padmala et al., in press). For instance, participants may be instructed to emphasize speed or accuracy (Bocagz et al., 2010; Verbruggen & Logan, 2009). Performance of the SST under a conservative mental set, where greater conflicts associated with a decision to respond, render stop errors more salient than stop successes; however, the difference in saliency between stop error and success trials may diminish in participants who chose to emphasize speed.

In the current study, we elicit changes in mental set by introducing an altered compensation scheme, in which the monetary reward is uncertain. Thus, while participants are

compensated a fixed amount of money for their participation in the standard version of the SST, their compensation in the “reward” version of the task would appear to depend entirely on chance (see below). The reward uncertainty as introduced in the latter task creates a change in mental set that alters response strategy, which allows us to categorize participants as risky or conservative. “Riskys” produce faster go trial reaction times (GORTs) and “Conservatives” produce slower GORTs in the reward than the standard condition. Our hypothesis is that, compared to Conservatives, Riskys may demonstrate less error-related activation in a contrast between stop error and stop success trials, analogous to our driving example, in which running a red light is more salient for those trying to be on their best driving behavior.

2. Methods

2.1. Participants and Apparatus

Twenty-seven healthy, right-handed adults (12 females, 15 males) with a mean age of 33.2 (SD = 6.1) years participated (Table 1) and signed a consent form after they were given a detailed explanation of the study, in accordance with a protocol approved by the Yale Human Investigation Committee. Participants had no history or diagnosis of any Axis I psychiatric or substance (except nicotine) use disorders; no current use of psychotropic medications; no significant current medical conditions including neurological, cardiovascular, endocrine, renal, hepatic, or thyroid disorders. Women who were pregnant or breast-feeding were not recruited. Participants tested negative for cocaine, amphetamines, opioids, and benzodiazepines prior to their fMRI. No dietary restrictions were set.

All participants were tested in two fMRI scan sessions on separate days, in a randomly assigned order (standard/reward or reward/standard), which was counter-balanced across subjects. Participants were briefly trained on the task prior to their first fMRI session to ensure that they understood the task. They were paid \$65 for their participation in the standard session and an average of \$65 for the reward session (compensation scheme detailed in 2.3). Each session was comprised of four runs of approximately 100 trials. The computer-controlled visual display was viewed via a mirror system inside the scanner. Each participant responded with their right index finger via a hand-held button box.

2.2. Standard SST condition

A trial sequence is depicted in Figure 1 (Chao et al., 2009; Duann et al., 2009; Li et al., 2006; 2009). On a typical go trial a light gray fixation dot is displayed at the center of a homogeneous, dark gray background. Following a random (1–5 s) fore-period (FP), the go signal (a light gray ‘O’) replaces the fixation dot and prompts a fast button press response. The proportion of go:stop trials was approximately 3:1, randomly selected with replacement on every trial. On stop trials, the same sequence of events occurred as in a go trial, except that after a staircase-modulated stop signal delay (SSD, detailed in 2.4) the stop signal (a light gray ‘X’) replaced the circle at fixation. Participants were instructed not to wait to begin their response to the go signal, but to withhold their response if the stop signal appears. Responses were recorded as: go success (G) – if the participant successfully responded within 1 s of the go signal being displayed on a go trial (the exact duration of the allowed response window was unknown to participants); go error (F) – if the participant did not respond on a go trial; stop success (SS) – if the participant inhibited his or her response on a stop trial; or stop error (SE) – if the participant failed to inhibit their response.

Reaction times tend to be slower after a shorter FP (Bertelson and Tisseyre, 1968; Woodrow, 1914) indicating a lower level of motor preparedness. We obtained a FP effect size for each participant in each condition by comparing the GORTs produced for trials with

a 3–5 s FP to those with a 1–3 s FP (Li et al., 2006; Tseng and Li, 2008). Reaction times also tend to be slower following an error, reflecting outcome monitoring and adjustment (Rabbitt, 1966). We obtained a post-error slowing (PES) effect size for each participant in each condition by comparing the GORTs produced in post-error, go success trials to post-success, go success trials.

2.3. Reward SST condition

The sequence of events within trial types and proportions of trial types (i.e. 3:1 proportion of go:stop trials) were the same as in the standard condition. The only difference was that on some trials the fixation, go signal, and stop signal (if a stop trial) were displayed in green. However, participants were told that, instead of a fixed \$65 for the session, they would be compensated \$0.80, regardless of their performance, for each “green” trial. Participants were told that the color of the stimuli was otherwise meaningless and they should just focus on the task. The proportion of gray:green trials (unknown to participants) was approximately 4:1, randomly selected with replacement on every trial. The selection of trial type (go or stop) and color (gray or green) were independent. In the end, participants received approximately the same level of compensation for completing the reward session as they did for the standard session.

The reward uncertainty – not knowing the amount they will be compensated along with the lack of control over the amount of compensation – is intended to increase participants' overall level of arousal. Though instructed to ignore the color of the stimuli, green trials are a continual reminder of the uncertainty of reward. We expect the increased cognitive load associated with the arousal induced in this condition to cause participants to rely to a greater degree on their underlying response tendencies. Further, we suspect that the neural mechanisms of cognitive control may show a differential pattern of activation between Riskys and Conservatives, specifically for stop errors compared to stop successes.

2.4. SSD staircase, critical SSD, and SSRT computation

The stop signal delay or SSD, the time between when the go and stop signals were displayed, started at 200 ms on the first stop trial of each session. The SSD then varied between stop trials according to a staircase procedure. If the participant succeeded (*failed*) at inhibiting his or her response on a stop trial, the SSD increased (*decreased*) by 67 ms on the next stop trial.

Stop signal reaction time (SSRT) is an estimate of the time it takes to stop the prepotentiated go response in the SST. To calculate SSRT, a critical SSD was first computed for each participant, representing the delay required for the participant to successfully withhold a response in half of the stop trials (Levitt, 1971). SSDs across trials were grouped into runs, with each run defined as a monotonically increasing or decreasing series. The critical SSD was the average of the mid-run estimates (median SSDs) of every second run. It was reported that, except for experiments with fewer than 30 trials, the mid-run estimate was close to the maximum likelihood estimate of X_{50} (50% SS in the SST; Wetherill et al., 1966). SSRT was then computed by subtracting the critical SSD from the median GORT (Logan et al., 1984).

2.5. Imaging protocol

Conventional T1-weighted spin-echo sagittal anatomical images were acquired for slice localization using a 3T scanner (Siemens Trio). Anatomical images of the functional slice locations were next obtained with spin-echo imaging in the axial plan parallel to the AC-PC line (the line connecting the anterior and posterior commissures) with TR=300 ms, TE=2.5 ms, bandwidth=300 Hz/pixel, flip angle=60°, field of view=220×220 mm, matrix=256×256,

32 slices with slice thickness=4 mm and no gap. Functional blood oxygenation level dependent (BOLD) signals were then acquired with a single-shot gradient-echo echo-planar imaging (EPI) sequence. Thirty-two axial slices parallel to the AC-PC line covering the whole brain were acquired with TR=2000 ms, TE=25 ms, bandwidth=2004 Hz/pixel, flip angle=85°, field of view=220×220 mm, matrix=64×64, 32 slices with slice thickness=4 mm and no gap. Three hundred images were acquired in each of four runs.

2.6. Spatial preprocessing of brain images

Data were analyzed with Statistical Parametric Mapping (SPM8, Wellcome Trust Centre for Neuroimaging, <http://www.fil.ion.ucl.ac.uk/spm/>). Images from the first five EPI volumes at the beginning of each run were discarded to enable the signal to achieve steady-state equilibrium between RF pulsing and relaxation. Images of each subject were realigned (motion-corrected) and corrected for slice timing. A mean functional image volume was constructed for each subject for each run from the realigned image volumes. These mean images were coregistered with the high resolution structural image and then segmented for normalization to an MNI (Montreal Neurological Institute) EPI template with affine registration, followed by nonlinear transformation (Friston et al., 1995; Ashburner & Friston, 1999). Finally, images were smoothed with a Gaussian kernel of 8 mm at Full-Width at Half-Maximum.

2.7. General linear models

Our first level analysis focused on dissociating stop error (SE) and stop success (SS) trials. To this end, we distinguished between go success (G), go error (F), stop success (SS), and stop error (SE) trials. A statistical analytical design was constructed for each participant using a general linear model (GLM) with the onsets of the go signal from every trial convolved with a canonical hemodynamic response function (HRF) and the temporal derivative of the canonical HRF (Friston et al., 1995b). For G trials, the go signal onset was parametrically modulated by the reaction time and its temporal derivative; for SS and SE trials, the go signal onset was parametrically modulated by the stop signal delay (SSD) and its temporal derivative (Li et al., 2006b). Realignment parameters in all 6 dimensions were also entered in the model. Serial autocorrelation of the time series was corrected by a first degree autoregressive or AR(1) model (Della-Maggiore et al., 2002; Friston et al., 2000). The data were high-pass filtered (1/128 Hz cutoff) to remove low-frequency signal drifts. The GLM estimated the component of variance that could be explained by each of the regressors. A contrast of SE>SS was made from the GLM for individual subjects.

In group-level, random effect analyses (Penny and Holmes, 2007), we categorized participants according to the change of go trial reaction time (GORT) in the reward as compared to standard SST. Twelve participants (the Conservatives) increased in GORT (94 ± 68 ms) and 15 participants (the Riskys) decreased in GORT (77 ± 46 ms) in the reward as compared to standard SST. In a flexible factorial model, we included condition (reward vs. standard) as a within-subject factor and change in response strategy (Conservative vs. Risky) as a between-subjects factor. Demographic variables did not differ significantly between groups (Table 1).

Regional activations were identified from the whole brain analysis at $p < .005$, uncorrected (50 voxels in the extent of activation). The regions of interest (ROIs) were localized using an atlas (Maldjian et al., 2003; Duvernoy, 2003). The effect size of SE>SS for each ROI were derived for each participant using MarsBaR (Brett et al., 2002).

3. Results

3.1 Behavioral performance

Behavioral measures observed for each response strategy group (Risky/Conservative) in each condition (Standard/Reward) are shown in Table 2 along with p values corresponding to main and interaction effects from a repeated measures analysis of variance (ANOVA) with condition as a within-participant and group as a between-participant variable. Participants' go trial success rate (GO%) was near perfect in both the standard and reward conditions for both Riskys and Conservatives. Participants' equivalent GO% performance suggests that they were engaged in the task and maintained vigilance throughout the sessions. There was a trend toward a significant difference in stop success rate (STOP%) between conditions, but not between groups, with correction for multiple comparisons. The difference in STOP% may indicate that participants overall monitor more carefully for stop signal in the reward condition. Taking these small deviations due to response strategy into account, our staircase procedure effectively tracked participants' performance as expected, yielding approximately 50% successful stop trials.

The significant interaction effect in GORT reflects Risky/Conservative group selection based on faster (Risky) versus slower (Conservative) GORTs in the reward condition (Fig. 2). Additionally, there is a significant interaction effect in FP effect, such that the FP effect was higher in the faster condition for each group (i.e. reward for Riskys and standard for Conservatives). The reversed patterns seen between GORT and the fore-period effect demonstrate the tendency for less motor preparedness (or higher fore-period effect) to be associated with faster reaction times. SSRT and PES were not significantly different between groups or conditions.

3.2 Whole Brain Analyses

The contrast $[(SE>SS)_{CONS}>(SE>SS)_{RISK}]$ (contrast A, Table 3; Fig. 3), reflecting the main effect of response strategy, revealed clusters in left somatosensory cortex, retrosplenial cortex extending to parahippocampal gyrus, right presupplementary motor area (preSMA), and left thalamus, at $p<.005$, uncorrected, and 50 voxels in the extent of activation. The opposite contrast $[(SE>SS)_{RISK}>(SE>SS)_{CONS}]$, as well as contrasts reflecting the main effect of response strategy and interaction effects between response strategy and condition, did not reveal significant regional activations at the same threshold. This compound contrast yielded a composite of activations that can potentially be attributed to increased activation during SE versus SS for Conservatives – $(SE>SS)_{CONS}$, as compared to Riskys. Alternatively, this interaction may be driven by increased activation during SS versus SE in the Riskys, as compared to Conservatives – $(SS>SE)_{CONS}$. To distinguish between these two scenarios, we plotted the hemodynamic responses of the ROIs during SS and SE trials, separately for Riskys and Conservatives (Fig. 4). The average effect sizes of $SE>SS$ for Riskys and Conservatives in each ROI are plotted in Figure 5 (see also Table 4a).

We further examined whether these changes reflect specifically the influence of the mental set – the induced change in response strategy – and not differences in GORT without the manipulation of this contextual information. To this end we divided the subjects into two groups on the basis of a median split of their mean GORT during the standard session. We computed the effect sizes of $SE>SS$, which did not differ between the two groups (Table 4b).

Also, an additional flexible factorial model replaced the response strategy group factor (i.e., change in GORT) with another performance factor – change in SSRT. Participants were categorized as “SSRT Decreasers” or “SSRT Increaseers” in the reward compared to the

standard condition. Contrasts reflecting the main effect of change in performance under reward uncertainty did not reveal significant regional activations at $p < 0.005$, uncorrected.

4. Discussion

The current findings support the hypothesis that the cerebral processes of cognitive control are influenced by mental sets. Specifically, individuals differ in how they change response strategy under a condition of reward uncertainty; some become more risky by speeding up, while some become more conservative by slowing down, in go trial reaction time. These risky and conservative mental sets influence the cerebral activations of conflict and error processing in cognitive control. Conservatives show greater error-related (SE>SS) activations in the somatosensory cortex, retrosplenial cortex, preSMA and thalamus, as compared to Riskys. In particular, retrosplenial cortical activation reverses by responding more to SS than SE trials for Riskys. Also, these changes in cerebral activations reflect specifically the differences in mental set or response strategy irrespective of response speed, as a comparison between fast and slow responders during the standard condition do not show these changes.

Greater activation of the retrosplenial cortex, thalamus, and preSMA during SE, compared with SS, is consistent with earlier work implicating these structures in error detection and feedback processing (Christoffels et al., 2007; Hendrick et al., 2010; Hester et al., 2004; Ide & Li, 2011a; Li et al., 2008b; Li et al., 2006; Tsukamoto et al., 2006; Wrase et al., 2007; Zhang et al., in press; Zhang and Li, in press). Greater activity in the retrosplenial cortex might reflect differential affective responses during errors, as compared with successfully resolved conflicts (Critchley, 2005; Maddock, 1999; Phan et al., 2004). Conservatives' relatively cautious behavior in the reward condition, trading speed for accuracy, suggests stop errors would be associated with higher salience and/or greater negative affect compared to Riskys, akin to the same experience associated with running a red light for the person driving under a conservative, in contrast to a risky, mental set. For the Riskys, who traded accuracy for speed, the saliency of stop errors diminished or even reversed, as compared to stop successes, reflecting what the driver may experience in deciding to stop at the traffic light, despite the primary goal of arriving as soon as possible.

Riskys and Conservatives both show greater error-related responses in preSMA and thalamus, consistent with these structures' role in error processing (Ide & Li, 2011b). Optimizing task performance requires outcome monitoring and post-error adjustment. That Conservatives' response strategy is aimed at minimizing errors suggests that Conservatives would also be more motivated to engage in outcome monitoring in an attempt to optimize their performance.

We also considered an alternative explanation for greater SE as compared to SS responses in the preSMA and thalamus in the Conservatives than Riskys. Our thalamus cluster appeared to cover bilateral subthalamic nucleus (STN, Li et al., 2008a). Response conflict is inherent in the SST, and must be resolved in order to decide whether and when to respond. The enhanced activations for Conservatives may reflect greater conflict associated with initiating a response compared to Riskys. Frank et al.'s (2007) neural network model proposed a mechanism of cognitive control that supports delayed responding. Based on the finding that deep brain stimulation of STN abolished conflict-induced slowing in Parkinson's disease patients, the model posits reciprocal activations between STN and thalamus as a mechanism that mediates go response selections. Specifically, response delays are driven by initial sustained activation in STN, and, when/if STN activity decreases, global inhibition is released, and thalamus activates, facilitating a go response selection through connections with preSMA. The go response is prepotentiated in the SST. It seems plausible that Riskys,

who exhibit a bias toward optimizing the speed rather than the accuracy of their responses, would have less conflict in deciding to go than Conservatives. Thus, a relatively small thalamic activation is sufficient to facilitate a go response in Riskys, whereas Conservatives yield a much greater activation associated with overcoming their go response conflict.

PreSMA's suggested role in resolving response conflict is also outlined by Frank et al. (2007). A direct projection to STN contributes to initial global inhibition, and further, preSMA maintains potential motor response choices, and incorporates information received from direct connections with somatosensory cortex and thalamus to select an appropriate response. Again for Riskys, a relatively small activation sufficiently represents these processes associated with resolving response conflict. Conservatives' much greater activation in preSMA, as with the thalamus, is associated with the greater relative incompatibility of selecting the go response choice under a conservative mental set. Altogether, both error monitoring and conflict resolution may contribute to Conservatives' increased error-related activations in preSMA and thalamus.

A few limitations of the study should be considered. First, perhaps because of the small sample size, the results are significant only at an uncorrected threshold and thus should be considered as preliminary. Second, it would have been informative to assess participants on a risk-prone personality trait and examine whether the change in response strategy between conditions is related to individual differences in personality trait. Third, it would have provided additional information, if participants were monitored for physiological arousal (for instance, with measurements of galvanic skin responses), to examine the relationship between arousal, effort, and the mental sets (Zhang and Li, in press).

In sum, we demonstrated that uncertain reward, a context that does not involve explicit instruction about response strategy, can induce changes in mental sets. Such changes in mental sets varied between individuals and altered cerebral processing of salient events and, potentially, of conflict resolution. These findings are specific to the influence of reward uncertainty rather than relative speed, as was manipulated typically by explicit instructions in previous studies. Overall, the current findings extended the potential utility of the stop signal task in elucidating contextual effects on cognitive control.

Acknowledgments

This study was supported by NIH grants R01DA023248 and K02DA026990. The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institute of Drug Abuse or the National Institutes of Health. We thank Drs. Jaime Ide and Sheng Zhang for their helpful discussions.

Abbreviations

SST	stop signal task
SSD	stop signal delay
G	go success trial
F	go failure trial
SS	stop success
SE	stop error
SSRT	stop signal reaction time
GLM	general linear model
HRF	hemodynamic response function

REFS	random effects analyses
GORT	go trial reaction time
REF	flexible factorial model
ROIs	regions of interest
ANOVA	analysis of variance
GO%	go trial success rate
STOP%	stop trial success rate
FP effect	fore-period effect
PES	post-error slowing
CONS	“Conservatives”
RISK	“Riskys”
preSMA	presupplementary motor area
STN	subthalamic nucleus

5. References

- Ashburner J, Friston KJ. Nonlinear spatial normalization using basis functions. *Hum. Brain Mapp.* 1999; 7:254–266. [PubMed: 10408769]
- Bertelson P, Tisseyre F. The time-course of preparation with regular and irregular foreperiods. *Quat J Exp Psychol.* 1968; 20:297–300.
- Brett, M.; Anton, J-L.; Valabregue, R.; Poline, J-P. Region of interest analysis using an SPM toolbox. Abstract presented at the 8th International Conference on Functional Mapping of the Human Brain; Sendai, Japan. June 2–6; 2002. Available on CD-ROM in *Neuroimage*, 16(2), abstract 497
- Bogacz R, Wagenmakers EJ, Forstmann BU, Nieuwenhuis S. The neural basis of the speed-accuracy tradeoff. *Trends Neurosci.* 2010; 33:10–16. [PubMed: 19819033]
- Chao HH, Luo X, Chang JL, Li C-SR. Activation of the pre-supplementary motor area but not inferior prefrontal cortex in association with short stop signal reaction time—an intra-subject analysis. *BMC Neurosci.* 2009; 10:75. [PubMed: 19602259]
- Christoffels IK, Formisano E, Schiller NO. Neural correlates of verbal feedback processing: an fMRI study employing overt speech. *Hum. Brain Mapp.* 2007; 28:868–879. [PubMed: 17266104]
- Critchley HD. Neural mechanisms of autonomic, affective, and cognitive integration. *J. Comp. Neurol.* 2005; 493:154–166. [PubMed: 16254997]
- Della-Maggiore V, Chau W, Peres-Neto PR, McIntosh AR. An empirical comparison of SPM preprocessing parameters to the analysis of fMRI data. *Neuroimage.* 2002; 17:19–28. [PubMed: 12482065]
- Duann JR, Ide JS, Luo X, Li CS. Functional connectivity delineates distinct roles of the inferior frontal cortex and presupplementary motor area in stop signal inhibition. *J Neurosci.* 2009; 29(32):10171–9. [PubMed: 19675251]
- Duvernoy, HM. *The human brain*, second edition. Springer-Verlag; Wien (Austria): 2003.
- Eysenck, MW. *Attentional Control Theory of Anxiety: Recent Developments*. Springer; New York, NY: 2010.
- Frank MJ, Samanta J, Moustafa AA, Sherman SJ. Hold your horses: impulsivity, deep brain stimulation, and medication in parkinsonism. *Sci.* 2007; 318:1309–12.
- Friston KJ, Ashburner J, Frith CD, Poline JB, Heather JD, Frackowiak RSJ. Spatial registration and normalization of images. *Hum. Brain Mapp.* 1995a; 3:165–189.
- Friston KJ, Holmes AP, Worsley KJ, Poline J-B, Frith CD, Frackowiak RSJ. Statistical parametric maps in functional imaging: a general linear approach. *Hum. Brain Mapp.* 1995b; 2:189–210.

- Friston KJ, Josephs O, Zarahn E, Holmes AP, Rouquette S, Poline J. To smooth or not to smooth? Bias and efficiency in fMRI time-series analysis. *Neuroimage*. 2000; 12:196–208. [PubMed: 10913325]
- Gerrig, RJ.; Zimbardo, PG. *Psychology and Life*. Allyn and Bacon; Boston, MA: 2002.
- Hendrick OM, Ide JS, Luo X, Li CS. Dissociable processes of cognitive control during error and non-error conflicts: a study of the stop signal task. *PLoS One*. 2010; 5(10):e13155. [PubMed: 20949134]
- Hester R, Fassbender C, Garavan H. Individual differences in error processing: A review and reanalysis of three event-related fMRI studies using the Go/NoGo task. *Cereb. Cortex*. 2004; 14:986–994. [PubMed: 15115734]
- Ide JS, Li C-SR. A cerebellar thalamic cortical circuit for error-related cognitive control. *Neuroimage*. 2011a; 54:455–464. [PubMed: 20656038]
- Ide JS, Li C-SR. Error-related functional connectivity of the habenula in humans. *Front. Hum. Neurosci*. 2011b; 5:25. [PubMed: 21441989]
- Levitt H. Transformed up-down methods in psychoacoustics. *J. Acoust. Soc. Am*. 1971; 49(Suppl 2): 467–477. [PubMed: 5541744]
- Li C-SR, Chao HH, Lee TW. Neural correlates of speeded as compared with delayed responses in a stop signal task: an indirect analog of risk taking and association with an anxiety trait. *Cereb. Cortex*. 2009; 19:839–848. [PubMed: 18678764]
- Li C-SR, Huang C, Constable RT, Sinha R. Imaging response inhibition in a stop-signal task: neural correlates independent of signal monitoring and post-response processing. *J. Neurosci*. 2006; 26:186–192. [PubMed: 16399686]
- Li C-SR, Yan P, Sinha R, Lee TW. Subcortical processes of motor response inhibition during a stop signal task. *Neuroimage*. 2008a; 41:1352–1363. [PubMed: 18485743]
- Li C-SR, Huang C, Yan P, Paliwal P, Constable RT, Sinha R. Neural correlates of post-error slowing during a stop signal task: a functional magnetic resonance imaging study. *J. Cogn. Neurosci*. 2008b; 20:1021–1029. [PubMed: 18211230]
- Logan GD, Cowan WB. On the ability to inhibit thought and action: A theory of an act of control. *Psychol. Rev*. 1984; 91:295–327.
- Lupien SJ, Maheu F, Tu M, Fiocco A, Schramek TE. The effects of stress and stress hormones on human cognition: Implications for the field of brain and cognition. *Brain and Cogn*. 2007; 65:209–237.
- Maddock RJ. The retrosplenial cortex and emotion: new insights from functional neuroimaging of the human brain. *Trends Neurosci*. 1999; 22:310–316. [PubMed: 10370255]
- Maldjian JA, Laurienti PJ, Kraft RA, Burdette JH. An automated method for neuroanatomic and cytoarchitectonic atlas-based interrogation of fMRI data sets. *Neuroimage*. 2003; 19:1233–1239. [PubMed: 12880848]
- Padmala S, Lim SL, Pessoa L. Pulvinar and Affective Significance: Responses Track Moment-to-Moment Stimulus Visibility. *Front Hum Neurosci*. 2010 Sep 24;4. pii: 64.
- Padmala S, Pessoa L. Reward Reduces Conflict by Enhancing Attentional Control and Biasing Visual Cortical Processing. *J Cogn Neurosci*. in press.
- Pessoa L. How do emotion and motivation direct executive control? *Trends in Cogn. Sci*. 2009; 13:160–166. [PubMed: 19285913]
- Pessoa L, Padmala S, Kenzer A, Bauer A. Interactions between cognition and emotion during response inhibition. *Emotion*. in press.
- Penny, WD.; Holmes, AJ. Random Effects Analysis. In: Friston, KJ.; Ashburner, JT.; Kiebel, SJ.; Nichols, TE.; Penny, WD., editors. *Statistical Parametric Mapping*. Academic Press; San Diego, USA: 2007. p. 156–165. Academic Press
- Phan KL, Wager TD, Taylor SF, Liberzon I. Functional neuroimaging studies of human emotions. *CNS Spectr*. 2004; 9(4):258–266. [PubMed: 15048050]
- Rabbitt PMA. Errors and error correction in choice-response tasks. *J Exp Psychol*. 1966; 71:264–272. [PubMed: 5948188]

- Tseng YC, Li C-SR. The effects of response readiness and error monitoring on saccade countermanding. *The Open Psychol J.* 2008; 1:18–25.
- Tsukamoto T, Kotani Y, Ohgami Y, Omura K, Inoue Y, Aihara Y. Activation of insular cortex and subcortical regions related to feedback stimuli in a time estimation task: an fMRI study. *Neurosci. Lett.* 2006; 399:39–44. [PubMed: 16490307]
- Verbruggen F, Logan GD. Proactive adjustments of response strategies in the stop-signal paradigm. *J. Exp. Psychol. Hum. Percept. Perform.* 2009; 35:835–854. [PubMed: 19485695]
- Wetherill GB, Chen H, Vasudeva RB. Sequential estimation of quantal response curves: A new method of estimation. *Biometrika.* 1966; 53:439–454.
- Woodrow H. The measurement of attention. *Psychol Monogr.* 1914; 17:1–158.
- Wrase J, Kahnt T, Schlagenhauf F, Beck A, Cohen MX, Knutson B, Heinz A. Different neural systems adjust motor behavior in response to reward and punishment. *Neuroimage.* 2007; 36:1253–1262. [PubMed: 17521924]
- Zhang S, Ide JS, Li C-SR. Resting-State Functional Connectivity of the Medial Superior Frontal Cortex. *Cereb Cortex.* 2012; 22:99–111. [PubMed: 21572088]
- Zhang S, Li C-SR. Functional networks for cognitive control in a stop signal task: Independent component analysis. *Hum Brain Mapp.* 2012; 33:89–104. [PubMed: 21365716]
- Zhang S, Hu S, Chao HH, Luo X, Farr OM, Li C-SR. Cerebral correlates of skin conductance responses in a cognitive task. *NeuroImage.* in press.

Highlights

We use reward uncertainty to elicit risk-seeking & risk-averse mental sets.

Stop errors are more salient for *Riskys* than *Conservatives*.

Stop successes yield a greater affective response in *Conservatives* than *Riskys*.

Fig. 1.

Trial sequence for go and stop trials in the stop signal task. The fixation dot is displayed at the beginning of each trial. After a variable (1–5 sec.) fore-period an imperative go signal 'O' is displayed, compelling the participant to respond quickly. On go trials (upper sequence) nothing more is displayed and the trial is recorded as a go success (G) if the participant responds within the time window, or a go error (F) if they do not. On stop trials (bottom sequence) a stop signal 'x' is displayed following a staircase-varied stop signal delay which requires withhold a response. A successful inhibition is recorded as a stop success (SS) and a failed trial as a stop error (SE).

Fig. 2.

GORT and FP effect size as a function of response strategy and condition. The circles plotted above each response strategy/condition combination represents the group mean GORT (top panel) and FP effect size (bottom panel). Error bars indicate the standard deviation. GORT: go trial reaction time; FP effect: fore-period effect.

Fig. 3.

Activations from $[(SE>SS)_{CONS}>(SE>SS)_{RISK}]$. BOLD contrasts from our second level flexible factorial analysis comparing differential (SE>SS) activations between participants increasing vs. decreasing in GORT, CONS>RISK ($p<.005$, uncorrected, 10 voxels extent) are superimposed on a T1 structural image in axial sections from $z=-30$ to $z=+80$. Adjacent sections are 5mm apart. Color bar represents voxel T value. Neurological orientation: right=right

Fig. 4.

Mean hemodynamic response (HDR) function by response strategy and stop trial outcome (SS and SE). The solid blue HDR function represents activation related to Conservatives' SE trials across 30 s following presentation of the go signal. The thinner blue '+', the dashed red, and the thinner red-starred functions correspond to Conservatives' SS, Riskys' SE, and Riskys' SS trials, respectively. Conservatives yield much greater SE than SS activation in (a) somatosensory cortex, (b) retrosplenial cortex, (c) preSMA, and (d) thalamus. Riskys show relatively diminished error-related activations in preSMA and thalamus and the reverse pattern in retrosplenial cortex, with greater SS than SE activation, seen to a lesser extent in somatosensory cortex.

Fig. 5. Mean Risky and Conservative SE>SS contrast effect sizes by ROI. Effect sizes reflect the differences between average SE and SS trial parameter estimates in all ROIs significant at $p < .005$, uncorrected (50 voxels extent).

Table 1

Participants' demographic data.

Group	n	Gender (%male)	Age (years)	Education (years)
Risky:	15	46.7	34.27 (5.38)	15.27 (1.39)
Conservative:	12	66.7	31.92 (6.84)	15.0 (2.13)
P values:		0.870	0.327	0.698

Note: Data presented as a % for gender (categorical) or Mean (SD). P values were derived using chi-squared analyses for categorical data or t-tests.

Table 2

SST performance grouped by condition (standard vs. reward).

Condition	Correct GO%	Correct STOP%	Median GORT (ms)	FP (effect size)	PES (effect size)	SSRT (ms)
Risky:						
Standard	96.2 (1.7)	52.2 (4.1)	667 (112)	2.07 (1.7)	0.91 (1.9)	205 (35)
Reward	97.1 (1.8)	53.2 (3.3)	590 (90)	2.27 (0.9)	0.69 (1.1)	218 (48)
Conservative:						
Standard	97.0 (1.9)	49.7 (2.9)	559 (135)	2.24 (0.8)	1.44 (2.1)	199 (28)
Reward	96.5 (1.7)	52.4 (2.1)	653 (125)	1.10 (1.0)	1.20 (1.6)	207 (43)
P values:						
Risk/Cons	0.830	0.112	0.601	0.190	0.333	0.461
Std/Rew	0.403	0.031*	0.933	0.135	0.556	0.270
Interaction	0.060	0.272	< 0.001*	0.015*	0.981	0.818

Note: Behavioral measures are presented Mean (SD). P values were derived using a repeated measures ANOVA with condition as the within-participant variable and response strategy group as the between-participant variable. SSRT: stop signal reaction time; GORT: go trial reaction time; GO%: go trial success rate; STOP%: stop trial success rate; PES: post-error slowing; FP effect: fore-period effect.

Table 3

Neural correlates of error processing.

Contrast and region	MNI Coordinates				Cluster size (voxels)	Z-value
	x	y	z	Side		
(A) (SE > SS) _{cons} > (SE > SS) _{RISK}						
Somatosensory cortex	-63	-22	40	L	124	3.56
Retrosplenial cortex/ parahippocampal gyrus	24	-49	10	R	102	3.53
PreSMA	6	8	55	R	91	3.38
Thalamus	-12	-16	1	L	123	3.18
(B) (SE > SSW) _{RISK} > (SE > SS) _{cons}						
n/a						

Note: All regions of interest are significant at (p<.005, uncorrected, 50 voxels extent). Second level contrasts comparing the neural correlates of error processing (a) between Conservatives (CONS) who produced slower GORTs in their reward than their standard session vs. Riskys (RISK) who produced relatively faster GORTs in their reward session. The opposite contrast (b) error processing for RISK>CONS yielded no significant ROIs. MNI: Montreal Neurological Institute.

Table 4a

Mean error-related (SE > SS) effect sizes by response strategy group

Group	Somatosensory	Retrosplenial/ Parahippocampal	PreSMA	Thalamus
Risky	−0.03 (1.57)	−0.53 (1.21)	0.14 (1.25)	0.42 (1.08)
Conservative	1.90 (1.33)	1.12 ± (0.96)	1.75 (1.09)	1.81 (1.55)
P values:	< 0.01*	< 0.001*	< 0.01*	0.01*

Note: Effect sizes are presented Mean (SD). P values are results of effect size comparisons using two sample t-tests: between Riskys and Conservatives (collapsed across condition).

Table 4b

Mean error-related (SE > SS) effect sizes by overall speed group

Group	Somatosensory	Retrosplenial/ Parahippocampal	PreSMA	Thalamus
Fast	0.78 (1.71)	0.68 (1.55)	0.99 (1.65)	0.71 (1.84)
Slow	0.94 (2.41)	−0.32 (1.68)	0.71 (1.95)	0.90 (1.55)
P values:	0.85	0.12	0.69	0.77

Note: Effect sizes are presented Mean (SD). P values are results of effect size comparisons using two sample t-tests: between groups of participants who exhibited faster versus slower standard GORTs (standard condition only).