

## **Beyond decision! Motor contribution to speed-accuracy trade-off in decision-making**

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## Beyond decision! Motor contribution to speed-accuracy trade-off in decision making

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**Abstract** Both in real life and experimental settings, increasing response speed typically leads to more error-prone actions. Processes underlying such a “speed-accuracy trade-off” (SAT) are usually assumed to be purely decisional: cautiousness would be determined only by the amount of sensory evidence required to select a response. The present data challenges this largely accepted view, by directly showing that motor processes are speeded up under time pressure. In a choice reaction time task where emphasis was put either on response speed or accuracy, motor processes were investigated through the analysis of muscular activity related to response execution. When response speed was emphasized, the time between electromyographic onset and behavioral response (motor time) was also speeded up (contributing for more than 20 % of the total effect on global reaction time). This speeded execution (likely due to a more efficient motor command) may also explain why participants are less able to interrupt incorrect response execution once started (Burle et al 2014), leading to more overt errors. Pointing to a speed-accuracy exchange within motor processes themselves, the present results call for a re-evaluation of widely accepted assumptions about SAT, and more generally, decision-making processes. They are discussed in the context of recent extensions of the drift diffusion model framework, questioning the strict separation between decisional and motor processes.

**Keywords** Decision-making · Speedaccuracy trade-off · Electrophysiology

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### Introduction

In everyday life, the accuracy of a decision largely depends on its speed: fast decisions tend to be less accurate than slower more thoughtful ones (Heitz 2014). Such a “speed-accuracy trade-off” (SAT) is pervasive, being present in a wide range of species, from drosophila (Das-Gupta et al 2014) to humans and in virtually all cognitive abilities (see Heitz 2014, for an overview). Understanding how displacements along the speed-accuracy trade-off are achieved is of major importance for the comprehension of decision-making processes, and information processing in general. Lately, SAT has received a regain of interest thanks to monkey neurophysiology data in relation to sequential sampling models, conceptualizing decision making as an accumulation of evidence in favor of competing alternatives (Brunton et al 2013; Heitz and Schall 2012; Ratcliff and Smith 2004): A decision is made as soon as the amount of evidence accumulated for one of the alternatives has reached a decision threshold (Ratcliff and Smith 2004). Varying the distance between accumulation starting point and decision threshold naturally accounts for SAT: a high decision threshold requires more evidence and hence leads to slow but more accurate responses, while a low decision threshold results in premature, more error-prone responses. This led to the widely accepted view that SAT affects only decision processes, and spares more perceptual and motor stages (Ratcliff and Rouder 1998; Reddi and Carpenter 2000; Bogacz et al 2009). This point of view was further reinforced by several fMRI studies (Ivanoff et al 2008; Forstmann et al 2008; van Veen et al 2008) reporting that instructions to emphasize response speed led to an increased BOLD activity of cortical areas associated with decision processes (such as the pre-Supplementary Motor Area), but not of sensory or primary motor areas.

Recently, however, single cell recordings in monkeys showed that the rate of accumulation, as indexed by neuronal mean firing rate, is also affected by SAT (Heitz and Schall 2012; Hanks et al 2014), casting doubt on a pure

decision-threshold effect. Furthermore, some recent modeling studies in humans have reported that the duration of non-decision processes (represented by  $T_{er}$  parameter in accumulator models) sometimes needs to vary to account for the differences observed between speed and accuracy conditions (Zhang and Rowe 2014; Mulder et al 2013; Dambacher and Hübner 2015; White et al 2011, see also Voss et al 2004 for a former study), suggesting a contribution of non-decision processes to SAT. However, as  $T_{er}$  models the compound duration of both pre- and post-decisional processes, it is impossible to determine whether sensory, motor or both processes are affected.

Few (older) studies suggested a motor locus of SAT effects. For example, it has been shown that response force is stronger when speed is emphasized (Jakowski et al 1994, 2000). At the cortical level, the response-locked lateralized readiness potential (LRP<sup>1</sup>) has been reported to be shorter under speed stress (Osman et al 2000; Rinkenauer et al 2004). This suggests that activity of primary motor cortices (M1s) is shorter in speed conditions, which has been interpreted as an impact of SAT on the duration of post-decisional processes. Such an interpretation however holds only under the assumption that M1 activation starts after decision stages have ended. This view has recently been questioned by several authors arguing that part of the decision to act is performed within the structures involved in the action execution, namely the motor structures (M1s: Donner et al 2009; O’Connell et al 2012; Kelly and O’Connell 2103; Frontal eye fields FEFs: Gold and Shadlen 2000; Purcell et al 2010, 2012, for examples).

If, at least part of the decision to act is performed within M1, the onset of response-locked LRP does not correspond to the post-decisional stage, but actually to the beginning of the action decision making process in M1. In such a case, a SAT effect on response-locked LRP is not an argument for a motoric effect.

To clarify this point, we investigated the impact of time pressure on neurophysiological measures that unambiguously index the motor component of the reaction process. To do so, we fractioned reaction time of *each trial* into two sub-intervals (Burle et al 2002) based on the responding muscles’ electromyographic (EMG) activity: premotor time (PMT), from stimulus presentation until response-related EMG onset, and motor time (MT), from EMG onset to mechanical response (Figure 1).

The MT provides a direct measure of the duration of execution processes, and has the advantage to be measurable on a trial-by-trial basis. As such, it does not suffer the distortions induced by averaging and has previously

proved to be efficient to reveal modulations of late motor processes (see *e.g.* (Possamaï et al 2002; Burle et al 2002; Hasbroucq et al 1995; Tandonnet et al 2003)). To further characterize potential SAT effects on motor processes, we also examined the shape of the response EMG burst. We analyzed both the surface under the EMG burst and the slope of mean EMG profiles, respectively linked to the strength and the efficiency of the motor command.

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## Method

### Participants

Sixteen participants (5 women, 11 men, ages 18-50) participated in this experiment. The necessary number of participants had to be a multiple of 8 (see below for counterbalancing), and was set to 16 in the current study. All participants had normal (or corrected to normal) vision, and gave their informed consent.

### Apparatus

The participants were seated in a dark room, facing a panel made of 5 digit presentation devices (model LTS-3401LP LITE ON, rise onset time < 1 ms) composed of seven-segment Light-Emitting Diodes on which the response signals (the letters “H” or “S”) were presented. The whole display was contained in a 1.4° visual angle. The responses were given by thumb key-presses of the right or the left hand on response micro-switch located under each response button. The electromyographic activity of the *flexor pollicis brevis* of both hands was recorded with two electrodes placed 2 cm apart on the thenar eminences. This activity was amplified, filtered (low/high frequency cut-off at 10 Hz/1kHz), and digitized on-line (A/D rate 2 kHz). The EMG signal was continuously monitored by the experimenter in order to avoid any background activity that could prevent reliable detection of EMG onset. If the signal became noisy, the experimenter immediately asked the participant to relax his/her muscles. In the end, less than 2 % of trials were too noisy for proper EMG detection and were excluded from analyses.

### Procedure

The central digit presentation device (the target) displayed the response signal (“H” or “S”). The four other devices, flanking the target, were distractors (Eriksen and Eriksen 1974). Distractors were either a replication of the target (“HHHHH” or “SSSSS”, compatible trials) or a replication of the alternative response signal (“HSHSH” or “SSHSS”, incompatible trials). The four

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<sup>1</sup> LRP is obtained by subtracting the mean EEG signal obtained on electrodes ipsilateral to a unimanual response from the mean signal obtained on electrodes contralateral to the response. Representing the difference of activity between the two motor cortices, it is classically considered as an index of motor preparation.

types of stimuli were equiprobable, and the first-order sequential effects for the trial-to-trial transition were balanced. In tasks like this, faster and more accurate responses are commonly observed in compatible compared to incompatible trials (see *e.g.* Kornblum et al 1990, for a review).

Each participant took part in two experimental sessions that comprised 10 blocks of 64 trials. In each session the participants were either asked to respond very accurately, (with the cost of reaction time lengthening, “ACC” instruction) or to respond very fast (with the cost of more errors, “SPD” instruction). SAT was thus manipulated solely by verbal instruction.

Half of the participants received the ACC instruction during the first session, whereas the other half received the SPD instruction during the first session. In each subgroup, the mapping between the target letters and the buttons was counterbalanced across participants.

## Evaluation of Motor processes

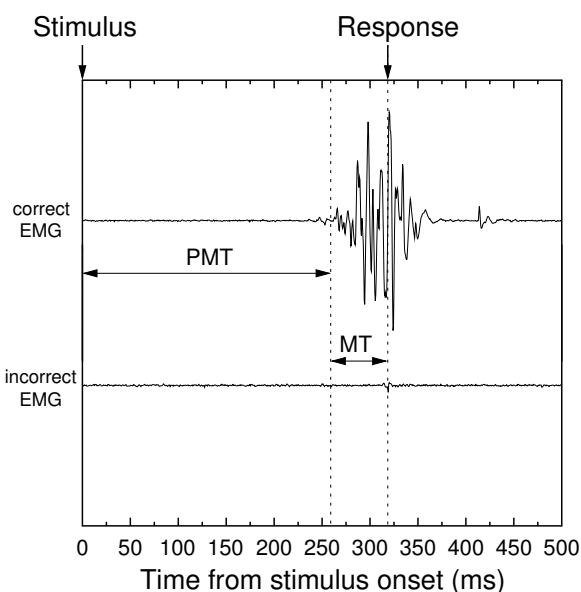
### Reaction time fractionating

EMG traces were inspected visually and the EMG onsets were hand-scored (human pattern recognition, although more time consuming, is superior to automated algorithms, Staude 2001). It should be emphasized that the experimenter was unaware of the type of trial he was looking at<sup>2</sup>. In some trials, several EMG bursts are visible (see Burle et al 2002, 2014 for a more detailed description of EMG trial types). In the present study, we focused on correct trials showing only the EMG burst related to the correct response (*i.e.*, “pure-correct” trials). Based on EMG onsets, the reaction time (RT) of each pure-correct trial was fractionated into premotor time (PMT), corresponding to the interval between stimulus occurrence and EMG onset and motor time (MT), the time between the EMG onset and the response (see Figure 1). We then analyzed the effect of speed instructions and trial compatibility, both known to impact global RT, on each of the motor and premotor time intervals.

### EMG signal analysis

The impact of speed instruction on the shape of the EMG burst was investigated through the analysis of both the

<sup>2</sup> The experimenter saw the two EMG traces plotted on a computer screen, along with two vertical lines indicating the moment of the stimulus and of the response. However, no indication of the nature of the trial – *i.e.* no stimulus code – was provided. Besides, we also ran a fully automatic EMG onset detection, using the “EMGOnsetDetection” solution of BrainVisionAnalyser 2 (default values in the software). Although the algorithm largely over-estimated MT and inflated the within participant MT variance, because of mis-detection of the true onset, the main results were replicated, that is an effect of SAT, but not of compatibility, on MT.



**Fig. 1** EMG signal recorded during an individual trial as a function of time (stimulus occurs at time 0), for both the correct response hand (top), and the incorrect response hand (bottom). In each trial, premotor time is defined as the time interval between stimulus occurrence and EMG burst onset, and motor time is defined as the interval between EMG onset and response occurrence.

surface under the EMG burst and the rising slope. For each participant and each experimental condition, individual EMG activities were rectified and averaged, time locked to the EMG onset. Based on mean profiles, burst surface was determined as the surface under EMG burst in a window from EMG onset to 100 ms after (covering a large part of the EMG burst for all participants). The slope of the rising flank of the burst was estimated through a linear regression computed on a window from 0 to 30 ms after EMG onset.

## Results

All statistical analyses were performed by means of repeated-measures canonical analyses of variance (ANOVA), with partial eta-squared statistics ( $\eta_p^2$ ) reported as a measure of effect size. Percentages of errors were arcsine transformed (Winer 1971) before being submitted to ANOVAs. For the critical results (related to potential motoric effects of SAT), these frequentist analyses were complemented by Bayesian ANOVAs using Jasp (Love et al 2015). Such Bayesian ANOVAs provide the so-called “Bayes factor” (*BF*) which describes the relative probability of data under competing statistical models (for example the presence or absence of an effect, see Rouder et al 2012<sup>3</sup>).

<sup>3</sup> The *BF* can be reported as evidence for the *H1* hypothesis, and will hence be noted *BF*<sub>10</sub>, or as evidence in favor of the null hypothesis *H0*. In this case, it will be noted as *BF*<sub>01</sub>.

For all analyses, the participant was treated as random effect.

### Overall performance and premotor processes

We first report overall performance, without reference to EMG data, for sake of comparison with what is usually reported. Overall mean RT and percentages of errors in each condition are shown in Table 1. As expected, SPD instruction resulted in shorter RTs ( $F(1, 15) = 33.35, p < .001, \eta_p^2 = .69$ ) and higher error rates ( $F(1, 15) = 27.03, p < .001, \eta_p^2 = .64$ ) compared to ACC instruction. A compatibility effect was also observed, with faster RTs ( $F(1, 15) = 71.42, p < .001, \eta_p^2 = .83$ ) and fewer errors ( $F(1, 15) = 178.5, p < .001, \eta_p^2 = .92$ ) in compatible than in incompatible trials. The interaction between the factors instruction and compatibility was not statistically significant for both RTs ( $F(1, 15) = 2.47, p = .14, \eta_p^2 = .14$ ) and error rates ( $F(1, 15) < 1, p = .79, \eta_p^2 = .005$ ).

Restricting the analysis to pure-correct trials reveals essentially the same pattern, with SAT and compatibility effects still significant on RT (respectively  $F(1, 15) = 38.23, p < .001, \eta_p^2 = .72$  and  $F(1, 15) = 25.11, p < .001, \eta_p^2 = .63$ ). The interaction proved significant in this case ( $F(1, 15) = 13.84, p < .05, \eta_p^2 = .48$ ), with the compatibility effect being larger under ACC instruction than under SPD instruction, although still significant in each condition (ACC: 21 ms;  $F(1, 15) = 30.80, p < .001, \eta_p^2 = .67$ ; SPD: 9 ms;  $F(1, 15) = 9.67, p < .05, \eta_p^2 = .39$ ). Pure-correct premotor times (Table 1) showed the same pattern, being shorter under SPD than ACC instruction ( $F(1, 15) = 39.38, p < .001, \eta_p^2 = .72$ ), and shorter in compatible compared to incompatible trials ( $F(1, 15) = 22.11, p < .001, \eta_p^2 = .60$ ). Again, a compatibility effect was present for the two instructions (ACC: 21 ms;  $F(1, 15) = 31.82, p < .001, \eta_p^2 = .68$ ; SPD: 8 ms;  $F(1, 15) = 6.23, p < .05, \eta_p^2 = .29$ ), but was stronger in ACC condition ( $F(1, 15) = 17.11, p < .001, \eta_p^2 = .53$ ).

### Evaluation of motor processes

To investigate the impact of SAT modulations on motor processes, we first compared MT duration between the two speed instructions, and then extracted and compared the surface, as well as the rising slope of the EMG bursts.

#### Motor time

MTs for the different conditions are presented in Table 1. MTs were affected by speed instruction: they were 23 ms shorter under SPD than under ACC instruction ( $F(1, 15) = 14.13, p < .01, \eta_p^2 = .49$ ). Compared to an

Importantly, the two are related, since  $BF_{10} = \frac{1}{BF_{01}}$ . Using this notation allows to always provide  $BF$  superior to 1.

Overall performance				
	RT (ms)		Errors (%)	
	ACC	SPD	ACC	SPD
compatible	547	440	5.0	22.7
incompatible	579	466	11.6	36.5
RT fractionating (pure-correct trials)				
	PMT (ms)		MT (ms)	
	ACC	SPD	ACC	SPD
compatible	325	261	106	82
incompatible	346	267	106	84

**Table 1** Top: Mean reaction times (RT) and percentages of errors for both ACC and SPD instructions, in compatible and incompatible trials. Bottom: Mean premotor times (PMT) and motor times (MT) for pure-correct trials under ACC and SPD instructions, in compatible and incompatible trials.

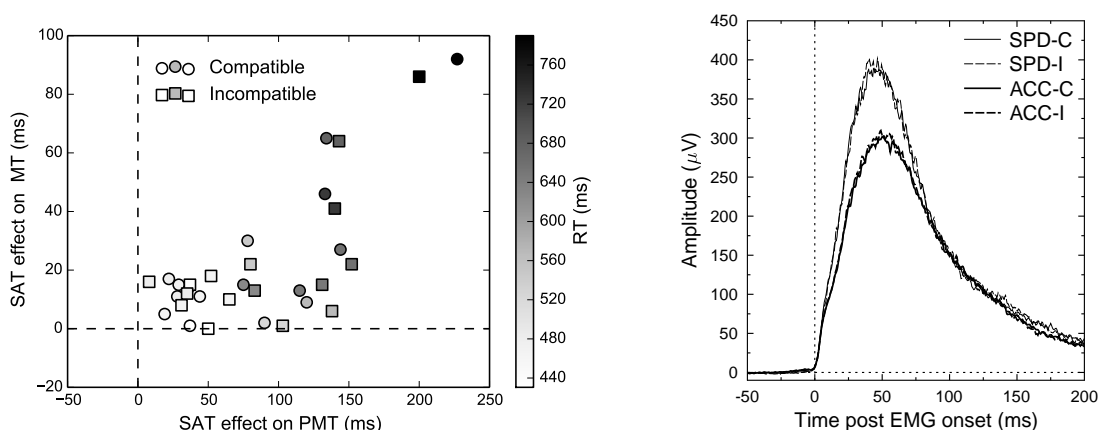
effect of about 110 ms on the whole RT, the effect on MT hence accounts for more than 20% of the overall SAT effect.

Trial compatibility, on the other hand, had a very small numerical effect on MT ( $< 1$ ms), yet marginally significant ( $F(1, 15) = 3.32, p = .09, \eta_p^2 = .18$ ). No interaction between speed instruction and compatibility was found ( $F(1, 15) = 1.43, p = 0.25, \eta_p^2 = .09$ ). Bayesian analyses confirmed the SAT effect on MT: the Bayes Factor ( $BF$ ) in favor of an effect of SAT was very high ( $BF_{10} = 247438$ ), while it tends to favor the absence of compatibility effect on MT, although the evidence is not very strong ( $BF_{01} = 3.7$ ). Hence, speed instruction had a clear impact on the duration of motor processes, while compatibility only had a small numerical effect, the  $BF$  suggesting a slight evidence against an effect (according to current scales for interpreting  $BF$ , see *e.g.* Jeffreys 1961). This contrasts with modulations of the duration of premotor processes, which were impacted by both speed instruction and compatibility.

As a SAT effect is present on both PMT and MT, we also evaluated the inter-participant correlation of the effects. This correlation was significant for both compatibility conditions ( $r_{14} = .78, p < .001$  and  $r_{14} = .66, p < .01$ , for compatible and incompatible, respectively, see left panel of Figure 2). However, as indicated by the color code, this correlation between effects is largely driven by the overall RT of the participants.

#### EMG burst analysis

Mean EMG bursts obtained under each speed instruction are presented in Figure 2 (right panel), for both compatible and incompatible trials. EMG traces clearly show that muscular activity is mainly affected by speed instruction, and not by compatibility. This was confirmed by statistical analyses showing that EMG surface was larger ( $F(1, 15) = 9.56, p < .01, \eta_p^2 = .39, BF_{10} = 5840$ ), and burst rising slope was steeper ( $F(1, 15) = 15.60, p < .01, \eta_p^2 = .51, BF_{10} = 180018$ ) under SPD compared to ACC instruction.



**Fig. 2** Left: SAT effect on MT as a function of the SAT effect on PMT for all participants, for both compatible (filled circles) and incompatible (open circles) conditions. The color code of the symbols corresponds to the mean RT of the participant in each compatibility condition. It appears that the correlation is largely driven by the mean RT. Right: Mean EMG burst for both ACC and SPD instructions, in compatible and incompatible trials (black: ACC, grey: SPD ; solid line: compatible, dotted line: incompatible).

While compatibility showed a trend for slightly larger bursts in incompatible trials ( $F(1, 15) = 4.22, p = .06, \eta_p^2 = .22$ ), this was not supported by Bayesian analysis ( $BF_{01} = 3.897$  slightly in favor of no effect), and slope analysis also supports the absence of a compatibility effect ( $F(1, 15) < 1, \eta_p^2 = .001, BF_{01} = 3.973$ ). No interaction between the speed instruction and the compatibility was observed (although this absence was not strongly supported by Bayesian analysis, surface:  $F(1, 15) < 1, \eta_p^2 = .03, BF_{01} = 1.36$ ; slope:  $F(1, 15) = 2.27, p = .15, \eta_p^2 = .13, BF_{01} = 1.19$ ).

## Discussion

Trading speed for accuracy is ubiquitous, present in virtually all cognitive processing and can also be evidenced in a large range of animal species, from insects to humans (see Heitz 2014 for a recent overview). As such, SAT appears to be a core property of information processing and has recently become a *de facto* critical benchmark for decision making models. Providing an adequate description of the mechanisms underlying SAT hence seems essential for our understanding of cognition. Based on the modeling literature, displacements along the speed-accuracy trade-off are generally assumed to be driven only by variations in the response threshold level (*i.e.*, the amount of evidence required to trigger the response (Bogacz et al 2009)). However, recent data, both in monkeys and humans, have challenged this pure threshold-related explanation, showing that the rate of information accumulation is also affected by SAT (see Heitz 2014 for a recent overview).

In the present study, we show that non-decisional, motor-execution-related components of the reaction are

also affected by SAT, contributing to more than 20% of the total effect. Importantly, although a SAT effect on motor processes duration has previously been suggested by LRP studies, these results might not be conclusive as recent data suggest that the decision to act is performed, at least in part, within the motor structures (see *e.g.* Donner et al 2009; Purcell et al 2012).

The present results, on an other hand, unambiguously establish that late motor processes also take part to RT shortening under speed stress. Such a motoric effect shows that even response execution stages can be affected by cognitive and strategic factors, indicating the necessity to consider motor components, which are often neglected, in all experimental tasks. Although not entirely incompatible, the current findings also question the assumption that response-related and decision processes are independent, and purely serial (Calderon et al 2015; Servant et al 2015)<sup>4</sup>.

Besides chronometry, analysis of EMG bursts further allowed for a clarification of the underlying physiological processes. In the present data, EMG bursts were shown to have larger surfaces and steeper slopes when response speed was emphasized. Steeper EMG signals index a better synchronization of the motor units discharges (Meijers et al 1976; Ulrich and Wing 1991), reflecting a more efficient motor command. Hence, the faster response execution observed under SPD instruction is due to a stronger and more efficient cortical motor command.

Improving execution processes comes at a cost, however: it impairs on-line correction processes, as suggested by recent results on “partial error” trials (sub-threshold incorrect muscular activity involving within-trial correction processes – Burle et al 2002; Spieser et al 2015).

<sup>4</sup> A single factor can affect two serial stages independently

Indeed, the efficiency to prevent “partial errors” to turn into overt errors has been shown to be reduced under time pressure (Burle et al 2014). This might be linked to recent studies showing that the decision process continues even after a motor command has been sent (Resulaj et al 2009; Freeman et al 2011; Calderon et al 2015; Servant et al 2015; van den Berg et al 2016), which allows to correct initial incorrect actions. However, speeding up response execution reduces the time available for a correction, thereby directly impacting correction efficiency. This hypothesis is further supported by the recent observation that participants with longer motor times present more *corrected* incorrect response activations (Servant et al 2015). As a consequence, SAT also occurs within the motor execution level, contributing to the global trade-off both in terms of chronometry and of error rate, as speeding up motor execution increases the number of overt (uncorrected) errors.

SAT effects on premotor and motor components appear correlated, and covary with reaction time, raising the possibility of a common origin. The common denominator could be our observed faster build-up rate of EMG activity and the recent observation that the rate of information accumulation in the decision stage is faster under speed than under accurate instruction (Heitz and Schall 2012; Hanks et al 2014), paralleling the steeper EMG burst observed in the present data. Whether the rate of EMG recruitment directly depends on the information accumulation rate remains an open question.

To conclude, the present results unambiguously demonstrate that motor components of the reaction processes are **not** spared by SAT. Not only are they affected by time pressure, but they also largely contribute to this trade-off, since shortening motor execution stages also increases the number of overt errors by reducing the likelihood of interrupting and correcting incorrect response activation (Burle et al 2014; Servant et al 2015). This confirms, in humans, that SAT is a more diverse effect than a simple modulation of threshold level, as also recently argued (Heitz and Schall 2012).

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