General Cognitive Ability and the Psychological Refractory Period: Individual Differences in the Mind's Bottleneck

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Identifying the precise locus of high-level ability differences in the flow of information between perception and action is an important goal of differential psychology. In order to localize the negative correlation between general cognitive ability (*g*) and reaction time to a specific processing stage, we administered a speeded number-comparison task to two groups differing in average *g*. The participants had to respond to two stimuli in each trial, producing the well-known slowing of the second reaction time known as the "psychological refractory period." The difference in the second reaction time favoring the high-*g* group doubled as the stimulus onsets became very close together. This finding affirms that the faster reaction times of higher-*g* individuals reflect an advantage exclusively in the serial bottleneck of central processing and not in the parallel peripheral stages.

Keywords: intelligence, response time, psychological refractory period, diffusion models

It is now well established that higher levels of general cognitive ability ("*g*" or "IQ") are associated with faster mean times and less trial-to-trial variability in simple speeded tasks (Deary, Der, & Ford, 2001; Hunt, 2005; Jensen, 2006). A mechanistic account of the *g*-RT correlation, however, remains elusive. One approach toward building such an account is to pinpoint the locus of the *g*-RT correlation within models partitioning the flow of information between perception and action into processing stages (Luce, 1986; Sanders, 1998; Pashler, 1998). Because the distinctive properties of different stages are potentially revealing with respect to deeper mechanisms, the integration of the *g*-RT correlation into stage models is a promising strategy for tracing individual differences to lower-level causes (Chabris, 2007; Deary, Penke, & Johnson, 2010).

There are two types of RT partitions to which one might turn. The first turns on the distinction between parallel and serial processing raised by studies of dual tasks, in which participants must respond to two stimuli presented close together in time. At very short asynchronies, the second RT becomes longer (Pashler, 1998; Lien, Ruthruff, & Johnston, 2006). A parsimonious account of this *psychological refractory period* (PRP) invokes three successive stages of processing: a perceptual stage (*P*), a central stage (*C*), and a motor stage (*M*). The *P* stage translates raw sensory input to a more abstract format that can be broadcast to downstream processors unconcerned with retinal locus, stimulus-background contrast, character font, and other low-level features. The *C* stage consists of a mapping from percept to response ("response selection"), and PRP theory posits that this stage alone gives rise to the slowing of the second RT in a dual task (Figure 1). The final *M* stage consists of implementing the motor response selected by the *C* stage. The corollary of the *C* stage being the only bottleneck is that, up to a certain limit, perceptual and motor processing for a given stimulus can take place concurrently with processing of any kind for another stimulus.

On the assumption of only one serial stage, it may seem more theoretically neutral to call the three stages in Figure 1 "pre-bottleneck," "bottleneck," and "post-bottleneck." However, the temporal position and time-sharing property of the stage affected by an experimental manipulation can be determined from the pattern of changes in the reaction times to the first and second stimulus (Schweickert & Townsend, 1989; Pashler, 1998), and the psychological nature of the manipulations that have been found to affect stages preceding, constituting, and following the serial bottleneck justify the use of the labels *P*, *C*, and *M*, at least as a rough mnemonic. For example, because a reduction of contrast normally prolongs reaction time but *does not* do so when applied to the second stimulus after a brief onset asynchrony, we may infer that contrast affects a pre-bottleneck stage (Pashler & Johnston, 1989; De Jong, 1993; Wong, 2002). This is because the elongation of the stage affected by contrast reduction must be absorbed into the "slack" (refractory period) opened up by the second instance of the serial stage waiting for the completion of the first instance (Figure 1). Guided by similar theoretical considerations, previous studies have found that the nu-

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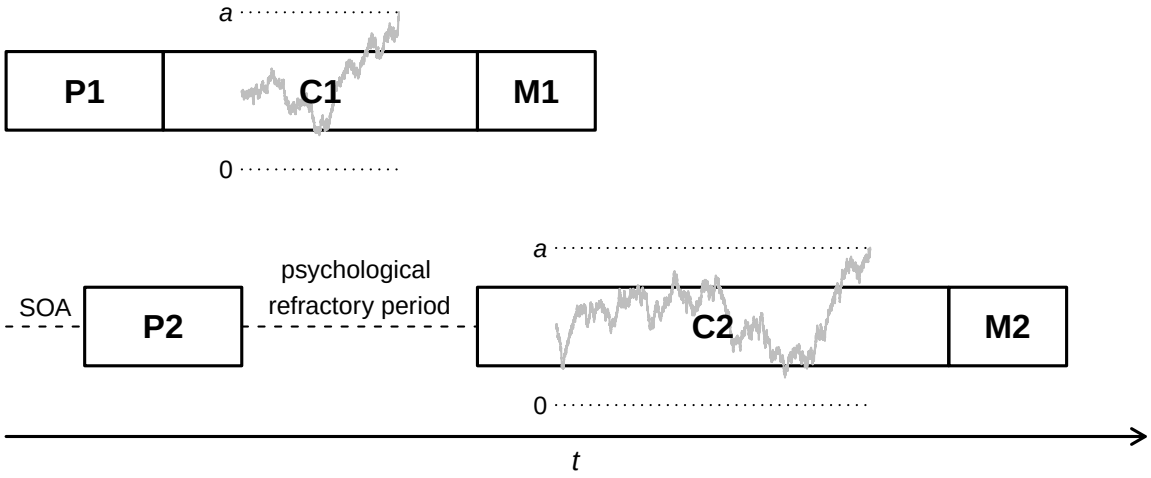

Figure 1. The unified PRP-diffusion model. The second stimulus follows the first after a stimulus onset asynchrony (SOA). The perceptual (*P*) and motor (*M*) stages vary little in duration from trial to trial and can be carried out in parallel with stages in the processing of another stimulus. The central (*C*) stage contains a noisy accumulation of evidence (diffusion) until a decision threshold is reached. C_2 cannot start until C_1 is finished, which results in the "slack" or "refractoriness" referred to as the PRP. See Lien, Schweickert, and Proctor (2003) and Sigman and Dehaene (2006) for refinements of this core model. If *g* is positively correlated with diffusion rate, it follows that *g* is also associated with more rapid progress through the serial bottleneck.

merical magnitude of the stimulus affects a bottleneck stage (Sigman & Dehaene, 2005, 2006; Corallo, Sackur, Dehaene, & Sigman, 2008) and that motor and articulatory demands affect a post-bottleneck stage (Ferreira & Pashler, 2002; Sigman & Dehaene, 2005). A number of EEG studies have also shown that the slack time responsible for the PRP must precede the lateralized readiness potential, an index of late central or early motor processing (Smulders & Miller, 2012), and these findings are consistent with the placement of the slack between P_2 and C_2 in Figure 1.

A second line of research has attempted to explain the characteristic dispersion and skewness of RT distributions. The most successful models of two-choice tasks posit a partition of RT into a stochastic process deciding between the two responses and a low-variability residual time (Ratcliff & Rouder, 1998; Ratcliff & McKoon, 2008). During the stochastic process, an internal variable undertakes a *diffusion* ("continuous random walk") between two boundaries, each of which corresponds to a response alternative (Figure 1). The diffusion thus represents the noisy accumulation of evidence in favor of one response. Variants of this diffusion model have attracted much attention in recent years, not only because of their excellent fit to human behavioral data, but also because of emerging links to neural mechanisms (Gold & Shadlen, 2007).

A recent series of investigations has begun unifying these three strands of research—individual differences, dual-task interference, and diffusion modeling—into a coherent whole. First, it has been shown that the stochastic evidence-accumulation stage of the diffusion model is encompassed wholly within the serial *C* stage of the PRP model (Sigman & Dehaene, 2005; Kamienkowski, Pashler, Dehaene, & Sigman, 2011). Second, it has been shown that the *g*-RT correlation reflects a correlation of *g* with the rate of the diffusion process and not with residual time (Schmiedek, Oberauer, Wilhelm, Süß, & Wittman, 2007; Ratcliff, Thapar, & McKoon, 2010, 2011).

On the basis of the unified model (Figure 1), we deduce a consequence for the nature of the *g*-RT correlation that has so far been untested: the serial bottleneck posed by *C* is the only stage in the PRP partition that contributes to the correlation. In fact, this is the deduction to be tested in the present work. While our hypothesis that *g* is correlated only with *C* and not with *P* or *M* is simple to state, it generates a number of stringent predictions for our experiment varying the time between stimulus onsets within a dual task. We will use Figure 1 to derive these predictions in the context of the speeded number-comparison task employed in our study. This task requires participants to press one key if the stimulus (a positive integer) is smaller in numerical magnitude than a reference

and another key if the stimulus is larger. We will call the reaction time to the first stimulus within a trial "RT1" and the reaction time to the second stimulus "RT2."

At a short stimulus onset asynchrony (SOA), the initiation of C_2 must wait for the termination of C_1, which provides a slack into which P_2 can expand without prolonging the overall RT2. If two individuals differ in the duration of P, then the difference is propagated only once into RT2. Specifically, at a short SOA, the additional time taken by the slower person's P_1 pushes forward C_1, which in turns pushes forward C_2 and results in a longer RT2. P_2, in contrast, expands into slack without pushing forward subsequent stages. At a long SOA, C_2 is no longer pushed from behind by C_1, but now the absence of slack means that prolonging P_2 must also prolong RT2. Therefore the difference between individuals in RT2 will not depend on SOA. The same invariance holds for a difference in a post-bottleneck stage or for a combination of differences in any parallel stages surrounding the bottleneck.

If individuals differ in the duration of a serial stage, however, then at a short SOA this difference is propagated twice into RT2. In this temporal regime, the two serial computations are arranged end-to-end, and therefore the slower individual's RT2 suffers a double cost. This leads to our primary prediction: any difference between individuals in a serial stage will result in an exact doubling of their RT2 difference as the SOA changes from large to small values.

If individuals differ in both parallel and serial stages, it is possible to observe other factors besides one or two by which their difference in RT2 increases as the SOA diminishes. However, given the now repeatedly replicated finding that g is associated with diffusion rate and not residual time, a division of the g difference across stages with different time-sharing properties is rather implausible. Such a division would amount to the diffusion of evidentiary strength between decision boundaries switching from seriality to parallelism while still in progress. Thus, we have set up a confrontation between the point predictions of one and two, and evidence in favor of the latter would support our hypothesis that g is associated with the rapidity of a serial processor.

We tested the logic of our theoretical predictions by manipulating the stimulus-reference numerical distances of both stimuli and, in a separate experiment with 9 participants (see the Supplemental Material), the stimulus-background contrasts of both stimuli. A simultaneous manipulation of the two stimuli mimics individual differences in the affected stage.

The unified PRP-diffusion model not only predicts the behavior of the mean difference in RT2 but also constrains the entire joint distribution of RT1 and RT2. If g is associated with a serial and stochastic stage, then we predict that the difference in RT2 variance between individuals varying in g will also increase by a factor of at least two as the SOA becomes small. The factor may exceed two as a result of positive correlations among the durations of the stochastic stages contributing to RT2.

Table 1
Summary Statistics of Participants

	high g	moderate g
total number	36	34
female	19	20
right-handed	32	30
age (years)	21.0 (1.6)	20.8 (1.6)
diffusion rate	.391 (.080)	.346 (.068)
boundary separation	.087 (.017)	.101 (.026)
residual time (ms)	323 (30)	326 (30)

Note: Discrete variables are summarized by counts. Continuous variables are summarized by the mean and, parenthetically, the standard deviation.

The validation of our hypothesis regarding the nature of the g-RT correlation would harmonize with many related proposals dividing human mental architecture into two broad components: (i) a number of parallel (modular) processors of sensory data and motor commands, operating inflexibly but with great precision; and (ii) a central workspace that can establish arbitrary links between processors through a serial chain of computations (*e.g.*, O'Reilly, 2006). As work along these lines continues to proceed at both algorithmic and neural levels of analysis, a firm placement of g in the second component would connect the study of ability differences to multiple lines of reductionistic investigation.

Method

Participants

Student volunteers qualified for the study by documenting either a score of 1560 or higher on the SAT (Critical Reading and Mathematics) or a score of 1280 or lower. Documentation was supplied either by signing a release to obtain scores from the university registrar or by logging in to the College Board website. Although lacking a component testing spatial ability, the SAT is otherwise an excellent measure of g (Frey & Detterman, 2004). We refer to participants documenting a score of 1560 or higher as the "high-g" group and to other participants as the "moderate-g" group. The difference between the moderate and high cut scores is about 1.3 standard deviations.

Seventy individuals participated and were not removed (Table 1). The Supplemental Material provides additional details regarding the one excluded participant.

Design

The stimuli were numbers ranging in magnitude from 1 to 9 (excluding 5). In each trial a number appeared just to the

left of the fixation cross, and participants had to press the Q key with the left middle finger if the number was smaller than 5 and the W key with the left index finger if the number was greater than 5. After the SOA a second number appeared just to the right of the fixation cross. Participants had to press the O key with the right index finger if the number was smaller than 5 and the P key with the right middle finger if the number was greater than 5.

The SOA was varied between 60 and 960 ms inclusive, in increments of 60 ms. Each of the 64 possible combinations of numbers (1-4, 6-9) was used 16 times, once for each SOA. After randomization of the order, these 1024 trials were broken up into 32 blocks of 32 trials each with a 20-s break between blocks. The interval between trials was 1000 ms. Additional details may be found in the Supplemental Material.

Analyses

The data were analyzed with R and the lme4, boot, and simpleboot packages. All models specified the participant-specific intercept and effect of stimulus-reference numerical distance on RT as random effects. When using the BC_a bootstrap algorithm to perform statistical inference with respect to ratios of RT2 differences (Efron & Tibshirani, 1993), we also resampled trials within individuals. We give each point estimate with ±1 standard error.

(T, a, v) are the parameters of the diffusion model (Ratcliff & McKoon, 2008). T is the duration of the low-variability residual stage—according to the unified model in Figure 1, the summed durations of P, M, and the non-diffusion portions of C. a is the separation between the decision boundaries (which determines speed-accuracy tradeoff). v can be thought of as the rate at which the process would travel from the starting point $(a/2)$ to the appropriate boundary in the absence of stochastic perturbations; in the unified model, this diffusion process takes place during C. We estimated each participant's diffusion parameters using the EZ2 package (Grasman, Wagenmakers, & van der Maas, 2009). This approach is based on analytical expressions for the moments of RT in terms of (T, a, v). In the simulation study of van Ravenzwaaij and Oberauer (2009), the EZ method outperformed approaches based on maximum likelihood and minimization of Kolmogorov-Smirnov distance in recovering individual differences.

Results

Figure 2 demonstrates the classic PRP effect for both high- and moderate-g groups: a prolonging of RT2 at short SOAs that declined with a slope of approximately −1 as the SOA increased from 60 to 180 ms (−.91 ± .04).

Before interpreting the PRP pattern in Figure 2, we present the estimates of the diffusion process governing a participant's RT in the absence of PRP interference. We used the trials from the two longest SOAs because, as suggested by Figure 2 and Table S1, by this time the distribution of RT2 in most participants no longer showed signs of interference. We found that whereas the high-g group enjoyed an advantage in diffusion rate, $t(67.3) = 2.56$, $p < .02$, $d = .62$, the two g groups showed a nonsignificant mean difference of only 3 ms in residual time, $t(67.6) = .40$, $p > .68$, $d = -.10$.

The difference between the groups in boundary separation was significant, $t(54.4) = 2.81, p < .007, d = -.69$. This association may have arisen because we rewarded both speed and accuracy (Supplemental Material). In other respects, however, our results replicate previous findings with respect to the association of g exclusively with diffusion rate and not residual time. Table 1 gives the sample statistics of the diffusion parameters.

In our separate experiment manipulating contrast, we compared trials with two low-contrast stimuli to trials with two high-contrast stimuli and found no evidence of the difference in RT2 varying with SOA; the difference was 24 ± 23 ms at the shortest SOA and 33 ± 6.3 ms at the other SOAs (see the Supplemental Material for more detailed results). In our main experiment, we found that the difference in RT2 between trials with two numerically near stimuli and two distant stimuli was 72 ± 6.8 ms at the two shortest SOAs and 34 ± 3.3 ms at the two longest. These effects of mimicking individual differences with experimental manipulations are consistent with our theoretical predictions: a difference in parallel stages remains constant as SOA varies, whereas a difference in C doubles at short SOAs.

Figure 2 shows that the difference in RT2 between the two g groups increased from 39 ± 13 at the short-SOA range (≥ 900 ms) to 106 ± 25 ms at the short-SOA range (≤ 120 ms)—a factor of 2.74, 99% CI [1.96, 5.47]. Note that RT1 in PRP tasks appears to be prolonged by an executive task-scheduling stage (E) between P_1 and C_1 that is itself composed of both low-variability and stochastic components (Jiang, Saxe, & Kanwisher, 2004; Sigman & Dehaene, 2006; Kamienkowski et al., 2011). After using the method described in the Supplemental Material to correct for the carryover of E into RT2, we found that reducing the SOA into the interference regime increased the difference in RT2 between the moderate- and high-g groups by the factor 1.96, 99% CI [1.22, 3.29]. We clearly cannot reject the hypothesis that the factor is precisely equal to two. In contrast, we reject the hypothesis that the factor is equal to one, $p < .001$.

Our repetition of the same task allowed us to decompose total RT into estimated durations of $P + M$ and C, quantities that are not separately available in most PRP studies (Supplemental Material). These estimates provide additional validation of our hypothesis to the extent that they are consistent with the results of other investigators. We found that the moderate- and high-g groups differed on average from each other in $P+M$ by less than 2 ms; the overall mean was 213±6

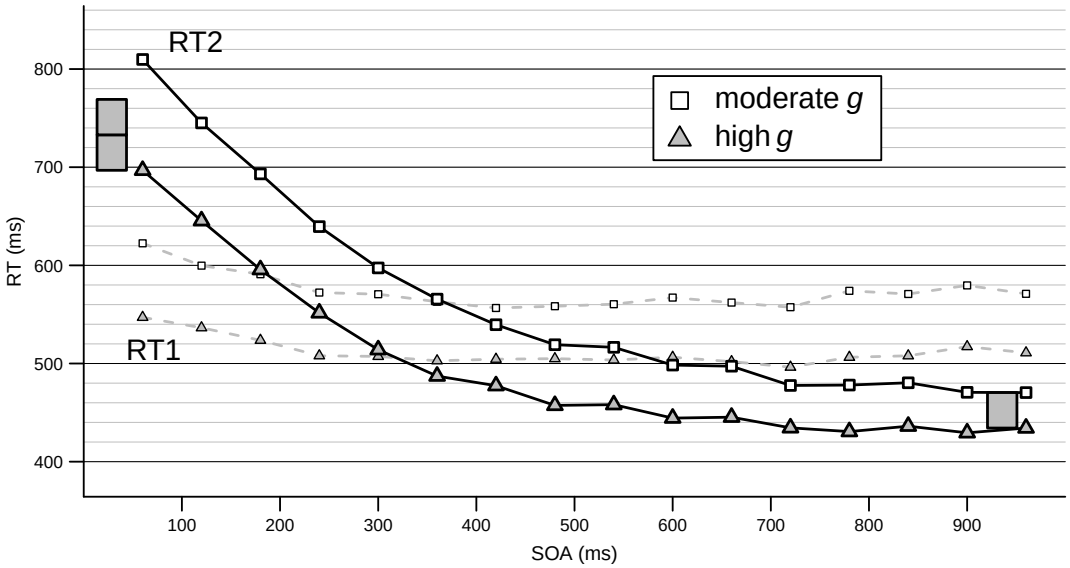

Figure 2. Mean reaction time as a function of stimulus order, SOA, and *g* group. The height of the gray bar corresponds to the asymptotic difference in the reaction time to the second stimulus in the trial (RT2) between the moderate- and high-*g* groups (39 ms). Note that the RT2 difference between the *g* groups more than doubles as the SOA diminishes.

ms. Previous studies employing the number-comparison task have estimated the average duration of P to be 180-190 ms (Dehaene, 1996; Sigman & Dehaene, 2005). Given the assumption that the average duration of M is relatively brief, lasting about 30 ms, our partition of RT is in good agreement with those obtained using other approaches. Note that our estimate of T, 324 ± 4 ms, is over 100 ms longer than our estimate of $P + M$, implying that the central bottleneck contains approximately 110 ms of additional processing that is not associated with *g*.

Given our estimate of $P + M$, we can say that the average duration of C was 218 ms in the high-*g* group and 258 ms in the moderate-*g* group. We also used the RT1 difference from 420 ms ≤ SOA ≤ 720 ms to estimate that the average asymptotic duration of E was 71 ms in the high-*g* group and 89 ms in the moderate-*g* group; bootstrapping showed that this difference was marginally significant, $p < .06$.

The RT variances also behaved in accordance with our predictions. At the longest SOAs, the variance of RT2 was about 4000 ms^2 greater in the moderate-*g* group. At the SOA became smaller, this difference progressively increased to 25,000 ms^2 (Table S1 of the Supplemental Material). The differences between the *g* groups in E, RT1 variance, and short-SOA RT2 variance suggest that the stochastic portion of E is also associated with *g*.

A particularly revealing parameter of the RT1-RT2 joint distribution is the within-trial correlation. Figure 3 shows that the RT1-RT2 correlation was initially of similar magnitude (∼.80) in both the moderate- and high-*g* groups. At an SOA of 240 ms, the correlation began to decline in both groups, but more precipitously in the high-*g* group. When the SOA is 180 ms or shorter, P_2 almost always finishes while the processing of the first stimulus is still somewhere within P_1, E, or C_1. Therefore, in this temporal regime, the initiation of C_2 is time-locked to the termination of C_1, producing the strong RT1-RT2 correlation. Starting at an SOA of 240 ms, C_1 is sometimes finished before the termination of P_2. This is because the stochasticity of the executive and central stages can occasionally result in trials with absorption times that are very short. When C_2 is free to start immediately, RT1 and RT2 are no longer locked together. C_1 is shorter, less variable, and less right-skewed in the high-*g* participants, freeing the initiation of C_2 on an increasingly greater proportion of their trials, and thus their RT1-RT2 correlation declines more steeply with SOA.

Discussion

We inferred that the *g* advantage in RT should reside in the serial stage encompassing the chief stochastic contribution to RT and reported several results upholding this prediction. The mapping of retinal stimulation to an abstract quantity representation and the final implementation of the selected motor response—two stages that can be executed with little trial-to-trial variability and in parallel for at least two stimuli—are not associated with *g*. The intervening serial stage contains a stochastic accumulation of evidence, and the positive correlation between *g* and the accumulation

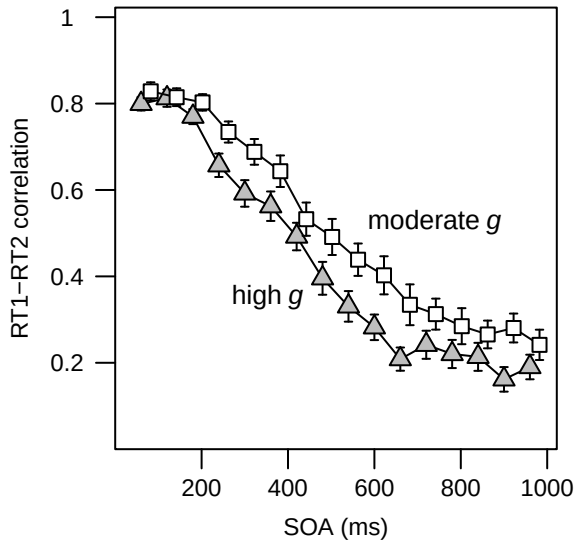

Figure 3. The zero-lag correlation between RT1 and RT2 as a function of SOA and *g* group. For clarity the data points for the moderate- and high-*g* groups are horizontally displaced by a small quantity. The moderate-*g* correlation is always slightly to the right of the high-*g* correlation corresponding to the same SOA. Each point is the average correlation of the participants in the group. The bars encompass ±1 standard error of the mean estimated correlation.

rate is what accounts for the overall *g*-RT association in the number-comparison task.

The importance of having isolated the *g*-RT association to a particular stage of course depends on the causal nature of the association. Our causal hypothesis is that individual differences in brain structure affecting central processing (diffusion rate) in laboratory tasks also affect central processing in natural settings, leading to the behavioral individual differences summarized as *g*. One might object that the *g*-*C* association predicted by this hypothesis might alternatively arise as a result of trivial confounding or reverse causation. However, the negative *g*-RT correlation has been found to hold within families (Jensen, Cohn, & Cohn, 1989), which rules out many conceivable sources of confounding. Also, higher-*g* individuals tend to respond more rapidly even in tasks such as detecting the onset of a single light (Jensen, 2006), and it seems unlikely that higher-*g* individuals have more experience with this kind of simple detection. Furthermore, the fact that *g* is not associated with residual time is inconsistent with an appeal to practice or familiarity, since practice has been found to affect residual time as well as diffusion rate (Dutilh, Vandekerckhove, Tuerlinckx, & Wagenmakers, 2009; Kamienkowski et al., 2011). Nevertheless we acknowledge the need for further evidence affirming the causal significance of the *g*-*C* correlation.

It has been proposed that the *g*-RT correlation is attributable to (i) the need for working memory to maintain stimulus-response bindings in arbitrary laboratory tasks and (ii) the causal contribution of working memory capacity to *g* (Wilhelm & Oberauer, 2006). Similar proposals have been given by Gray, Chabris, and Braver (2003) and Kane, Conway, Hambrick, and Engle (2007). These hypotheses are not necessarily in conflict with our PRP results, as the strength of stimulus-response bindings in working memory may be an important determinant of diffusion rate. More generally, given the potential of brain-imaging tools to spatially circumscribe the temporal signature of the *g*-associated *C* stage (Sigman & Dehaene, 2008; Tombu et al., 2011), the PRP paradigm is a promising source of unifying insights into the cognitive and neural underpinnings of ability variation.

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