



# Psychopathy is associated with an exaggerated attention bottleneck: EEG and behavioral evidence from a dual-task paradigm

Scott Tillem<sup>1</sup> · Hannah Weinstein<sup>1</sup> · Arielle Baskin-Sommers<sup>1</sup>

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## Abstract

Psychopathy is a personality disorder associated with a chronic disregard for the welfare of others. The attention bottleneck model of psychopathy asserts that the behavior of individuals higher on psychopathy is due to an exaggerated attention bottleneck that constrains all information processing, regardless of the information's potential goal-relevance. To date, the majority of research on the attention bottleneck model of psychopathy conceptually applied the tenets of the model but did not implement methods that directly test an exaggeration of the bottleneck in psychopathy. Accordingly, the presence of an exaggerated bottleneck, the exact expression of that bottleneck, and its potential mechanistic relevance for behavior in individuals higher on psychopathy remains untested. To address these gaps, a sample of 78 male community members, evaluated for psychopathic traits using the Self-Report Psychopathy-III scale, completed an EEG-based dual-task paradigm examining short stimulus onset asynchrony (SOA; 300 ms), long SOA (1,100 ms), and single-task baseline conditions. Additionally, participants were asked about their frequency of real-world risky, impulsive, and antisocial behaviors. Psychopathy was associated with slower reaction times to second targets (T2s) presented during the dual-task conditions, relative to the baseline condition. Psychopathy also was associated with blunted P300 responses, a neural index of stimulus evaluation, across all types of T2 events. Finally, bottleneck-related interference during the short SOA events mediated the relationship between psychopathy and real-world behavior. These findings suggested that individuals higher on psychopathy exhibit an exaggerated bottleneck which produces intense and long-lasting interference, impacting all information processing and partially contributing to their maladaptive behavior.

**Keywords** Attention · Psychopathy · Dual-task · erp · Risk-taking behavior · Attention Bottleneck

Individuals higher on psychopathy are notorious for their prolific antisocial behavior and their ability to be interpersonally manipulative and charming. They are known for their tendency to engage in elaborate cons, callously assault others, impulsively look for adventures, and chronically commit antisocial acts to obtain their goals (e.g., money, power, thrills; Hare, 2003, 2006). Decades of research suggest that the disruptive behaviors of individuals higher on psychopathy are partly due to their uncanny ability to focus myopically on their selected goal when asked to respond to goal-relevant information while ignoring contextual distractors (Baskin-Sommers et al., 2011, 2013; Hiatt et al., 2004; Hoppenbrouwers et al.,

2015; Larson et al., 2013; Newman et al., 2010; Wolf et al., 2012; Zeier et al., 2009).

As an extension of the prominent response modulation model (Gorenstein & Newman, 1980; Newman et al., 1990; Newman & Kosson, 1986; Newman & Schmitt, 1998), the attention bottleneck model asserts that the ability of individuals higher on psychopathy to focus on their selected goals is due to the exaggeration of an attention bottleneck, a selective attention mechanism identified from research in neurotypical individuals. In neurotypical individuals, reaction times (RTs) are slower to a second target when presented quickly following a first target (i.e., 0-500 ms apart) during dual-task paradigms (Brisson & Jolicœur, 2007; Dell'Acqua et al., 2005; Dux et al., 2006; Filmer et al., 2013; Hesselmann et al., 2011; Jentsch et al., 2007; Kida et al., 2004; Klapötke et al., 2011; Luck, 1998; Reimer et al., 2017; Sigman & Dehaene, 2008; Tombu et al., 2011). However, there is no evidence of a delay to a subsequent goal-relevant target when neurotypical individuals are given enough time between the

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✉ Scott Tillem  
scott.tillem@yale.edu

<sup>1</sup> Department of Psychology, Yale University, 2 Hillhouse Ave., New Haven, CT 06511, USA

first and second targets (e.g., 1,100 ms between stimulus onsets; Brisson & Jolicoeur, 2007; Dell'Acqua et al., 2005; Dux et al., 2006; Filmer et al., 2013; Hesselmann et al., 2011; Jentsch et al., 2007; Kida et al., 2004; Klapötke et al., 2011; Luck, 1998; Reimer et al., 2017; Sigman & Dehaene, 2008; Tombu et al., 2011). This pattern of findings initially led researchers to theorize the presence of a “psychological refractory period” (PRP), which inhibits simultaneous response selection (Pashler, 1994). More recent findings from dual-task paradigms examining the PRP, along with convergent lines of research examining perceptual load and attention blink paradigms (Dux & Marois, 2009; Linnell & Caparos, 2011), have expanded upon this initial theory and ultimately led to the assertion that multiple elements of human cognition (e.g., response selection, conscious perception and attention, etc.) are constrained by the presence of a central information processing bottleneck (Dux et al., 2006; Tombu et al., 2011). These various lines of research demonstrate that as neurotypical individuals engage in various aspects of information processing (e.g., decoding perceptual inputs, selecting responses to stimuli, etc.), neural information passes through a bottleneck which constrains parallel processing. Consequently, information is processed in series until it clears the bottleneck, with goal-relevant information being prioritized. This serial processing, in turn, is thought to delay the processing of both goal-relevant (e.g., second target cues during dual-task paradigms) and goal-irrelevant information (e.g., goal-irrelevant affective cues) presented before an initial stream of goal-relevant information clears the bottleneck.

The attention bottleneck model of psychopathy extends this research from neurotypical individuals to a clinical disorder. This model asserts that the effects of the bottleneck are amplified in psychopathy such that the processing of secondary streams of information (regardless of their potential value or relevance) is delayed even more or is completely inhibited (Baskin-Sommers et al., 2011, 2013; Baskin-Sommers & Newman, 2013). In this way, the attention bottleneck model of psychopathy provides a biologically plausible theoretical framework for explaining the attention abnormalities commonly observed in individuals higher on psychopathy.

Across experimental contexts, individuals higher on psychopathy show an intact, or even enhanced, ability to attend and respond to goal-relevant and/or perceptually simple information; however, they appear insensitive to streams of information that are goal-irrelevant or imbedded in visually complex stimuli. For example, during various neurocognitive tasks (e.g., Flanker-type, modified Stroop, visual search, etc.) individuals higher on psychopathy display superior performance (e.g., faster RTs) in task conditions that require them to respond to a single, goal-relevant target, while ignoring contextual distractors (Hiatt et al., 2004; Hoppenbrouwers et al., 2015; Wolf et al., 2012; Zeier et al., 2009). Additionally, during tasks that evoke affective responses

(e.g., instructed fear-conditioning, passive-avoidance learning), individuals higher on psychopathy show intact or enhanced affective responses when affective content is goal-relevant but blunted affective responses when the same affective content is made goal-irrelevant (Baskin-Sommers et al., 2011; Baskin-Sommers, Curtin, et al., 2013; Decety et al., 2013; Larson et al., 2013; Newman et al., 2010; Schultz et al., 2016; Tillem & Baskin-Sommers, 2018; Tillem et al., 2016). Lastly, during passive picture-viewing, individuals higher on psychopathy show deficits in responding to affective content when it is embedded in visually complex stimuli but intact affective responses when the perceptual load of those stimuli is reduced (e.g., through simplification or familiarization; Baskin-Sommers, Curtin, et al., 2013; Sadeh & Verona, 2012).

While these findings support the predictions made by the attention bottleneck model of psychopathy, questions regarding the presence, expression, and overall relevance of this psychopathy-related bottleneck remain. First, a core assertion of the attention bottleneck model of psychopathy is that individuals higher on psychopathy exhibit an exaggerated attention bottleneck that impedes the processing of all secondary streams of information (regardless of their goal-relevance). However, the presence of such a bottleneck in psychopathy has not yet been tested using methods similar to the work in neurotypical samples. As noted above, support for an attention bottleneck among neurotypical individuals largely is derived from research examining dual-task paradigms, which shows slowed responses to a secondary stream of information (i.e., second targets) when it is presented shortly after a first target stimulus, even though that secondary stream is equally goal-relevant. Therefore, in addition to constraining the processing of secondary streams of goal-irrelevant information, an exaggerated version of this attention bottleneck in psychopathy also should constrain the processing of secondary streams of goal-relevant information. More specifically, an exaggerated bottleneck in psychopathy should lead individuals higher on psychopathy to be significantly slower when processing and responding to second targets during dual-task paradigms. However, prior research using dual-task paradigms in psychopathy has been unable to fully explore how psychopathy may impact the speed of information processing for secondary streams of goal-relevant information due to ill-suited task designs (e.g., RT data not reported and no designation of primary and secondary targets; Jutai et al., 1987) or “insufficient data points” (Kosson, 1996, p. 395) to evaluate completely RT data. Accordingly, the impact of psychopathy on the speed of processing and response to secondary streams of goal-relevant information has yet to be tested directly. Without direct evidence that psychopathy impacts the speed of processing of secondary streams of goal-relevant information, specifically, it is possible that the attention abnormalities evident in individuals higher on psychopathy are limited to

impacting the processing of goal-irrelevant information, rather than reflecting the presence of an exaggerated bottleneck that restricts the processing of all information presented close to an initial target, regardless of its relevance.

Second, the specific expression of a psychopathy-related exaggeration of the attention bottleneck currently is underspecified. Research on neurotypical individuals highlights features related to the intensity, duration, and multiple sensory modality of the bottleneck (Brisson & Jolicœur, 2007; Dell'Acqua et al., 2005; Dux et al., 2006; Filmer et al., 2013; Hesselmann et al., 2011; Jentsch et al., 2007; Kida et al., 2004; Klapötke et al., 2011; Luck, 1998; Reimer et al., 2017; Sigman & Dehaene, 2008; Tombu et al., 2011). As of yet, such information on the nature of the bottleneck is not available for individuals higher on psychopathy. In psychopathy, an exaggerated bottleneck could increase the intensity of interference caused by information clearing the bottleneck so that it takes individuals higher on psychopathy longer to process and respond to information presented shortly (e.g., 0–500 ms) after an initial target. Additionally, an exaggerated bottleneck in psychopathy may increase the duration of bottleneck-related interference to such a degree that information presented long after (e.g., 1,100 ms) an initial target is still impacted by the psychopathy-related bottleneck. Lastly, prior research on attention in psychopathy exclusively used visual stimuli; however, theoretically, the bottleneck constrains the processing of all information, regardless of the specific sensory modality of the stimuli (Dux et al., 2006; Tombu et al., 2011). Therefore, the multisensory expression of the bottleneck in psychopathy is untested.

Finally, no study has specifically linked any aspect of the attention bottleneck to real-world behaviors. Individuals higher on psychopathy commit more crimes than other individuals (Hare, 2003, 2006; Hare & Neumann, 2008). Additionally, individuals higher on psychopathy engage in a wider variety of antisocial behaviors (Hare, 2003, 2006; Hare & Neumann, 2008) and are more likely to try a wider array of illicit substances than individuals lower on psychopathy (Brennan, Hyde, & Baskin-Sommers, 2017a; Brennan et al., 2017b). Currently, there is only speculation about the mechanistic role of an exaggerated bottleneck in the wide variety of callous, impulsive, and antisocial behaviors associated with individuals higher on psychopathy. Accordingly, the overall relevance and importance of a psychopathy-related bottleneck remain unclear.

The present study employed an electroencephalogram (EEG)-based, dual-task paradigm using both auditory and visual stimuli to address these gaps in our understanding of the attention bottleneck in psychopathy. The dual-task paradigm was adapted from work on the attention bottleneck in neurotypical community samples (Dux et al., 2006). Specifically, we examined whether individuals higher on psychopathy exhibited: (a) more intense bottleneck-related

interference by determining whether they exhibited slower RTs to a second auditory or visual target following a short, 300 ms, stimulus offset asynchrony (SOA) during a dual-task condition (T2Short) relative to a single-task baseline condition; (b) longer-lasting bottleneck-related interference by determining whether they exhibited slower RTs to a second auditory or visual target following a long, 1,100 ms, SOA during a dual-task condition (T2Long) relative to a single-task baseline condition; or (c) both attention bottleneck-related issues. In addition to RT data, we recorded EEG data during the task to index the amplitude of the P300 response, an event-related potential (ERP) that is sensitive to dual-task interference (Hesselmann et al., 2011; Kida et al., 2004; Luck, 1998; Reimer et al., 2017) and is thought to reflect the amount of neural resources used for stimulus evaluation during decision-making (Polich, 2007). Similar to the behavioral evaluations, examining the P300 response allowed us to evaluate if individuals higher on psychopathy exhibited evidence of: (a) more intense bottleneck-related interference in neural information processing (i.e., blunted P300 responses to T2Short events); (b) longer-lasting bottleneck related interference in neural information processing (i.e., blunted P300 responses to T2Long events); or (c) both. Finally, following from the attention bottleneck model of psychopathy, we explored the relevance of this psychopathy-related bottleneck by examining whether abnormalities in dual-task performance, in general (i.e., any and all abnormalities in behavioral task-performance and/or neural responding associated with psychopathy), mediated the relationship between psychopathy and real-world risky, impulsive, and antisocial behaviors.

## Methods

### Participants

Participants were 78 males between the ages of 18 and 65 ( $M = 38.13$ ,  $SD = 12.98$ ; see Table 1 for sample demographics). Given that the prior research on the attention bottleneck in psychopathy used male samples (Baskin-Sommers et al., 2011; Baskin-Sommers, Curtin, et al., 2013; Baskin-Sommers & Newman, 2013) and research has shown that the neurocognitive underpinnings of psychopathy may differ by sex (O'Leary et al., 2007; Rogstad & Rogers, 2008), the present sample included only male participants. All participants were recruited from the New Haven area. Nationally, New Haven ranks in the 96<sup>th</sup> percentile for crime; on average, 337 crimes are committed per square mile, compared to a statewide rate of 19 and the national median of 31.1 (<http://www.neighborhoodscout.com/ct/new-haven/crime/>, accessed on 06/05/2020). The rate of violent crime is 8.50 (per 1,000 residents) compared with a statewide rate of 2.07 and a national median of 4.00. Reflecting these trends, 66.7% of

**Table 1** Sample characteristics and Zero-Order correlations (N = 78)

| Variable                   | n  | Mean   | Std. dev. | Min    | Max    | Correlations |      |                |                |       |                |
|----------------------------|----|--------|-----------|--------|--------|--------------|------|----------------|----------------|-------|----------------|
|                            |    |        |           |        |        | 1            | 2    | 3 <sup>a</sup> | 4 <sup>b</sup> | 5     | 6 <sup>c</sup> |
| 1. Age                     | 78 | 38.13  | 12.98     | 18.00  | 65.00  | —            | -.03 | -0.02          | 0.00           | -0.09 | 0.14           |
| 2. IQ                      | 78 | 110.08 | 10.07     | 130.00 | 88.00  |              | —    | 0.58*          | 0.06           | -0.09 | 0.22           |
| 3. Race <sup>a</sup>       | 78 |        |           |        |        |              |      | —              | 0.13           | -0.04 | 0.07           |
| White                      | 52 |        |           |        |        |              |      |                |                |       |                |
| Black                      | 24 |        |           |        |        |              |      |                |                |       |                |
| Other                      | 2  |        |           |        |        |              |      |                |                |       |                |
| 4. Handedness <sup>b</sup> | 78 |        |           |        |        |              |      |                | —              | -0.07 | -0.12          |
| Right-handed               | 69 |        |           |        |        |              |      |                |                |       |                |
| Left-handed                | 9  |        |           |        |        |              |      |                |                |       |                |
| 5. SRP                     | 78 | 159.82 | 28.29     | 101.00 | 234.00 |              |      |                |                | —     | 0.46*          |
| 6. RISQ <sup>c</sup>       | 76 | 27.74  | 19.43     | 4.00   | 84.00  |              |      |                |                |       | —              |

\*  $p < 0.001$

<sup>a</sup> Spearman correlation was used to examine the effect of race (dichotomously coded, white vs. nonwhite)

<sup>b</sup> Spearman correlation was used to examine the effect of handedness (dichotomously coded, right-handed vs. left-handed)

<sup>c</sup> Two participants were missing data from the RISQ

the present sample reported being arrested and 25.6% of the sample reported being incarcerated.

Before data collection, all participants were prescreened with both a phone interview and an in-person assessment to exclude those who performed below the fourth-grade level on a standardized measure of reading (Wilkinson, 1993); scored below 70 on a brief measure of IQ (Zachary & Shipley, 1986); had a diagnosis with psychotic symptoms; had taken or were currently taking antipsychotic medication; or had a history of neurological issues which may impact their comprehension of study materials (e.g., uncorrectable auditory or visual deficits, colorblindness, head injury with loss of consciousness for greater than 30 minutes, etc.). All participants provided written consent approved by the Yale University Human Investigation Committee. Participants were paid an hourly rate based on the current minimum wage.

## Power Analysis

*A priori* power analysis based on previous studies examining attention abnormalities in psychopathy (Hiatt et al., 2004; Hoppenbrouwers et al., 2015; Wolf et al., 2012; Zeier et al., 2009) was conducted using G\*Power (Faul et al., 2009). Power analysis indicated that a sample size of 72 participants would be needed to produce sufficient power (80%) to detect a moderate effect size for the predicted omnibus interaction between SOA conditions (T2Short vs. T2Long vs. Single-Task Baseline) and a continuous predictor (i.e., psychopathic trait scores).

## Measures

### Self-Report Psychopathy-III Scale (SRP-III; Paulhus et al., 2015)

Participants were assessed for psychopathy using the SRP-III. The SRP-III consists of 64 self-report items measuring the presence of different psychopathic traits (e.g., callousness, shallow affect, impulsivity, etc.) on a 5-point Likert scale (1 = *disagree strongly* to 5 = *agree strongly*). The SRP-III has a minimum total score of 64 and maximum score of 320. In prior validation studies, the SRP-III has shown strong convergent validity with the Psychopathy Checklist-Revised (the current “gold standard” for assessing psychopathy in incarcerated samples; Hare, 2003; Neumann et al., 2015) and is well-documented as a valid and reliable means of assessing psychopathy in community samples (Gordts et al., 2017; Neumann et al., 2015). In the current sample, Cronbach’s  $\alpha = 0.914$  for SRP-III total score. Following prior work examining the attention bottleneck model of psychopathy, the current study conceptualized psychopathy as a unitary construct and limited analyses to examining SRP-III total score (see [Supplemental Material](#) for analyses examining SRP-III factor and facet scores).

An examination of the distribution of SRP-III total scores in the current sample showed that the sample’s SRP-III total scores were normally distributed (Shapiro-Wilk test of normality,  $p = 0.362$ ) and consistent with a sample elevated on psychopathic traits. More specifically, prior studies have established standardized norms for SRP-III/IV total scores (Paulhus et al., 2017) in community samples, designating four different score ranges (“Low”: 64-99, “Average”: 100-147,



“Elevated”: 148-172, “Extremely Elevated”: 173-320), and the mean SRP-III total score for the current sample ( $M = 159.82$ ,  $SD = 28.29$ ) falls into the “Elevated” score range. Additionally, 24.4% of the current sample fell into the “Extremely Elevated” range ( $n = 19$ ) and 44.8% fell into the “Elevated” range ( $n = 35$ ), while only 30.8% of the sample fell into the “Average” score range ( $n = 24$ ) and no participants scored in the “Low” range.

**Risky, Impulsive, and Self-destructive Behavior Questionnaire (RISQ; Sadeh & Baskin-Sommers, 2017)** Real-world impulsive and antisocial behaviors were measured using the RISQ. The RISQ is a self-report measure that assesses 38 different risky, impulsive, self-destructive, and/or antisocial behaviors across 8 different categories (drug use, aggression, gambling, sex, alcohol use, self-harm, impulsive eating, and reckless behavior). As part of this assessment, participants are asked how many times in their life they engaged in each of the 38 behaviors. Based on the recommended scoring procedures, to reduce skewness in the frequency of behaviors, bins (0 times, 1-10, 11-50, 51-100, >100) were created for the total frequency counts of behaviors across each of the 8 categories. Since psychopathy is associated with a wide variety of impulsive and antisocial behaviors (Brennan, Hyde, et al., 2017; Brennan, Stuppy-Sullivan, et al., 2017; Hare, 2003, 2006; Hare & Neumann, 2008), we used RISQ total frequency score (summing the bin scores across all 8 categories of behavior) as our metric of real-world behavior.

**Covariates** Participants’ age, IQ, and handedness were assessed as part of the initial in-person assessment and included as simultaneous covariates in all regression and mediation models. Age and IQ (as measured by the Shipley Institute of Living Scale; Zachary & Shipley, 1986) were included in all analyses because both have been associated with differences in allocation of attention, in general, and performance on dual-task paradigms, in particular (Fogarty & Stankov, 1982; Lonie et al., 2009; Madden, 2007; Nęcka, 1996; Verhaeghen et al., 2003). Handedness was included in all analyses since it has been reliably linked to differences in neural organization and responding (e.g., differences in the P300 response; Eskikurt et al., 2013; Galin et al., 1982; Hatta, 2007).

### Dual-task paradigm

Participants completed a version of Dux et al.’s (2006) dual-task paradigm that was modified to be compatible with EEG data collection. More specifically, participants completed 12 alternating blocks of trials based on the dual-task detailed in Task 1 and the single-task detailed in Task 2 of Dux et al. (2006).

During dual-task trials, participants were presented with two targets with one of two possible stimulus onset

asynchronies (SOAs), a short SOA (300 ms between onset of the first target [T1] and the onset of the second target [T2]), or a long SOA (1,100 ms between the onset of T1 and the onset of T2). On each trial, target stimuli included a visual stimulus (a red circle or a blue circle) presented on the screen and an auditory stimulus presented over headphones. Auditory targets were one of two discriminable sounds taken from Dux et al.’s (2006) paradigm. These sounds consisted of natural sounds, man-made sounds, and/or complex tones that were altered by reversing the waveforms and/or adding noise. All target stimuli were presented for 200 ms before stimulus offset.

During single-task trials, participants were presented with a single target stimulus for 200 ms, consisting of either a visual stimulus (a red circle or a blue circle) or an auditory stimulus (sound X or sound Y). On all trials, regardless of dual- or single-task, participants were instructed to respond, via one of two button boxes, to visual stimuli by pressing a button on one button box with one hand and auditory stimuli by pressing a button on another button box with the other hand as quickly and accurately as possible (e.g., press button 1 on the left button box with the left hand whenever a red circle appeared, press button 2 on the left button box with the left hand for a blue circle, press button 3 on the right button box with the right hand whenever target sound X was heard and press button 4 on the right button box with the right hand for sound Y). Participants were instructed to respond to all stimuli with their second and fifth fingers.

Specific response mappings between stimuli and hand (e.g., visual stimuli left hand/button box, auditory stimuli right hand/button box and vice versa) were counterbalanced across participants; however, the specific stimuli and response mappings used for a given participant stayed constant throughout the entirety of their session, across both dual-task and single-task conditions. Instruction regarding specific response mappings was provided before the start of the task. For all trials, participants had 3 s to complete their responses following the final target onset for that trial. If the participant completed their responses before the end of the response window, the trial would end early. Following each trial, there was a 2- to 3-s (jittered, mean 2.5 s) intertrial interval before the next trial began.

Trials were grouped into 12-blocks that alternated between a dual-task condition (6 blocks) and a single-task (baseline) condition (6 blocks). Whether the first block of the task was a dual-task block or a single-task block was counterbalanced across participants. Each dual-task block consisted of 48 dual-task trials, balancing SOA (short vs. long) and stimulus-order ( $T1_{\text{visual}}-T2_{\text{auditory}}$  [VA] vs.  $T1_{\text{auditory}}-T2_{\text{visual}}$  [AV]) within each block (i.e., each block consisted of: 12 Short SOA, VA trials; 12 Short SOA, AV trials; 12 Long SOA, VA trials; and 12 Long SOA, AV trials). Dual-task trial order was randomized within each block. Each single-trial

block consisted of 24 trials: 12 visual target trials, and 12 auditory target trials. Trial order was randomized within each single-task block. A white central fixation point was presented throughout the entirety of each block. At the end of each block, participants were given a 15-s break during which they were informed of their accuracy on the previous block and reminded whether the next block would be a single-task or dual-task block. Before completing the task, participants completed two practice blocks, one dual-task practice block, and one single-task practice block, both consisting of 16 trials.

## EEG data acquisition and processing

EEG was recorded throughout the session from Ag-AgCl electrodes mounted on a standard 32-channel elastic cap (Electro Cap International, Eaton, OH) utilizing Neuroscan Synamps amplifiers and acquisition software (Compumedics, Charlotte, NC). Vertical eye-movement was recorded with electrodes placed above and below the left eye in line with the pupil. The online reference channel was M1.

Offline preprocessing of midline channels (i.e., Fz, Cz, Pz, and Oz) was completed using EEGLab (Delorme & Makeig, 2004) and consisted of: global re-referencing (i.e., channels were re-referenced against the averaged global signal taken from all channels on the cap), low-pass filtering (at 30 Hz using a 2nd-order Butterworth low-pass filter), blink correction (using regression methods based on Semlitsch et al., 1986), epoching, baseline correction (−1,000 ms through −100 ms before T1 presentation), and artifact rejection ( $\pm 75$   $\mu$ V). The exact time window examined during epoching varied by the specific event being examined due to differences in the timing of events across conditions. Specifically, epochs examining T1 in the single-task condition (BaselineT1<sub>visual</sub> and BaselineT1<sub>auditory</sub>) spanned −1,000 ms through 2,500 ms post BaselineT1 onset. For T2 events in the dual-task, short SOA condition (T2Short<sub>visual</sub> and T2Short<sub>auditory</sub>), epochs spanned −1,300 ms through 2,000 ms after T2Short onset. For T2 events in the dual-task, long SOA condition (T2Long<sub>visual</sub> and T2Long<sub>auditory</sub>) epochs spanned −2,400 ms through 2,000 ms post T2Long onset. Only trials in which the participant provided the correct response were processed. All incorrect trials automatically were rejected. Following preprocessing, nine participants were removed from ERP analysis due to issues with data quality (e.g., excess noise, broken sensors, failures in blink correction, etc.). This resulted in a subsample of  $n = 69$  participants who had valid data (number of valid trials per event after preprocessing:  $M = 65.30$  trials,  $SD = 8.38$ , Min. = 25, Max. = 72) for the ERP analysis.

The ERPLab plugin for EEGLab (Lopez-Calderon & Luck, 2014) was used to average epochs together to generate ERPs for each event of interest (i.e., BaselineT1<sub>visual</sub>, BaselineT1<sub>auditory</sub>, T2Short<sub>visual</sub>, T2Short<sub>auditory</sub>, T2Long<sub>visual</sub>, and T2Long<sub>auditory</sub>). Given the timing of events in the task, T1 neural activity

continued to occur after T2 onset, particularly for the Short SOA events, contaminating any examination of T2-related neural activity. Accordingly, to examine neural responses to T2 events, neural activity to T1 events had to be accounted for (see also Dell'Acqua et al., 2005 & Luck, 1998). In the current study, we addressed this issue by creating contrast T2 ERPs which subtracted out BaselineT1 neural activity from the dual-task T1 response (e.g., T1Short<sub>auditory</sub> ERP − BaselineT1<sub>auditory</sub> ERP = T2Short<sub>visual</sub> ERP). This resulted in estimates for the T2 ERPs unbiased by T1-related neural activity. After the contrast ERPs were generated, the grand average ERP waveforms were inspected to identify the best channel and time window to examine the P300 response for T2 events. Of all the midline channels analyzed, a clear P300 response across all T2 events only occurred at Pz (between 300 ms and 800 ms post T2 onset; Fig. 1). Accordingly, mean ERP amplitude at Pz was extracted for each participant from the 300 to 800 ms time window for all T2 events.

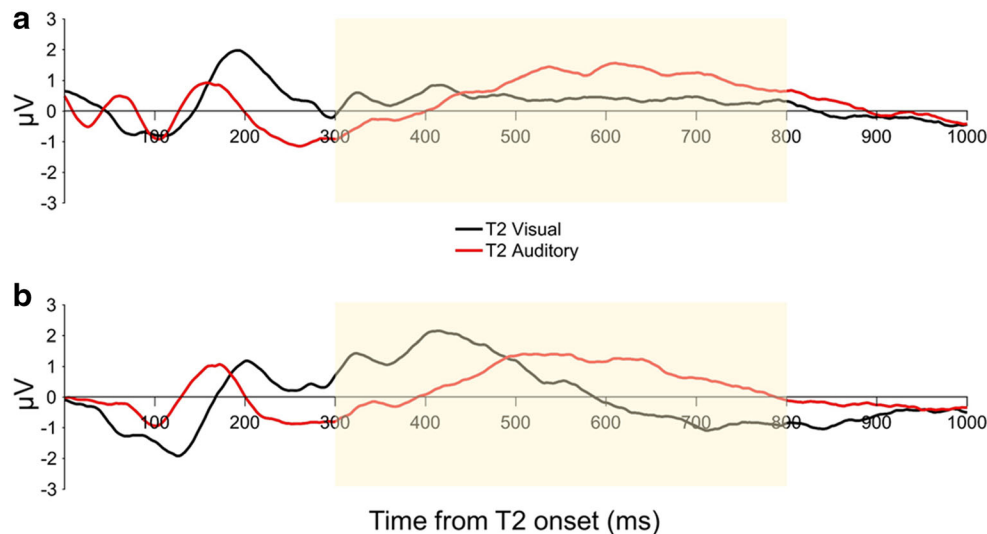
## Results

### Behavioral Analysis

For the behavioral analysis, a 3 (Condition: BaselineT1, T2Short, T2Long) by 2 (Sensory: Visual, Auditory) repeated measures GLM with SRP-III total score included as a continuous, between-subject factor, and age, IQ, and handedness (dichotomously-coded, right-handed vs. left-handed) included as covariates, was run examining RT data. Any significant Condition effects or interactions were followed by two planned simple interaction contrasts, the T2Short vs. BaselineT1 RT contrast and the T2Long vs. BaselineT1 RT contrast, specifically chosen to test our *a priori* hypotheses regarding potential psychopathy-related differences in bottleneck intensity and duration, respectively. While not relevant to our *a priori* hypotheses, for the sake of completeness, an additional analysis of the behavioral data was conducted examining response accuracy (see [Supplemental Materials](#) for details regarding this accuracy analysis).

This analysis revealed several significant basic task effects. For example, consistent with prior research in neurotypical samples (Dux et al., 2006), there was a significant within-subject effect of Condition<sup>1</sup>,  $F(1.423, 103.92) = 445.80$ ,  $p < 0.001$ ,  $\eta_p^2 = 0.859$ , 90% confidence interval (CI) [0.818–0.884]. Planned simple contrasts showed that RT to T2Short was significantly slower than BaselineT1 RT,  $F(1, 73) = 295.76$ ,  $p < 0.001$ ,  $\eta_p^2 = 0.802$ , 90% CI [0.732–0.842], and T2LongRT was significantly faster than BaselineT1 RT,  $F(1, 73) = 217.35$ ,  $p < 0.001$ ,  $\eta_p^2 = 0.738$ , 90% CI [0.673–0.807]. There also was a significant within-subject effect of Sensory where participants were faster to respond to visual targets than

<sup>1</sup> To protect against violations of sphericity, Huynh-Feldt corrected  $p$ -values and  $df$  are reported for all GLM analyses.



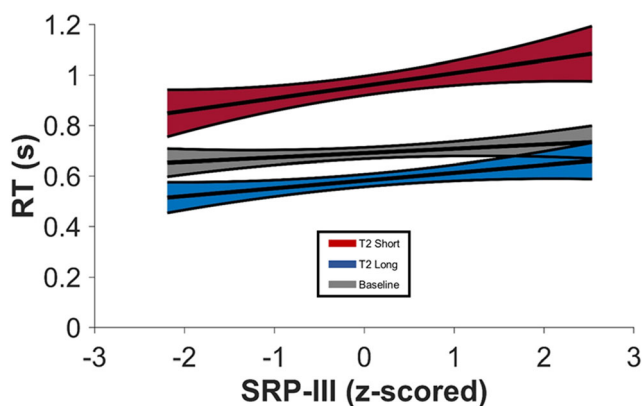
**Fig. 1** T2 Events evoked a P300 response between 300–800 ms post T2 onset at Pz. **A** Grand-average waveforms for T2Short events at Pz. **B** Grand-average waveforms for T2Long events at Pz. T2 time-locked

grand-average waveforms controlled for T1 responses by subtracting out the BaselineT1 waveform from the matching T1 event. Shaded regions depict the 300 to 800 ms time window examined.

auditory targets,  $F(1, 73) = 24.75, p < 0.001, \eta_p^2 = 0.253$ , 90% CI [0.119–0.377]. However, both of these effects were qualified by a significant Condition  $\times$  Sensory interaction,  $F(1.85, 134.70) = 86.13, p < 0.001, \eta_p^2 = 0.541$ , 90% CI [0.444–0.609]. When this interaction was broken down by sensory modality, the within-subject effect of Condition was significant for both visual,  $F(1.36, 99.12) = 437.86, p < 0.001, \eta_p^2 = 0.857$ , 90% CI [0.814–0.883], and auditory targets,  $F(1.79, 130.99) = 242.07, p < 0.001, \eta_p^2 = 0.768$ , 90% CI [0.710–0.805]. Simple contrasts showed that T2Short RT was significantly slower than BaselineT1 RT for both visual  $F(1, 73) = 320.84, p < 0.001, \eta_p^2 = 0.815$ , 90% CI [0.748–0.852], and auditory targets  $F(1, 77) = 125.52, p < 0.001, \eta_p^2 = 0.632$ , 90% CI [0.516–0.706], and T2Long RT was significantly faster than BaselineT1 RT for both visual,  $F(1, 77) = 80.48,$

$p < 0.001, \eta_p^2 = 0.524$ , 90% CI [0.388–0.617], and auditory targets  $F(1, 77) = 168.70, p < 0.001, \eta_p^2 = 0.698$ , 90% CI [0.597–0.759].

In addition to these basic task effects, this model revealed significant SRP-III-related effects. More specifically, consistent with our *a priori* predictions of exaggerated bottleneck-related interference in individuals higher on psychopathy, there was a significant SRP-III  $\times$  Condition interaction,  $F(1.42, 103.92) = 3.57, p = 0.047, \eta_p^2 = 0.047$ , 90% CI [0.000–0.122]. Planned follow-up simple contrasts revealed that SRP-III scores were related to significantly increased RT for T2Short,  $F(1, 73) = 4.92, p = 0.030, \eta_p^2 = 0.063$ , 90% CI [0.003–0.167], relative to BaselineT1, consistent with greater intensity of bottleneck-related interference in individuals higher on psychopathy. Similarly, SRP-III scores were associated with increased RT for T2Long,  $F(1, 73) = 5.32, p = 0.024, \eta_p^2 = 0.068$ , 90% CI [0.005–0.174], relative to BaselineT1, suggesting longer-lasting bottleneck interference in individuals higher on psychopathy (Fig. 2).<sup>2</sup> No other SRP-III interactions were significant (SRP-III  $\times$  Sensory interaction:  $F(1, 73) = 0.18, p = 0.677, \eta_p^2 = 0.002$ , 90% CI [0.000–0.051]; SRP-III  $\times$  Condition  $\times$  Sensory interaction:  $F(1.85, 134.70) = 2.29, p = 0.110, \eta_p^2 = 0.030$ , 90% CI [0.000–0.084]). Similarly, the main effect of SRP-III was not significant,  $F(1, 73) = 1.88, p = 0.175, \eta_p^2 = 0.025$ , 90% CI [0.000–0.109].



**Fig. 2** Individuals higher on psychopathy show longer reaction times (RTs) during dual-task conditions relative to baseline. Regression lines for T2Short RT (red), T2Long RT (blue), and BaselineT1 RT (gray) as a function of SRP-III total scores, controlling for age, IQ, and handedness. Error bands represent one standard error.

<sup>2</sup> To help ensure that these effects were not being primarily driven by the impact of SRP-III scores on BaselineT1 RT, a follow-up linear regression model was run with BaselineT1 RT as the dependent variable and SRP-III scores as the primary predictor of interest, controlling for age, IQ, and handedness. Results showed that SRP-III scores did not significantly predict BaselineT1 RT,  $F_{\text{overall}}(4, 73) = 4.58, p = 0.002, \beta = 0.082, p = 0.442$ , 90% CI [−0.017–0.046].

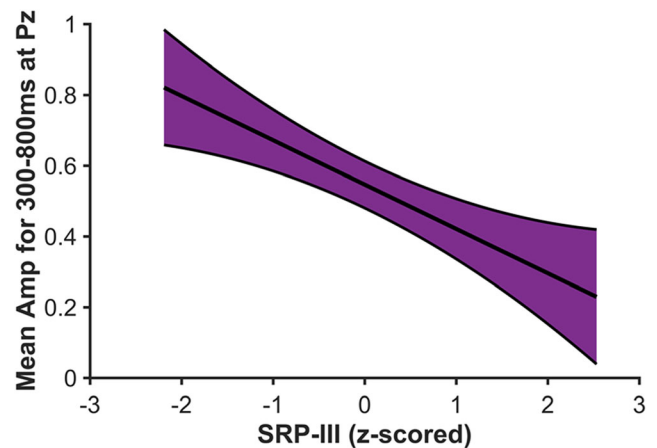
## ERP Analysis

For the ERP data analysis, we entered the mean amplitude of neural activity at Pz (300–800 ms post T2 onset) for the T2 vs. Baseline contrast ERPs into a 2 (SOA: Short, Long)<sup>3</sup> by 2 (Sensory: Visual, Auditory) repeated measures GLM, with SRP-III total score included as a continuous, between-subject factor and age, IQ, and handedness (dichotomously coded) included as covariates. There was a significant main effect of SRP-III,  $F(1, 64) = 4.33, p = 0.041, \eta_p^2 = 0.063, 90\% \text{ CI } [0.001-0.175]$ . Individuals who scored higher on psychopathy showed lower mean P300 amplitude across T2 events (Fig. 3), suggesting a general blunting of neural resources available for stimulus evaluation in these individuals when processing secondary streams of information.<sup>4</sup> No other SRP-III effects were significant (SRP-III x SOA interaction:  $F(1, 64) = 0.02, p = 0.880, \eta_p^2 < 0.001, 90\% \text{ CI } [0.000-0.013]$ ; SRP-III x Sensory interaction:  $F(1, 64) = 2.30, p = 0.134, \eta_p^2 = 0.035, 90\% \text{ CI } [0.000-0.132]$ ; SRP-III x SOA x Sensory interaction:  $F(1, 64) = 0.59, p = 0.446, \eta_p^2 = 0.009, 90\% \text{ CI } [0.000-0.081]$ ).

## Mediation Analysis

To estimate the indirect effect, a mediation analysis using bootstrapping with 5,000 nonparametric samples was run with the SPSS macro “PROCESS” Model 4 (Hayes, 2013). SRP-III was the independent variable and RISQ total score was the dependent variable. The mediators were selected based on the significant behavioral (intensity effect: T2Short vs. BaselineT1 RT contrast; duration effect: T2Long vs. BaselineT1 RT contrast) and neural (mean P300 amplitude) effects reported above. Age, IQ, and handedness (dichotomously coded) were included as covariates. Significance of all indirect effects were evaluated via 95% bootstrapped CI.

The indirect effect of SRP-III on RISQ total through bottleneck intensity (T2Short vs. BaselineT1 RT contrast) was significant,  $\beta = 0.047, 95\% \text{ CI } [0.002-0.158]$ . The total indirect effect,  $\beta = 0.024, 95\% \text{ CI } [-0.062-0.124]$ , the indirect effect through T2Long vs. BaselineT1 RT contrast,  $\beta = -0.019, 95\% \text{ CI } [-0.127-0.023]$ , and the indirect effect through mean P300 amplitude were not significant  $\beta =$



**Fig. 3** Individuals higher on psychopathy show lower neural activity to T2 events, as measured by the mean amplitude of the P300 response. Regression line for the mean amplitude of P300 activity at Pz across all T2 events, measured between 300ms and 800ms post T2 onset, as a function of SRP-III total scores, controlling for age, IQ, and handedness. Error band represents one standard error.

$-0.004, 95\% \text{ CI } [-0.084-0.047]$  (see Fig. 4 for all path coefficients). Additionally, after accounting for the potential indirect effects, the direct effect of SRP-III on RISQ total remained significant,  $\beta = 0.552, p < 0.001, 95\% \text{ CI } [0.306-0.739]$ .

## Discussion

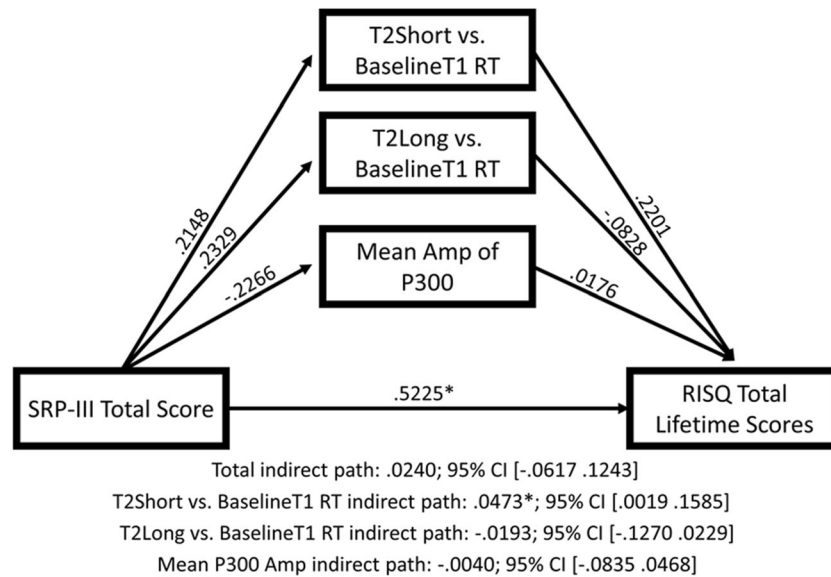
Selective attention is a process that is fundamental to human cognition. It impacts what information is available, and subsequently, how that information is used in both basic and more complex decision-making (e.g., from deciding which button to press during a task to whether or not to commit a crime; Armel et al., 2008; Dux et al., 2006; Tombu et al., 2011). Selective attention dysfunctions characterize a variety of psychiatric disorders, including psychopathy. The attention bottleneck model of psychopathy suggests that individuals higher on psychopathy exhibit an exaggerated attention bottleneck. This exaggerated bottleneck constrains the allocation of selective attention resources, limiting the simultaneous processing of multiple streams of information regardless of their potential relevance (Baskin-Sommers et al., 2011; Baskin-Sommers, Curtin, et al., 2013; Baskin-Sommers & Newman, 2013).

The present study is the first to demonstrate, directly, the presence of an exaggerated bottleneck among individuals higher on psychopathy. During an EEG-based dual-task paradigm, individuals higher on psychopathy exhibit an exaggeration of the attention bottleneck demonstrated by those lower on psychopathy (and previously documented in neurotypical individuals; Dux et al., 2006; Filmer et al., 2013; Tombu et al., 2011). Moreover, these findings show that this psychopathy-related bottleneck interferes with information processing even

<sup>3</sup> A baseline condition could not be included in the model directly because the BaselineT1 ERP amplitudes were used to generate the T2 subtraction ERPs.

<sup>4</sup> To help ensure this finding was not being driven by a general blunting of the P300 response to all stimuli in individuals high on psychopathy, an additional 2 level (Sensory: BaselineT1<sub>visual</sub> Mean Amp. vs. BaselineT1<sub>auditory</sub> Mean Amp.) repeated-level GLM was run with SRP-III scores as a predictor of interest, controlling for age, IQ, and handedness. This showed that neither the SRP x Sensory interaction,  $F(1, 64) = 2.07, p = 0.155, \eta_p^2 = 0.031, 90\% \text{ CI } [0.000-0.126]$ , nor the main effect of SRP-III,  $F(1, 64) = 1.53, p = 0.221, \eta_p^2 = 0.023, 90\% \text{ CI } [0.000-0.112]$ , were significant suggesting that psychopathy does not significantly impact mean P300 amplitude to BaselineT1 events.





**Fig. 4** Intensity of bottleneck-related interference mediates the relationship between psychopathy and real-world impulsive, antisocial behaviors. The mediation model tests the relationship between psychopathy (as measured by SRP-III total score), intensity of bottleneck-related interference (as measured by T2Short vs. BaselineT1 RT contrast), duration of

bottleneck-related interference (as measured by T2Long vs. BaselineT1 RT contrast), neural resources used to evaluate T2 stimuli (as measured by mean P300 amplitude to T2 events), and real-world impulsive and antisocial behaviors (as measured by the RISQ), controlling for age, IQ, and handedness.

when these individuals are attending to two independent, but equally relevant, streams of information. The current study also elucidates the expression of this exaggerated bottleneck in psychopathy. Individuals higher on psychopathy experience both a more intense (i.e., greater slowing of behavioral responses) and longer-lasting period of bottleneck-related interference (i.e., extending into the 1,100-ms delay-condition) regardless of the sensory modality of the information. Results from the mediation analysis highlight the connection between the intensity of bottleneck-related interference and the real-world behaviors associated with psychopathy.

While an attention bottleneck that inhibits the simultaneous processing of multiple streams of information may intuitively seem disadvantageous, particularly in the context of multitasking, this same process allows neurotypical individuals (e.g., community members lower on psychopathy) to focus on goal-relevant information without getting overwhelmed by irrelevant, less salient, or distracting streams of information (Brisson & Jolicœur, 2007; Dell'Acqua et al., 2005; Dux et al., 2006; Dux & Marois, 2009; Filmer et al., 2013; Hesselmann et al., 2011; Jentsch et al., 2007; Kida et al., 2004; Klapötke et al., 2011; Luck, 1998; Reimer et al., 2017; Sigman & Dehaene, 2008; Tombu et al., 2011). The exaggerated version displayed by individuals higher on psychopathy appears to perform a similar function by allowing them to complete basic tasks that require minimal information processing without getting distracted (e.g., superior performance on Flanker-type tasks; Zeier et al., 2009). However, this exaggeration comes at a steeper cost than simply the difficulty in multitasking displayed by neurotypical

individuals. In individuals higher on psychopathy, the exaggerated bottleneck may slow information processing to such a degree that neurocognitive functions which require the processing of complex stimuli (e.g., complex visual scenes; Baskin-Sommers, Curtin, et al., 2013; Sadeh & Verona, 2012) and/or rapid integration of multifaceted information (e.g., decision-making; Baskin-Sommers et al., 2016) become impaired. As a result, real-world information processing, which is both complex and multifaceted, may be compromised.

Findings from the mediation analysis support the idea that an exaggerated bottleneck in psychopathy is related to real-world behavior. In particular, the greater intensity of the bottleneck is relevant to the impulsive and antisocial behavior characteristic of individuals higher on psychopathy, suggesting that a psychopathy-related inability to process multiple simultaneous streams of information may partially underlie their characteristic behavior. The apparent link between impaired simultaneous processing and antisociality suggests that in situations where complex, multifaceted information is presented (e.g., social interactions), individuals high on psychopathy may not be able to integrate rapidly and effectively all elements of the situation into their learning and decision-making processes (e.g., failure to detect, learn from, and integrate salient cues). This exaggerated bottleneck may lead psychopathic individuals to have a fractionated perception of complex situations, resulting in behavior that appears impulsive and antisocial, but that may more fundamentally reflect a failure to complete the integration of important information into their decisions to act.

Of note, bottleneck duration did not mediate the association between psychopathy and real-world behavior. One reason for this could be that the enhanced duration of the bottleneck found in psychopathy may not be sufficient to impair functioning. During the paradigm, individuals higher compared to lower on psychopathy were slower to respond to the second target presented at 1,100 ms relative to the single-task baseline. However, the sample as a whole responded to the 1,100-ms second-target significantly faster than the baseline. Accordingly, the longer duration of the bottleneck may not impair dual-task (or real-world) functioning in individuals higher on psychopathy. Instead, it may simply limit the enhancements that participants low on psychopathy displayed in the long delayed second-target condition.

The present study also showed that the blunted P300 response did not mediate the relationship between psychopathy and real-world behavior. This may be because P300 amplitude at Pz is an indirect neural metric of the attention bottleneck. In general, an interpretation of the relationships among neural metrics, neurocognitive functioning, and/or real-world behavior is more suitable when examining neural responses in a specific brain region, which is tightly linked to the neurocognitive functions being examined (Nash et al., 2015). Research establishing the neural underpinnings of the bottleneck in the neurotypical community has linked the bottleneck with neural functioning in the posterior portion of the lateral prefrontal cortex (IPFC), superior medial frontal cortex, and insula (Dux et al., 2006; Filmer et al., 2013; Tombu et al., 2011). Similarly, prior empirical and theoretical work on attention abnormalities in psychopathy suggests that the psychopathy-related abnormalities in the bottleneck may be rooted in abnormal functioning of the IPFC (Larson et al., 2013), an atypical organization of the dorsal attention network (Tillem et al., 2019), and/or global impairments in neural functioning and communication (Hamilton et al., 2015; Kiehl, 2006). Accordingly, more direct neural measures of IPFC, dorsal attention network, and/or global neural functioning may be needed to examine properly the relationships among psychopathy, the neural metrics of the attention bottleneck, and real-world behavior.

Overall, the current behavioral and neural findings are largely consistent with the predictions made by the attention bottleneck model of psychopathy. However, two alternative interpretations should be considered. First, it is possible that the slower reaction to the 1,100-ms second-target, relative to baseline, found in individuals higher compared to lower on psychopathy may not reflect prolonged bottleneck interference. In prior studies that used neurotypical community samples, RT to the long delayed second target did not significantly differ from RT to a first target (Dux et al., 2006). As noted above, the current study found that participants were significantly faster to respond to the long delayed second target than the baseline. This difference may have been due to variation in

task difficulty. Prior studies required participants to respond to four different potential second target stimuli, making it difficult to anticipate and prepare fully for all possible responses in the time between first and second target onset (Dux et al., 2006). The current study only required participants to respond to one of two potential second target stimuli. Accordingly, there were fewer potential responses to prepare for during the long stimulus onset asynchrony after responding to the first target. Thus, it is possible that participants in the current study were able to better anticipate and prepare for those two potential responses, speeding their reaction to the 1,100-ms second-target compared to baseline. If this is the case, then, individuals higher on psychopathy may simply fail to anticipate and/or prepare for long delayed second target events due to either diminished effort or a neurocognitive deficit unrelated to the bottleneck instead of prolonged bottleneck-related slowing. Therefore, individuals higher on psychopathy may fail to display long delayed second target-related speeding even at the point when the bottleneck no longer impacts information processing. While this alternative interpretation may explain the bottleneck duration effect found in psychopathy, it is worth noting that such an explanation is inconsistent with the current findings demonstrating a greater intensity of bottleneck-related interference for the short delayed second targets in individuals high on psychopathy.

Second, it is conceivable that the blunted P300 responses to second target events may not be related to an exaggerated bottleneck in psychopathy. The timing of our events meant that there was temporal overlap in the participants' neural responses to the first and second targets (particularly for the 300-ms second-target events). In order to mitigate the impact of this timing feature on second target ERPs, single-task baseline ERPs, which were time-locked to the onset of the first target, were subtracted from second target ERPs. Consequently, the ERP analysis could not have a true control condition (since the single-task baseline could not be entered simultaneously into our models independently of the second target). Accordingly, it is still possible that these findings simply represent a general blunting of the P300 response in psychopathy (Brazil et al., 2012), regardless of the specific condition, context, or bottleneck involvement. However, such an explanation seems unlikely given the follow-up analysis demonstrating that psychopathy did not appear to significantly impact P300 responses to single-task baseline targets (see Footnote 4). These alternative interpretations do not diminish the importance of the present study in providing direct evidence of an exaggerated multisensory bottleneck in psychopathy and in demonstrating how such a bottleneck may be relevant to real-world impulsive and antisocial behaviors.

In addition to these alternative explanations, the present results should be considered in light of three limitations. First, while the current sample was elevated on antisocial behavior and psychopathic traits, it was a community sample,

potentially limiting the generalizability of the current findings to incarcerated samples with higher base-rates of psychopathy. Notably, though, the current results are highly consistent with prior research conducted in incarcerated samples demonstrating and characterizing attention abnormalities in psychopathy. Second, only adult participants were included in this study. This impacted our ability to assess at what age aberrancies in the attention bottleneck may emerge, what neurodevelopmental processes may contribute to their emergence, and how these bottleneck-related aberrancies may relate to the initial development and onset of psychopathic traits across the spectrum of psychopathy. Future research examining the relationship between the attention bottleneck and psychopathic traits in developmental samples would be an interesting and necessary step to evaluate these points. Finally, while the results indicate that the duration of bottleneck interference in psychopathy is longer than the duration found in neurotypical individuals (i.e., longer than 1,100 ms), the task was not designed to determine the precise length (i.e., duration) of the bottleneck exaggeration. To identify the true duration of bottleneck-related interference in psychopathy, future research would need to evaluate the effect of psychopathy in various long SOA conditions (in excess of 1,100 ms). This type of manipulation would allow researchers to determine at what point psychopathy-related effects are no longer present.

Overall, despite these limitations, the findings from the present study, combined with previous research, strongly suggest that individuals higher on psychopathy display more intense and longer-lasting bottleneck-related interference. This interference impacts all information processing, regardless of the potential relevance of that information, and contributes to the exceptionally disruptive and maladaptive behaviors displayed by individuals higher on psychopathy. An important extension of this work on psychopathy is the idea that small shifts in basic cognitive functions can have a dramatic impact on behavior and that characterizing these shifts across cognitive functions is important for future work on the neurocognitive underpinnings of psychiatric illness, more generally.

**Supplementary Information** The online version contains supplementary material available at <https://doi.org/10.3758/s13415-021-00891-z>.

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