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# Lexical Inhibition After Semantic Violations Recruits a Domain-General Inhibitory Control Mechanism

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Language processing is incremental. As language signals—for example, words in a sentence—unfold, humans predict and activate likely upcoming input to facilitate comprehension. Prediction not only accelerates understanding but also prompts reassessment in the case of prediction error, fostering learning and refining comprehension skills. Therefore, it is paramount to understand what happens when linguistic predictions are violated—for example, when a sentence ends in an unpredicted word. One theory, which we test here, is that the originally predicted word is actively inhibited after semantic violations. Furthermore, we tested whether this purported lexical inhibition process is achieved by a domain-general mechanism—that is, one that also inhibits other processes (e.g., movement). We combined a semantic violation task, in which highly constrained sentences primed specific words but sometimes continued otherwise, with a motoric stop-signal task. Across two experiments, semantic violations significantly impaired simultaneous action-stopping. This implies that lexical and motor inhibition share the same process. In support of this view, multivariate decoding of electroencephalographic recordings showed early overlap in neural processing between action-stopping (motor inhibition) and semantic violations (lexical inhibition). Moreover, a known signature of motor inhibition (the stop-signal P3) was reduced after this initial overlap period, further suggesting the presence of a bottleneck due to shared processing. These findings show that semantic violations trigger inhibitory processing and suggest that this lexical inhibition recruits a domain-general inhibitory control mechanism. This provides a new perspective on long-standing debates in psycholinguistics, extends the range of a well-characterized cognitive control mechanism into the linguistic domain, and offers support for recent neurobiological models of domain-general inhibitory control.

***Public Significance Statement***

Human language is complex and unfolds rapidly. To process it efficiently, humans automatically predict likely future input in real time: Reading the sentence "Dad cut the turkey with the…" automatically activates the word "knife." But what if the prediction was wrong and the sentence unexpectedly ends in another word? We propose that in such cases, humans actively inhibit the originally predicted word. Indeed, we here found that processing semantic violations (hearing another word when expecting "knife") makes it harder to simultaneously stop an action. Moreover, multivariate decoding of electroencephalography data suggests that there is substantial overlap in the neural activity found during action-stopping (motor inhibition) and the processing of semantic violations (lexical inhibition). This suggests that semantic violations not only trigger an inhibitory process but that this process is shared with other domains (in this case, movement).

*Keywords:* lexical inhibition, inhibitory control, language processing, stop-signal task, stop-signal P3

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Human language is complex, diverse, and often ambiguous. Extensive past research has shown that human language processing is an active process. To aid fast and efficient comprehension of both spoken and written language, the human brain constantly makes predictions about upcoming input (Altmann & Kamide, 1999; Balota et al., 1985; Bannon et al., 2024; Ehrlich & Rayner, 1981; Kamide et al., 2003; Levy, 2008; Ness & Meltzer-Asscher, 2018; Rayner & Well, 1996; Schwanenflugel & LaCount, 1988; Schwanenflugel & Shoben, 1985; Van Berkum et al., 2005). For example, when reading or listening to the sentence "Dad cut the turkey with the …," individuals activate the word "knife" prior to actually seeing or hearing the word. A question of great interest is what happens when these real-time, online predictions of upcoming linguistic input are violated—that is, when the above sentence unexpectedly ends in another word (DeLong & Kutas, 2020; Kim et al., 2023; Kutas, 1993; Ness & Meltzer-Asscher, 2018; Schwanenflugel & Shoben, 1985). One proposed consequence of failed prediction is that the representation of the originally predicted word (knife) is actively suppressed by an inhibitory mechanism (DeLong et al., 2005; Kim et al., 2023; Ness & Meltzer-Asscher, 2018). For example, lexical decision tasks can be used to measure the relative activation of specific words after semantic violations. Studies using this method have shown increased reaction times (RT) for the originally activated word ("knife" in the above example), compared to semantically unrelated words (Kim et al., 2023; Ness & Meltzer-Asscher, 2018). These authors and others (e.g., Kutas, 1993) have argued that this reaction time delay is caused by a suppression of the originally primed word by an inhibitory control mechanism.

Indeed, inhibitory processes are found in many aspects of language processing, including word production, syntax, and pragmatics (Doebel et al., 2018; Ibbotson & Kearvell-White, 2015; Martin-Rhee & Bialystok, 2008; Poarch & van Hell, 2012; Trude & Nozari, 2017). Moreover, primary language disorders, such as developmental language disorder (DLD), are accompanied by atypical inhibitory control (Bishop, 1997; Dosi, 2021; Henry et al., 2012; Im-Bolter et al., 2006; Marton et al., 2007; Spaulding, 2010; Vissers et al., 2015; Weismer et al., 1999). However, there is vigorous debate regarding the nature of the exact mechanisms underlying these inhibitory processes across different aspects of language processing.

On the one hand, classic connectionist models of language processing (e.g., McClelland & Elman, 1986) suggest that during word recognition, lexical representations (i.e., the activation of specific words) are inhibited by a local-level inhibitory mechanism. According to this proposal, inhibition is implemented through lateral inhibition, where activated nodes (target word; e.g., "knife") inhibit the activation of neighboring phonological competitors (e.g., "night"). If semantic violations do indeed lead to inhibition of the initially activated word, this would be one potential candidate mechanism. However, lexical inhibition during semantic processing may involve a different process than phonological processing. For example, some have argued that sentence-level processing may utilize domain-general cognitive control processes, based on the finding that semantic ambiguity is more readily resolved immediately after the performance of an incongruent Stroop trial (Hsu & Novick, 2016). This suggests that processes involved in resolving response conflict may also benefit language processing (see also Adler et al., 2020). Demonstrations of such sequential benefits cannot definitely identify which exact process triggered by the cognitive control task confers the benefit onto subsequent linguistic processing. However, the suggestion that domain-general processes are involved in some types of lexical inhibition is in line with recent neurobiological models, which suggest that a domain-general inhibitory control mechanism may be responsible for inhibiting a broad array of processes, perhaps including language (Depue et al., 2016; Wessel & Anderson, 2024). According to influential models in cognitive psychology, inhibitory control is one of the few general-purpose control mechanisms that regulate all of human cognition (Diamond, 2013; Miyake et al., 2000). Work on the neural underpinnings has shown that inhibitory control is implemented via a fronto-basal ganglia neural circuit (Apšvalka et al., 2022; Aron et al., 2014; Depue et al., 2016; Wessel & Anderson, 2024). This circuit is typically studied during the inhibitory control of action (Aron et al., 2014), memory (Anderson & Green, 2001), or attention (Soh et al., 2024). It achieves inhibition through the interruption of thalamocortical invigoration that underpins those processes (Wessel & Anderson, 2024). For example, active movements, mnemonic, and attentional processes are supported by thalamic invigoration of the cortical aspects of the motor (Dacre et al., 2021), memory (Aggleton & O'Mara, 2022; Staudigl et al., 2012), or attention systems (Kastner et al., 2020; Shine et al., 2023). The inhibitory control circuit can inhibit such thalamocortical invigoration (Wessel & Anderson, 2024). Importantly, similar patterns of thalamocortical invigoration are also found in language processing (Hebb & Ojemann, 2013; Klostermann et al., 2013)—including during the processing of semantic violations (Wahl et al., 2008). Therefore, linguistic representations during semantic violations could be subject to the same, domain-general inhibitory mechanism that regulates movement, memory, and attention. However, the idea that domain-general cognitive control processes, such as inhibitory control, contribute to lexical inhibition is still controversial (Blomquist & McMurray, 2023; Diachek et al., 2020). Indeed, whether semantic violations necessitate inhibition at all is still debated (Kim et al., 2023; Ness & Meltzer-Asscher, 2021). Here, we therefore attempt to provide a direct experimental test of whether lexical inhibition during semantic violations does take place and whether it involves domain-general inhibitory control.

We designed a novel experimental paradigm that required the simultaneous processing of semantic violations and the stopping of an action. We aimed to test whether semantic violations hamper the simultaneous implementation of inhibitory *motor* control—that is, the ability to stop an action. If there is a cost associated with simultaneously processing semantic violations and stopping an action, dual-task logic implies the involvement of a shared, domain-general mechanism (Townsend & Ashby, 1983). If two tasks depend on overlapping and limited processes, the presence of a second task will impair the performance of the first task (Pashler, 1994; Townsend & Ashby, 1983). Conversely, if the processes are independent, the performance of the first task will be unaffected by the second task. While theoretical models differ on the nature of this cost—for example, whether it results from a processing bottleneck (Pashler, 1994), from resource limitation (Navon & Miller, 2002; Tombu & Jolicoeur, 2003), or from crosstalk (Lien & Proctor, 2002; Navon & Miller, 1987)—dual-task costs are ubiquitous in cognitive psychology.

After confirming that there was indeed a substantial dual-task cost in a behavioral experiment, we then performed a second experiment using scalp electroencephalography (EEG) recordings to further investigate the nature and timing of the implied overlap in processing. First, we aimed to compare event-related potentials (ERPs) involved in action-stopping between trials with and without semantic

violations. The goal was to identify the exact stage of the motor inhibition process that is affected by a simultaneously recruited linguistic inhibition process. During the processing cascade underlying action-stopping, (visual) stop-signals are initially followed by a posterior visual N1, which reflects stimulus detection and is common to all visual stimuli (Haider et al., 1964; Mangun & Hillyard, 1991). This activity is subsequently followed by a complex of fronto-central ERPs—chiefly, the N2 and P3 waves—which have been associated with different aspects of the motor inhibition process (de Jong et al., 1990; Diesburg et al., 2024; Huster et al., 2013; Kok et al., 2004; Wessel & Aron, 2015). Investigating these known ERPs would allow us to identify the stage at which the action-stopping process was impaired by the dual-tasking cost. In addition, we then used multivariate cross-conditional decoding analyses (Choo et al., 2023; King & Dehaene, 2014) to further investigate the nature and timing of the overlapping processes between motoric and linguistic inhibition using the whole-scalp EEG data.

## Method

### Participants

A sample size of $N = 31$ was determined a priori to achieve at least 80% statistical power to detect a medium-sized effect (mean difference of .5 with a standard deviation of 1) at a critical α level of .05 for two correlated variables ($r = .4$). The critical effect of interest was the within-subject condition difference in stop-signal reaction time (SSRT; see the Behavioral Analysis section). These calculations were performed using GPower 3 (Faul et al., 2009).

Participants were recruited from the online subject pool in the Department of Psychological and Brain Sciences at the University of Iowa. In Experiment 1, 31 healthy young adults ($M_{age}$ = 19.94, range = 18–28; two left-handed, five self-reported their gender as male, 26 as female) participated in exchange for course credit. In Experiment 2, 32 healthy young adults ($M_{age}$ = 18.34, range = 18–19; seven left-handed, 11 self-reported their gender as male, 21 as female) participated in exchange for course credit. Demographic information was provided via self-report with N/A options for each item. One subject in Experiment 2 did not finish the experiment due to nausea.

### Apparatus and Procedure

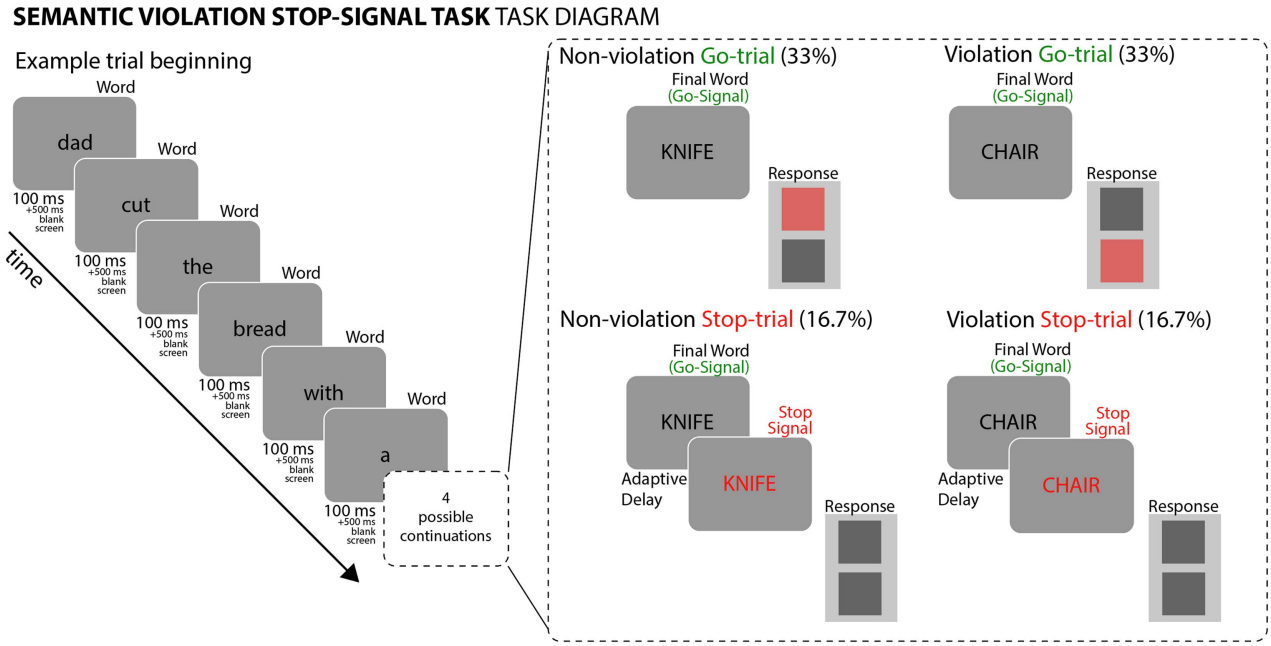
All experiments were conducted in accordance with the Declaration of Helsinki and approved by the University of Iowa Institutional Review Board (IRB No. 201511709). Stimuli were presented using Psychtoolbox 3 (Brainard & Vision 1997) using MATLAB 2021a (TheMathWorks, Natick, Massachusetts) on Ubuntu Linux computers connected to a BenQ XL2420B monitor featuring a 120-Hz refresh rate and a resolution of 1920 × 1080 pixels. Responses were made using a standard QWERTY keyboard.

### Experimental Task

The task is a combination of the classic stop-signal (Logan et al., 1984; Vince, 1948) and semantic violation paradigms (Kutas & Federmeier, 2011; Kutas & Hillyard, 1980). It is depicted in Figure 1. Participants were presented with high-constraint sentences, one word at a time. Sentence stimuli were taken from a published set of sentences with established cloze probability and completion norms (Block & Baldwin, 2010). All sentences ($n$ = 45) were nine words in length with an average

**Figure 1**

*Task Diagram for Experiments 1 and 2*




*Note.* The actual sentences were nine words in length, but a shorter version is presented here to ensure readability. See the online article for the color version of this figure.

cloze probability of .90 (range = .81–.98). To create the violation sentences, the same 45 sentence frames (the first eight words of the sentence) were paired with the sentence endings for other sentences. Precautions were taken to avoid any phonological, semantic, or associative overlap between the violation words and the high-cloze targets (see Supplemental Materials). As a result, some ($n$ = 12) of the predicted words could not be used as violating words and were replaced with other sentence-final words from the completion norms database. Words were presented centrally in black font on a gray background. Each word was presented for 100 ms, followed by a 500-ms blank screen. The final word of the sentence was presented in all caps.

There were two experimental sentence conditions. In the NONVIOLATED sentence condition, the sentence-final word was the predicted, high-cloze word. In the VIOLATED sentence condition, sentences terminated in a semantic violation. Each subject saw each of the 45 sentences eight times, four times with the violated and four times with the nonviolated ending, resulting in 360 trials overall. Participants were instructed to respond to the final word as fast as possible, indicating whether the sentence was sensible (nonviolated endings, "u" key on keyboard) or nonsensical (violated endings, "j" key on keyboard). We chose a vertical key arrangement to avoid potential interactions with reading direction. The final word remained on the screen until a response was made. A response deadline was set at 1,500 ms.

One third of the VIOLATED and NONVIOLATED trials were STOP trials. On those trials, the final word changed from black to red after an adaptive delay (the stop-signal delay [SSD]). Participants were instructed that on such trials, they had to attempt to abort the response and not press any button. This delay was initially set to 250 ms and then adjusted after each STOP trial; after successful STOP trials, the SSD was increased by 50 ms, whereas after failed STOP trials, the SSD was decreased by 50 ms. This was done separately for the VIOLATED and NONVIOLATED trials. It ensures that stopping is consistently challenging and results in a probability of successful stopping of ∼.5 in both conditions, which facilitates the computation of SSRT (see below).

A feedback message appeared 500 ms after the response deadline and remained on the screen for 500 ms. Correct responses on GO trials and correctly withheld responses on STOP trials were followed by "Correct" and "Correct Stop," respectively. Misses (no response within deadline window on GO trials) were followed by a "Faster!" message, whereas errors (wrong response on GO trials) were followed by "Incorrect." Finally, failed STOP trials (STOP trials with a response) were followed by "Please stop." After this feedback and a 500-ms blank screen intertrial interval period, the next trial began.

The 360 total trials were divided into eight blocks of 45 trials. Trials were presented in pseudorandom order, with two constraints: The maximum number of STOP trials in a row was kept to four, and the maximum proportion of STOP trials per block was .4.

## Behavioral Analysis

The primary variable of interest was SSRT (Logan et al., 1984). SSRT is a latent variable that can be derived from the response time data in the stop-signal task, and expresses the speed of the stopping process. Longer SSRT values reflect slower stopping, whereas shorter SSRT values reflect faster stopping. In line with the latest consensus recommendations for SSRT calculation (Verbruggen et al., 2019), SSRT was quantified using the integration method with replacement of go-trial misses. In short, error trials were removed from the SSRT calculation, and RT on miss trials were replaced with the maximum GO trial RT (GORT) value for the same subject. Prior to the SSRT calculation, we also checked whether the assumptions of the horse-race model underlying the SSRT computation were met. Specifically, the horse-race model predicts that GORT needs to be longer than failed STOP trial RT (FSRT), which was confirmed in each subject. In the integration method, SSRT is then estimated by "integrating" the RT distribution and identifying the point at which that integral is equal to the empirical probability of responding on a stop-trial (i.e., $p$(respond|signal)). SSRT then corresponds to the $n$th RT, with $n$ representing the number of RTs in the GORT distribution times $p$(respond|signal). For example, in our case, we presented 240 GO trials. For a hypothetical $p$(respond|signal) of .5 (which is the target outcome of the tracking algorithm), the $n$th RT would then be the 120th fastest GORT. SSRT is then equal to that RT value minus the average SSD.

Finally, we also quantified and compared the other task parameters across the VIOLATION and NONVIOLATION conditions: GORT, FSRT, miss rate, and error rate. All comparisons were made using *paired-samples t tests* comparing VIOLATION and NONVIOLATION trials.

## EEG Recording (Experiment 2 Only)

EEG was acquired using a 63-channel active electrode cap (actiCap Slim, *Brain Products, Garching, Germany*) connected to a *Brain Products* ActiCHamp amplifier. The ground and reference electrodes were placed at AFz and Pz, respectively. EEG was sampled at a rate of 500 Hz with online recording filters at direct current (high pass) and 140 Hz (low pass). Electrode resistances were kept below 10 kΩ.

## EEG Preprocessing

EEG data were imported into MATLAB and preprocessed using an automated pipeline adapted from our previous work (Wessel, 2020), which uses functions from the EEGLAB toolbox (Delorme & Makeig, 2004). Pause periods between blocks were removed from the data. Data were then offline filtered between .3 and 50 Hz and cut into segments of 1 s. These segments were then scanned for artifacts using EEGLAB's jointprob() and rejkurt() functions, with values of 5 *SD*s for each method (Delorme et al., 2011). After rejection of segments outside of those boundaries, the data were rereferenced to a common average montage and submitted to an infomax independent component analysis decomposition. The resulting component matrices were then visually inspected for stereotypic artifact components (blinks, saccades, electrode artifacts), which were removed from the data by means of selective backprojection. The resulting, cleaned channel EEG data were subjected to further analyses. Data from two subjects were removed prior to final EEG analyses due to excessive overall noise levels.

## ERP Analysis

For the purposes of the primary ERP analyses, the cleaned data were epoched from −500 ms to 1,000 ms relative to the event of interest, which was the stop-signal. The primary ERPs of interest were

the posterior visual N1, which reflects detection of the visual stop-signal, as well as the fronto-central stop-signal N2 and P3, a complex of negative positive voltage deflections over central electrode sites on successful STOP trials.

The STOP trial ERP was time-locked to the stop-signal. Since there was no stop-signal on GO trials, we instead selected the nearest STOP trial (in terms of absolute difference in the trial sequence) of the same condition (VIOLATION, NONVIOLATION) for each individual GO trial, identified the SSD on that STOP trial, and time-locked the GO trial activity to the same time point after the GO signal. In essence, this is the time point at which the STOP signal would have appeared if the trial had been a STOP trial. This approach ensures that STOP and GO trial activity are closely matched, especially in the baseline period before the "STOP" signal did (on STOP trials) or would have (on GO trials) appeared (Wessel & Aron, 2015). We then compared the four condition ERPs (STOP-VIOLATION, STOP-NONVIOLATION, GO-VIOLATION, GO-NONVIOLATION) using sample-to-sample repeated measures analyses of variance. The resulting vectors of $p$ values (one $p$ value per ERP time point for each of the main effects for STOP and VIOLATION, as well as their interaction) were corrected for multiple comparisons using the false discovery rate method (FDR, Benjamini et al., 2006). This sample-to-sample testing approach prevents bias in the analysis because it does not necessitate the choice of specific analysis windows in the ERP period, a common researcher degree of freedom. In accordance with the existing literature, the posterior visual N1 was quantified at electrode Oz and the fronto-central stop-signal N2/P3 complex was quantified at electrode Cz. These locations were also confirmed post hoc via voltage topographies. We expected that the N2/P3 complex would only be present on STOP trials (i.e., a main effect of STOP vs. GO) and that the effect of VIOLATIONS on this P3 would be exclusive to STOP trials (i.e., a significant interaction). We also directly compared the STOP trial ERPs between the VIOLATION and NONVIOLATION conditions using sample-to-sample paired-samples $t$ tests, once again corrected for multiple comparisons across samples using the FDR method.

Furthermore, to illustrate and verify the established association between the P3 and SSRT (Huster et al., 2020; Wessel & Aron, 2015) in our data set, we correlated the P3 peak amplitude with SSRT across subjects using separate Pearson correlations for both trial types (VIOLATION, NONVIOLATION). This association has been shown previously in larger samples (Huster et al., 2020; Wessel & Aron, 2015).

Finally, as a manipulation check, we also plotted the N400 ERP (Kutas & Hillyard, 1980) time-locked to the onset of the terminal word. In line with the previous literature, we expected an increase in the N400 for VIOLATION compared to NONVIOLATION trials. This was tested with the same sample-to-sample, FDR-corrected $t$ test method as the main hypothesis regarding the stop-signal P3. In accordance with the existing literature, the N400 was quantified at centro-parietal electrode CPz. This location was also confirmed post hoc via the voltage topography at the peak of the N400.

## Multivariate EEG Decoding Analysis: Motor Inhibition and Linguistic Inhibition

In addition to the ERP analysis, we also conducted multivariate pattern analysis of the whole-scalp EEG activity using the MVPA-Light toolbox (Treder, 2020), following the same approach from our previous work (Choo et al., 2023). The preprocessed data were downsampled to 250 Hz and segmented around −100 to 1,000 ms with respect to the stimuli of interest. Each epoch was baseline corrected with the 100-ms prestimulus window. We trained and tested two multivariate classifiers for pair-wise comparisons. A STOP-NONVIOLATION versus GO-NONVIOLATION decoder was trained to distinguish EEG activity related to motor inhibition, in the absence of linguistic inhibition demands. Conversely, a GO-VIOLATION versus GO-NONVIOLATION decoder was trained to distinguish EEG activity related to linguistic inhibition, in the absence of motor inhibition demands. Trial numbers were matched for each decoder via random sampling from the condition with more trials. The motor inhibition decoder used 24.31 trials per condition on average (min: 17, max: 30), due to the fact that only a third of trials were STOP trials. The linguistic inhibition decoder used 63.93 trials per condition on average (min: 49, max: 78). A linear discriminant classifier was trained and tested at each time point for each subject separately, using a fourfold cross-validation procedure. Trials from each subject were randomly separated to four sets, with one set held out as a test set and the remainder of the data used as training sets. This procedure was iterated four times until each set served as a testing data set. This procedure was repeated five times, and the resulting decoding performance was averaged across each repetition. Each subject's decoding performance was then averaged together to be used in group-level analysis. We used the area under the curve metric to evaluate the quality of decoding performance for both decoders. To assess statistical inference, each participant's area under the curve per time point was submitted to paired-samples Wilcoxon signed-rank test against chance-level performance (0.5). The resulting $p$ values were corrected for multiple comparisons using cluster-based permutation tests (1,000 iterations, $p < .05$, "maxsum" cluster correction, Maris & Oostenveld, 2007).

## Multivariate EEG Decoding Analysis: Cross-Decoding

After training individual classifiers to detect EEG activity related to motor or linguistic inhibition, we then tested whether the EEG data supported the notion of shared processing. This was tested using a cross-decoding analysis (Choo et al., 2023; King & Dehaene, 2014), wherein a decoder trained on one condition difference (in our case, the motor inhibition decoder trained to distinguish STOP-NONVIOLATION from GO-NONVIOLATION trials) was tested for its ability to decode another condition contrast (in our case, GO-VIOLATION from GO-NONVIOLATION trials). For this analysis, the segmented EEG data were first $z$-scored. A time-by-time temporal generalization matrix was then obtained for each subject. As this process is stochastic (due to random assignment of trials for each data set), we repeated this process 10 times and averaged the results to derive a single, reliable generalization matrix for each subject. Each participant's decoding performance in this time-by-time matrix was then submitted to Wilcoxon sign-rank test against chance-level performance. Multiple comparisons were again corrected with cluster-based permutation test with the critical cluster $p$ value of 0.05 (1,000 iterations, $p < .05$; weighted cluster mass correction; weight = 1, Maris & Oostenveld, 2007).

## Transparency and Openness

We report how we determined our sample size, all data exclusions, all manipulations, and all measures in the study. All raw data, analysis code, and task material and code are available on the Open Science Framework at https://osf.io/6jen8. Data were analyzed using MATLAB 2023b (TheMathWorks, Natick, Massachusetts) and the EEGLAB toolbox (Delorme & Makeig, 2004).

# Results

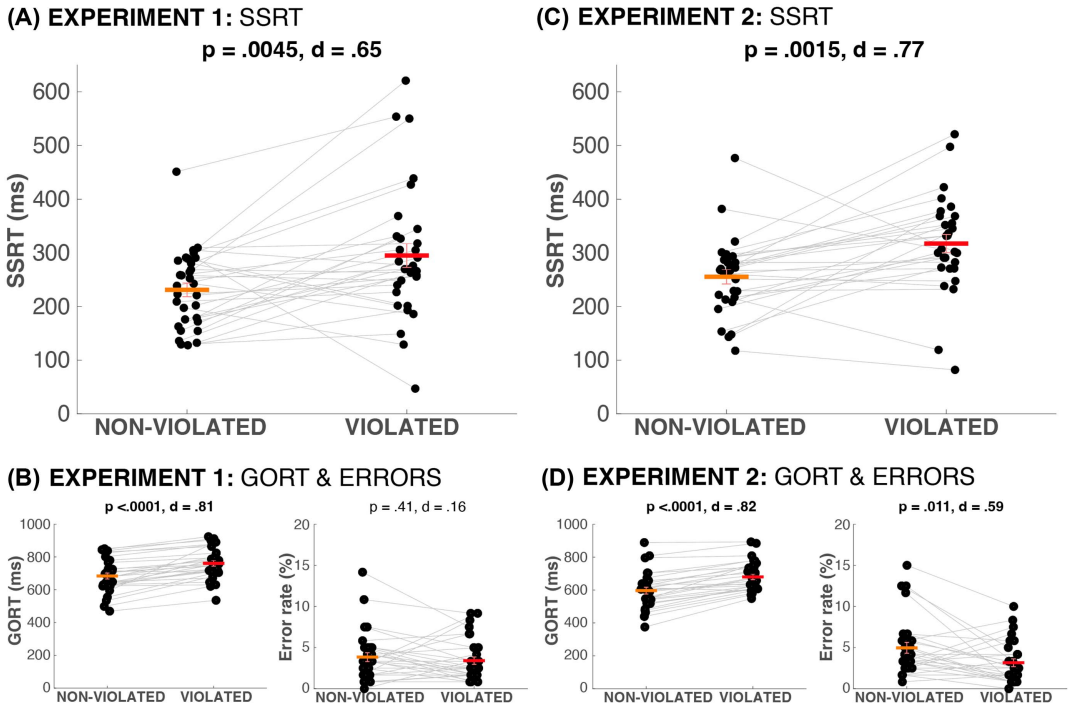
## Behavior (Experiments 1 and 2)

Behavioral results for Experiment 1 are depicted in Figure 2A–2B. All subjects' behavioral data conformed to the predictions of the horse-race model (i.e., GORT was longer than FSRT), and the SSD staircases for both sentence conditions successfully converged around a stopping success rate of .5 (.54 in the NONVIOLATION and .55 in the VIOLATION condition, with no differences between conditions, $p > .5$). The average SSD was 411 ms in the NONVIOLATION condition and 413 ms in the VIOLATION condition, with no significant differences between conditions ($p > .915$, $d = .015$). With regard to our primary hypothesis test, VIOLATION trials featured significantly increased SSRT values compared to NONVIOLATION trials, $t(30) = 3.07$, $p = .0045$, $d = .65$. The average increase in SSRT was 64 ms ($M$: 231 ms, standard error of the mean [SEM]: 13 ms vs. $M$: 295 ms, SEM: 22 ms).

With regard to the secondary variables, GORT, 683 ms versus 761 ms, $t(30) = 7.21$, $p < .0001$, $d = .81$, and FSRT, 562 ms versus 643 ms, $t(30) = 7.63$, $p < .0001$, $d = 1.1$, were also increased in the VIOLATION condition. This is particularly interesting because in the stop-signal task, slower responding (i.e., longer GORT) typically benefits stopping and, hence, reduces SSRT (e.g., Chikazoe et al., 2009; Greenhouse & Wessel, 2013; Verbruggen & Logan, 2009). However, in our VIOLATION condition, SSRT was elongated despite the slower GORTs. Moreover, GO trial error rates, 3.84% versus 3.41%, $t(30) = .84$, $p = .41$, $d = .16$, and miss rates, 1.48% versus 1.42%, $t(30) = .19$, $p = .85$, $d = .04$, were both low and did not differ between the two conditions. This shows that the SSRT effect was not due to a general performance decrement in the VIOLATION condition.

Experiment 2 directly replicated these results (Figure 2C–2D). Once again, all subjects' behavioral data conformed to the predictions of the horse-race model (GORT > FSRT) and the SSD staircases for both sentence conditions successfully converged around a stopping success rate of .5 (.52 in both conditions, with no significant differences, $p > .2$). The average SSD was 316 ms in the NONVIOLATION condition and 338 ms in the VIOLATION condition, with no significant differences between conditions ($p > .07$, $d = .2$). With

**Figure 2**
*Behavioral Results*




*Note*. (A) Main hypothesis test for Experiment 1. SSRT was significantly elongated on VIOLATION trials. Black dots depict individual subjects, and gray lines connect the subjects between conditions. Horizontal lines depict the condition mean, and error bars depict the standard error of the mean. (B) Other relevant data from Experiment 1, showing increased GORT for VIOLATION trials and no difference in error rates between conditions (demonstrating that the SSRT effect is not due to a general performance decrement in the VIOLATION condition). (C–D) As in A and B, but for Experiment 2. SSRT = stop-signal reaction time; GORT = GO trial RT; RT = reaction time. See the online article for the color version of this figure.

regard to our primary hypothesis test, VIOLATION trials once again featured significantly increased SSRT values compared to NONVIOLATION trials, $t(28) = 3.499$, $p = .0015$, $d = .77$. The average increase in SSRT was 62 ms (*M*: 255 ms, SEM: 13 ms vs. *M*: 317 ms, SEM: 17 ms).

With regard to the secondary variables, GORT, 597 ms versus 680 ms, $t(28) = 8.49$, $p < .0001$, $d = .82$, and FSRT, 482 ms versus 577 ms, $t(28) = 8.92$, $p < .0001$, $d = 1.1$, were again increased in the VIOLATION condition. Moreover, GO trial error rates, 4.97% versus 3.16%, $t(28) = 2.735$, $p = .011$, $d = .59$, and miss rates, .83% versus 1.03%, $t(28) = .72$, $p = .47$, $d = .16$, were once again low. In this experiment, the error rate was actually significantly decreased in the VIOLATION condition, once again at odds with the possibility that the elongated SSRT in the VIOLATION condition could be due to a general performance decrement.

Since both experiments showed the same pattern of effects, we also combined the two samples to derive a more reliable estimate of the true effect size of our effects of interest. In this combined sample, the *t* test of SSRT between the two conditions resulted in $t(59) = 4.6$, $p < .0001$, with an effect size of $d = .69$ and a raw SSRT difference of 63 ms (245 ms vs. 308 ms). GORT, $t(59) = 11.1$, $p < .0001$, $d = .75$, and FSRT, $t(59) = 11.7$, $p < .0001$, $d = .99$, were increased in the VIOLATION condition, just like in the individual experiments. GO trial error rates were significantly lower in the VIOLATION condition across the samples, 4.4 versus 3.3%, $t(59) = 2.61$, $p = .011$, $d = 0.37$. SSD and miss rate did not differ significantly (both $p > .27$).

In response to a reviewer suggestion, we also tested whether the repeated usage of the same 45 sentences had an influence on the SSRT effect. We split each datafile into subsets that included either only the first or only the second half of the individual instances of each sentence. The SSRT effect was significant in both cases, first half of instances: $t(59) = 2.81$, $p = .007$, $d = 0.41$; second half of instances: $t(59) = 2.82$, $p = .007$, $d = 0.52$.

## Event-Related Potentials (Experiment 2)

Figure 3A shows the stop-signal locked ERP at electrode Cz. As expected, the ERP was dominated by a sizable stop-signal P3 component with a preceding N2, which were present in both STOP conditions, but not in the GO conditions. This resulted in a long-lasting main effect of STOP (significant time ranges: 2–102 ms and 130–818 ms, FDR-corrected critical *p* value: .0457). Crucially, the P3 amplitude was significantly reduced in VIOLATION compared to NONVIOLATION trials. This was shown both by a significant STOP × VIOLATION interaction at the exact time of the P3 peak (360–370 ms; FDR-corrected critical *p* value: .00035; red-and-black line at the bottom of the graph), as well as a direct comparison of the two STOP conditions via a paired-samples *t* test ($p < .05$, FDR-corrected to a critical *p* of .00064), which was significant in the time period from 362 to 382 ms after the STOP signal (see gray highlighting on the ERP plot). The topography of the condition difference at the peak latency of the ERP (368 ms) confirmed the typical location of the effect, centered around electrode Cz (Figure 3A, inset). Finally, there was main effect of VIOLATION in the time range after the P3 component (554 ms–716 ms, FDR-corrected critical *p* value: .0059). There was no similar effect observed in the preceding fronto-central N2, nor in the posterior-visual N1 to the stop-signal (Figure 3C), suggesting that the first stage of motor inhibition that was impaired by the processing of semantic violations was reflected in the fronto-central stop-signal P3.

We also performed a peak-amplitude analysis on the P3 window (250–450 ms), following a reviewer suggestion. This analysis showed the same result: a significant main effect of STOP, $F(1, 28) = 176.4$, $p < .0001$, $\eta^2 = .86$; no main effect of VIOLATION, $F(1, 28) = .81$, $p = .38$, $\eta^2 = .03$; and a significant STOP × VIOLATION interaction, $F(1, 28) = 6.78$, $p = .015$, $\eta^2 = .19$.

Moreover, illustrating the relationship between the P3 ERP and inhibitory motor control in our data set and replicating prior work, the P3 amplitude was significantly correlated with SSRT in both conditions, $r = -.47$, $p = .0086$ in the NONVIOLATION and $r = -.37$, $p = .047$ in the VIOLATION condition (Figure 3B). Subjects with shorter SSRTs (faster stopping) had larger P3 amplitudes.

In response to a reviewer suggestion, we also quantified the variance in the individual P3 condition peak latencies, to rule out that any amplitude effects were caused by jitter. There was no significant difference in peak latency between the STOP-VIOLATION (356 ms) and STOP-NONVIOLATION (360 ms) conditions, $t(28) = .47$, $p = .64$, $d = .078$, and the standard error of the peak latency distributions was virtually identical (10.88 ms vs. 10.03 ms).

For the sake of completion (and as a manipulation check), Figure 4 shows the terminal-word locked ERP in the VIOLATION and NONVIOLATION conditions. As expected, the ERP was dominated by a sizable N400 component, centered around electrode CPz. Significant periods (FDR-corrected critical *p* value: .027) were observed from 220 to 450 ms, 668 to 692 ms, 700 to 988 ms, and 994 to 1,000 ms after the onset of the terminal word (see gray highlighting on the ERP plot).

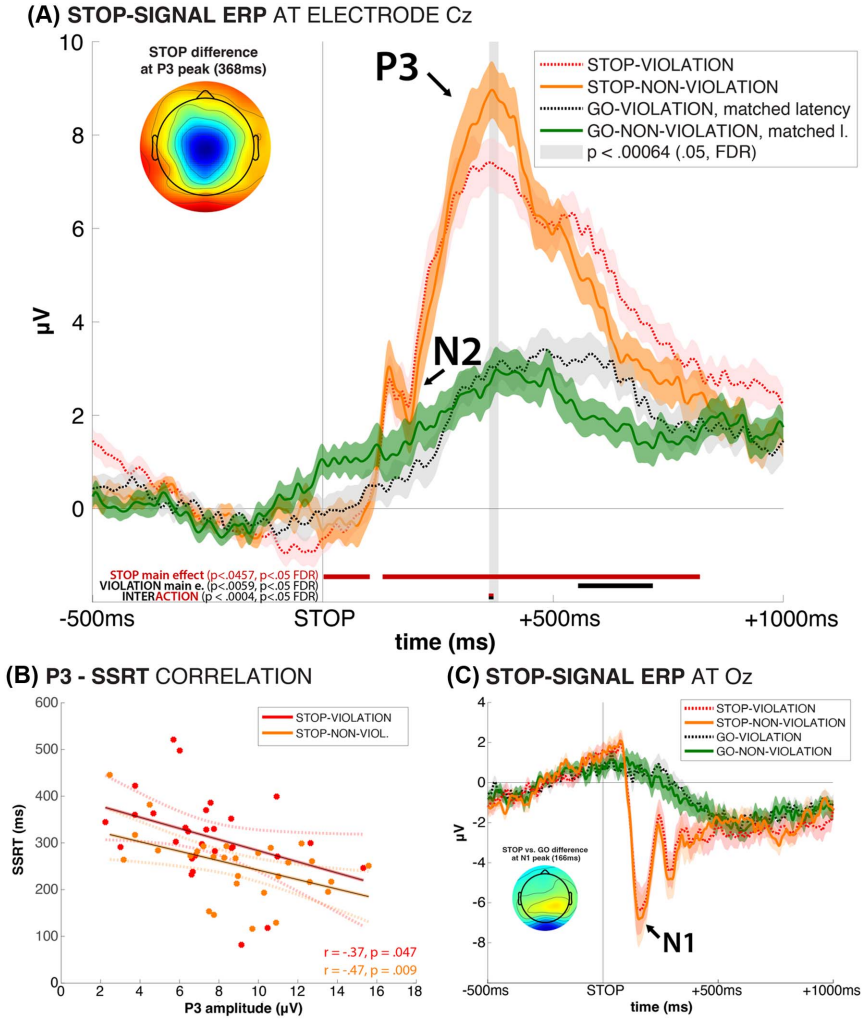
## Multivariate EEG Cross-Decoding (Experiment 2)

Both separately trained decoders were able to reliably decode the condition differences of interest from the whole-scalp EEG data (Figure 5A): STOP-NONVIOLATION versus GO-NONVIOLATION for the "motor inhibition" decoder (purple line), and GO-VIOLATION versus GO-NONVIOLATION for the "lexical inhibition" decoder (turquoise line), respectively. For the ease of reading, we will from here on refer to these decoders as the "motor inhibition" and "lexical inhibition" decoders.

The cross-condition decoding analysis revealed three significant clusters at which the motor inhibition decoder could also distinguish between GO-VIOLATION and GO-NONVIOLATION trials (Figure 5B). These clusters indicate time points at which neural processing is shared. The inverse analysis—using the lexical inhibition decoder to delineate STOP-NONVIOLATION from GO-NONVIOLATION trials—showed the same three clusters, as well as a fourth cluster, which was not significant in this analysis (cf. Supplemental Figure 1).

The first cluster (black outline) suggests why the stop-signal P3 is reduced—and motor inhibition is impaired—on VIOLATION trials. This cluster indicates that the process that distinguished STOP from GO in the 100–200 ms period after the stop-signal (i.e., right before the emergence of the stop-signal P3) also distinguished GO-VIOLATION from GO-NONVIOLATION trials, approximately 400–700 ms after semantic violations. Hence, on STOP-VIOLATION trials, there is a period of processing bottleneck, crosstalk, or resource sharing

**Figure 3**
*ERP Results*

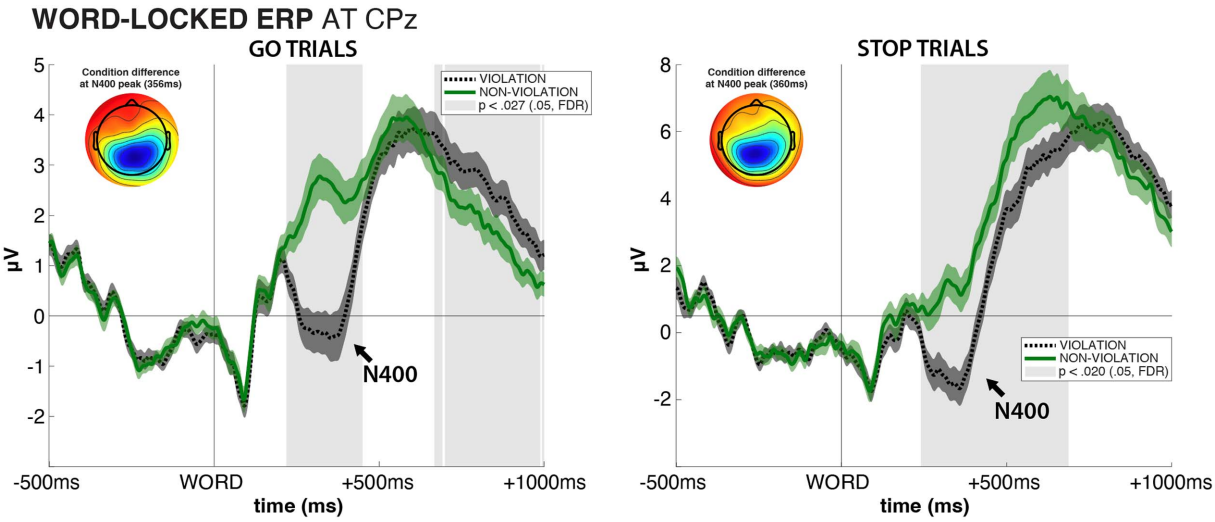



*Note*. (A) Grand-average stop-signal locked ERP at electrode Cz. A sizable and significant P3 component is visible in both STOP conditions, with a significantly decreased amplitude in the VIOLATION condition ($p < .05$, sample-to-sample paired $t$ test, FDR-corrected to .00064; gray highlighting). The P3 is preceded by an N2 waveform, which does not differ between the two STOP conditions. Waveform shading depicts the standard error of the mean. The topographical difference between both STOP conditions shows that the typical P3 scalp distribution centered around Cz. The lines at the bottom show FDR-corrected periods of significant main effects and interactions from the sample-to-sample analysis of variance, alongside their FDR-corrected critical $p$ values. (B) The P3 amplitudes were negatively correlated with SSRT in both conditions; larger P3 amplitudes accompanied faster stopping/shorter SSRT. (C) Grand-average stop-signal locked ERP at electrode Oz. A sizable and significant visual N1 component is visible in both STOP conditions, with no significant condition differences. Waveform shading depicts the standard error of the mean. The topographical difference between the STOP and latency-matched GO conditions shows that the typical posterior-occipital N1 scalp distribution. SSRT = stop-signal reaction time; ERP = event-related potential; FDR = false discovery rate; VIOL. = VIOLATION. See the online article for the color version of this figure.

right before the stop-signal P3 period. Indeed, since the SSD was ~300–350 ms on average (i.e., the stop-signal was presented ~300–350 ms after the presentation of the terminal word on STOP-VIOLATION trials), the process reflected in this cluster would be taxed by the stop-signal 100–200 ms later. That time period overlaps with the 400–700 ms period during which the terminal word also activates that same process (Figure 5C).

In addition, the second cluster (red outline) reflected a second period of overlapping processing, which, in the STOP condition, matched the P3 time period (roughly 250–600 ms after the

**Figure 4**
*N400 Event-Related Potential*




*Note*. Grand-average ERP time-locked to the terminal word in VIOLATION (black) and NONVIOLATION (green) conditions, showing a sizable and significant N400 component in the VIOLATION condition ($p < .05$, FDR-corrected to .027, gray highlighting). Left: GO trials. Right: STOP trials. Waveform shading depicts the standard error of the mean. ERP = event-related potential; FDR = false discovery rate. See the online article for the color version of this figure.

stop-signal). That process is also active around 700–1,000 ms after semantic violations.

The third cluster (blue outline) begins at around 450 ms following both types of critical event (the stop-signal in the "motor inhibition" decoder and the terminal word in the "linguistic inhibition" decoder). Since this is right around the time of the motor response, it is likely that this cluster reflects response-related activity in both contrasts.

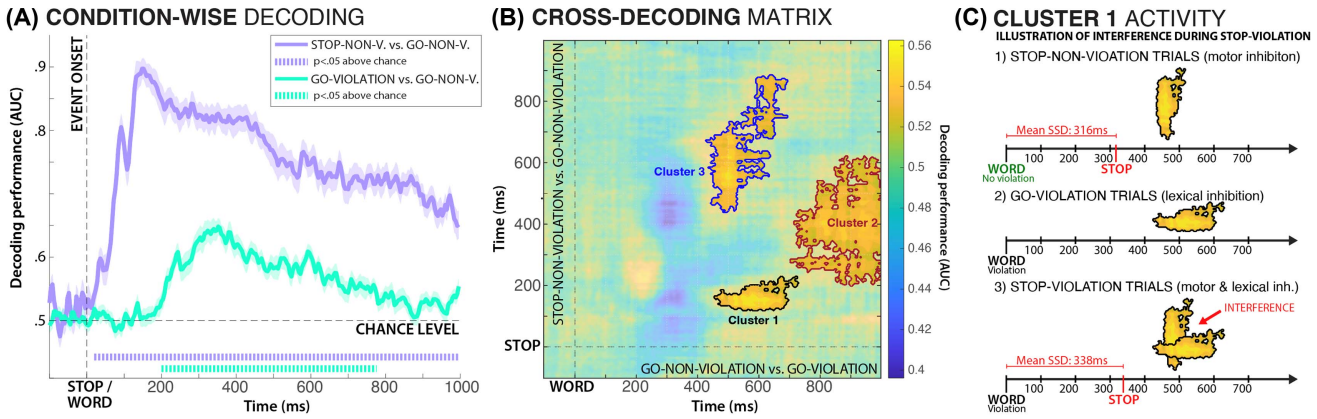
## Discussion

We present evidence suggesting that the processing of semantic violations involves an inhibitory process. Using a dual-task design and multivariate EEG decoding analyses, we found that pairing semantic violations with a simultaneous motoric stop task requirement led to substantial elongation of SSRT, a latent variable that expresses the speed of action-stopping. This effect replicated across two studies, with a medium-to-large effect size ($d = .69$, raw difference: 63 ms). According to dual-task logic, this indicates shared processing between action-stopping and the processing of semantic violations. This is in line with the proposal that semantic violations trigger a lexical inhibition process (Kutas, 1993), which suppresses the initially activated but since-violated word (Kim et al., 2023; Ness & Meltzer-Asscher, 2018). Furthermore, these results indicate that this type of lexical inhibition during semantic violations recruits a domain-general inhibitory control mechanism—one that is also recruited to stop actions. Engaging both processes simultaneously results in interference (either due to resource limitations, a processing bottleneck, or crosstalk; see introduction), as evident from the impaired SSRT in STOP-VIOLATION trials in the current experiment. Our ERP and EEG decoding analyses provide additional insights regarding the timing of the shared processing. First, the ERPs indicate that the presence of a semantic violation does not affect the posterior N1 to the visual stop-signal nor the fronto-central N2 wave. However, semantic violations did lead to a significant reduction of the stop-signal P3. This suggests that the interference occurs prior to the processing stage reflected in the stop-signal P3. Indeed, the multivariate cross-condition decoding analysis of the whole-scalp EEG data suggests that this is indeed the case, as both motor inhibition and linguistic inhibition draw upon the same process that occurs around 100–200 ms after stop-signals or 400–650 ms after semantic violations.

This core finding was bolstered by two additional, important behavioral observations, which rule out alternative interpretations: the concomitant increases in GO trial reaction times (GORT) in the VIOLATION condition, as well as the decreased error rates in that condition. First, in addition to SSRT increases, semantic violations also led to a significant increase in GORT. In the stop-signal paradigm, such increases in GORT should be beneficial to stopping, as the success of stopping depends on the outcome of a race between the stop-process (triggered by the stop-signal) and the go-process (Logan et al., 1984). Hence, all else being equal, systematically slower GORT should make it easier for the stop-process to "win the race" and lead to more successful stopping—and hence, faster SSRT (Verbruggen & Logan, 2009). For example, when humans know that they may very likely have to stop a particular movement, they can trade off GORT speed, slow down their responses, and thereby shorten their SSRT (Chikazoe et al., 2009; Verbruggen & Logan, 2009). Therefore, it is remarkable that the SSRT deficit in the VIOLATION condition was observed *despite* the concomitant increase in GORT. There are two potential explanations for this pattern of results. The first explanation is that the VIOLATION condition was more difficult or involved greater conflict than the NONVIOLATION condition. This would lead to a nonspecific impairment on behavior—that is, performance would decrease across the board. However, this explanation was contradicted by the second important behavioral observation from the current set of

**Figure 5**

*EEG Decoding Results*




*Note.* (A) Decoders trained separately on the "motor inhibition" contrast of STOP-NONVIOLATION versus GO-NONVIOLATION trials (purple) and on the "lexical inhibition" contrast of GO-VIOLATION versus GO-NONVIOLATION trials (turquoise) were both successful in distinguishing the respective trial types. (B) The cross-decoding matrix shows that the "motor inhibition" decoder can be used to decode the "lexical inhibition" contrast at three different temporal clusters, which indicates significant periods of shared neural processing. (C) Hypothetical illustration of the source of processing interference on STOP-VIOLATION trials, illustrated by the neural activity in Cluster 1 shown in B. On STOP-NONVIOLATION trials (no lexical inhibition requirement), Cluster 1 activity was found ~100–200 ms after stop-signal presentation (compared *y*-axis in B). On GO-VIOLATION trials (no motor inhibition requirement), Cluster 1 activity was found ~450–650 ms after the terminal word (compare *x*-axis in B). On STOP-VIOLATION trials (where both motor and lexical inhibition are required), both functions draw upon the Cluster 1 process at the same time. This crosstalk/bottleneck/resource limitation is likely responsible for both the reduced stop-signal P3 on STOP-VIOLATION trials, and—more importantly—for the impaired motor inhibition on these trials. AUC = area under the curve; SSD = stop-signal delay; V. = VIOLATION; inh. = inhibition; EEG = electroencephalography. See the online article for the color version of this figure.

experiments: Error rates did not significantly differ between the two conditions in Experiment 1 and were in fact significantly *decreased* in the VIOLATION condition in Experiment 2 and the combined sample. This pattern is incommensurate with higher conflict in that condition. Further speaking against this hypothesis, Bissett et al. (2023) have paired a large battery of tasks with stop-signal requirements in a dual-task situation (similar to what we did here) and have found that the presence of conflict in one task component (e.g., in a flanker paradigm) does not increase SSRT to a simultaneously presented stop-signal. In other words, in existing work, conflict by itself did not elongate SSRT, unlike semantic violations in the present study. Therefore, we instead propose that both the SSRT and GORT effects in the present study reflect increased inhibitory control activity during the processing semantic violations. On STOP-VIOLATION trials, the dual strain on this process interferes with stopping and elongates SSRT, as further suggested by our EEG decoding analysis (Figure 5C). On GO-VIOLATION trials, we suggest that the inhibitory motor control mechanism underlying SSRT is partially recruited in an effort to "brake" behavior, rather than to stop it outright (Aron et al., 2014; Wessel & Aron, 2014).

The finding that lexical inhibition, at least in the case of semantic violations, is carried out by a domain-general mechanism has important theoretical implications for language processing, executive functions, and beyond. In the linguistic domain, it speaks to a long-standing debate regarding the nature of lexical inhibition. Inhibitory processes have been investigated or proposed in several aspects of language processing (DeLong & Kutas, 2020; Gernsbacher & Faust, 1991; Kim et al., 2023; Mirman & Britt, 2014; Schwanenflugel & Shoben, 1985; Shokrkon & Nicoladis, 2022). Prominent computational models of word recognition, for example (McClelland & Elman, 1986), typically suggest that lexical inhibition recruits a local-level mechanism. For example, during spoken word recognition, listeners coactivated multiple words that are consistent with the unfolding signal (e.g., when hearing *cat*, *can* is also activated). In this context, lexical inhibition is often conceptualized as a highly localized process by which the more dominant word (*cat*) actively suppresses competitor words (*can*) via lateral inhibition. However, others have suggested that linguistic processes can also involve domain-general control processes. For example, performing a task that requires cognitive control can benefit subsequent language processing (e.g., Hsu & Novick, 2016). Conversely, performing a linguistic operation (such as a code-switch between two languages) can benefit performance in a subsequent nonlinguistic cognitive control task (Adler et al., 2020). Of course, while work of this kind may indeed be indicative of processing overlap between nonlinguistic control situations and language processing, there are potential alternative explanations associated with the presence of these sequential benefits. For example, sequential benefits can arise if one type of event triggers a process that is helpful to the processing of a second type of event, but would not be triggered by the second type of event on its own. Here, we approached the question using a different method, demonstrating both dual-task costs that occur during the simultaneous occurrence of action-stopping and semantic violations, as well as EEG-based cross-decoding—both of which strongly suggest that both events (here, semantic violations and stop-signals) trigger the same process. As such, the present study suggests that—at a very minimum—lexical inhibition during semantic violations does indeed recruit a domain-general inhibitory control mechanism. Notably, this does not imply that lexical inhibition is solely implemented by this

domain-general mechanism, nor does it imply that such a mechanism contributes to all instances of lexical inhibition (especially those outside of sentence-level semantic processing). Moreover, it is possible that domain-general and domain-specific processes both play a role, depending on the exact linguistic function (e.g., Fedorenko, 2014; Fedorenko et al., 2012). Indeed, semantic violations might be a special case; in other cases, lexical inhibition may be implemented solely via a domain-specific mechanism. Finally, some have also argued that control processes are not active during passive listening tasks (e.g., Diachek et al., 2020), and that hence, most findings of cognitive control activity during language may be an artificial by-product of task design. However, in that work, the presence (or absence) of control is usually indirectly inferred from lack of activity within one specific brain network that is shared by several control processes (i.e., reverse inference; Poldrack, 2011). In contrast, the current results clearly provide direct, positive evidence for the presence of a specific process for domain-general inhibitory control during lexical inhibition after semantic violations.

In addition to these considerations, these results also have strong implications for influential models of executive functions. According to one of the most influential theories in cognitive psychology (Miyake et al., 2000), inhibitory control is one of three general purpose mechanisms that regulate the dynamics of human cognition (the others being updating and switching; Diamond, 2013). Indeed, neurobiological models have proposed a domain-general view of inhibitory control. One recent model posits that the principle of "inhibition by interruption of thalamocortical drive" can be applied to all processes that are governed by thalamocortical circuits (Wessel & Anderson, 2024). In accordance with this, recent work has shown that the same inhibitory neural mechanism that is involved in stopping actions also suppresses intrusive memories (Apšvalka et al., 2022), emotions (Depue et al., 2016), and attention (Soh et al., 2024; Soh & Wessel, 2021). The present study suggests that the same may be true for aspects of language—in line with the known role of thalamocortical processes during language processing (Hebb & Ojemann, 2013; Klostermann et al., 2013). The possibility of extrapolating existing knowledge about how this mechanism inhibits memories and actions into the linguistic domain opens the door to a vast array of future research. For example, it is valuable to know that the shared process between semantic violations and action-stopping appears to take place early after a stop-signal. The cross-decoding analysis in Figure 5 and the reduction of the stop-signal P3 on STOP-VIOLATION trials suggest that activity that begins ~400 ms after semantic violations also takes place around 100–200 ms after stop-signals—but before the stop-signal P3. According to recent theories of inhibitory motor control, actions are stopped in a cascade of two processes (Diesburg & Wessel, 2021; Schmidt & Berke, 2017; Wessel, 2024). The first process is a broad inhibitory "pause," which is shared between action-stopping and other control-demanding situations, such as action errors (Guan & Wessel, 2022) and unexpected perceptual events (Vasilev et al., 2023). This "pause" is ostensibly signified by a broad inhibition of the motor system, which takes place within the first 200 ms of a stop-signal (Wessel et al., 2022). The second proposed phase of action-stopping is an action-specific, selective "cancel" process that is unique to action-stopping (and selective to the stopped response, rather than broadly affecting the entire motor system). This "cancel" process is ostensibly reflected in the stop-signal P3 (Tatz et al., 2021). Therefore, our present study suggests that semantic violations share the initial "pause" stage with action-stopping, thereby impairing the implementation of the subsequent "cancel" phase when actions have to be simultaneously stopped. This extrapolation generates immediately testable hypotheses for future work—for example, that semantic violations should lead to a broad suppression of the motor system, similar to other "pause"-generating events.

Finally, this work may have important clinical implications. For example, extensive behavioral research has suggested a role for inhibitory control in DLD (Bishop, 1997; Dosi, 2021; Henry et al., 2012; Im-Bolter et al., 2006; Marton et al., 2007; Spaulding, 2010; Vissers et al., 2015; Weismer et al., 1999). DLD is one of the most common neurodevelopmental disorders and affects an estimated 7%–12% of children (Tomblin et al., 1997). It is characterized by difficulties in the ability to learn and use spoken and written language. The development of inhibitory control has been shown to be delayed in children with DLD compared to children with typical language. Specifically, individuals with DLD have greater difficulty inhibiting their actions and prepotent responses (Im-Bolter et al., 2006; Spaulding, 2010); visual, auditory, and linguistic distractors (Im-Bolter et al., 2006); and, as in the present study, out-of-date lexical representations (Weismer et al., 1999). Thus, an understanding of the behavioral and neurophysiological mechanisms of inhibitory control during language processing provides insights into the fundamental processes involved in language and communication. This knowledge is essential for developing targeted assessments and interventions to address specific aspects of the language system affected by disorders like DLD.

In sum, we here provide behavioral and neural evidence for the involvement of a domain-general inhibitory control mechanism in the inhibition of linguistic information. This addresses a long-standing debate in the field of cognitive psychology and psycholinguistics and has important additional implications for neurobiological and clinical research.

## Constraints on Generality

We report that semantic violations triggered domain-general inhibitory processing. However, there are many other situations in which language processing may require inhibitory control (e.g., inhibition of phonemes rather than words), but does not draw upon a domain-general mechanism. There may also be situations in which lexical inhibition (i.e., of words) is not carried out by the same mechanism—that is, the mechanism described here may be specific to semantic violations.

As mentioned in the discussion, semantic violations within a passive listening situation (i.e., outside of an active task) may not trigger the same inhibitory mechanism either.

Furthermore, the composition of our sample limits interpretations to healthy, English-speaking college undergraduates at a large state-funded institution of higher education in the United States. These mechanisms may well be altered during healthy aging (or over the lifespan more generally) and may not represent those in populations with atypical language development.

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