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Effects of phonological and semantic deficits on facilitative and inhibitory consequences of item repetition in spoken word comprehension

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Abstract

Repeating a word can have both facilitative and inhibitory effects on subsequent processing. The present study investigated these dynamics by examining the facilitative and inhibitory consequences of different kinds of item repetition in two individuals with aphasia and a group of neurologically intact control participants. The two individuals with aphasia were matched on overall aphasia severity, but had deficits at different levels of processing: one with a phonological deficit and spared semantic processing, the other with a semantic deficit and spared phonological processing. Participants completed a spoken word-to-picture matching task in which they had to pick which of four object images matched the spoken word. The trials were grouped into pairs such that exactly two objects from the first trial in a pair were present on screen during the second trial in the pair. When the second trial's target was the same as the first trial's target, compared to control participants, both participants with aphasia exhibited equally larger repetition priming effects. When the second trial's target was one of the new items, the participant with a phonological deficit exhibited a significantly more negative effect (i.e., second trial response slower than first trial response) than the control participants and the participant with a semantic deficit. Simulations of a computational model confirmed that this pattern of results could arise from (1) normal residual activation being functionally more significant when overall lexical processing is slower and (2) residual phonological activation of the previous trial's target having a particularly strong inhibitory effect specifically when phonological processing is impaired because the task was phonologically-driven (the spoken input specified the target). These results provide new insights into perseveration errors and lexical access deficits in aphasia.

Keywords

repetition; perseveration; access deficit; aphasia; spoken word comprehension

1. Introduction

Understanding the facilitative and inhibitory dynamics among partially active representations is a major theme across studies of memory, language, and cognitive control. In each of these domains, partially active representations have been shown to facilitate

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performance in some contexts and inhibit or compete in other contexts. For example, in the domain of lexical processing, *lexical neighbors* - words that are similar in spelling, sound, or meaning, and are thus partially activated during processing - have been shown to exert both inhibitory and facilitative effects on target word processing (for a comprehensive review see Chen & Mirman, 2012). Chen and Mirman used computational model simulations to demonstrate that the complex pattern of facilitative and inhibitory effects could be captured by a simple computational principle: strongly active neighbors exert a net inhibitory effect and weakly active neighbors exert a net facilitative effect.

Item repetition also has both facilitative and inhibitory consequences. Perhaps the most robust example of facilitation is *repetition priming*: processing is faster and more accurate on the second presentation of an item than on the first (e.g., Cave & Squire, 1992; Goldinger, 1998; Ratcliff & McKoon, 1988; Scarborough, Cortese, & Scarborough, 1977; Van Petten et al, 1991). The flip side of repetition priming is *perseveration errors*: unintentional and erroneous repetition of a previously produced response (e.g., Martin & Dell, 2007; Fischer-Baum & Rapp, 2012). Perseveration errors in aphasia have been studied for over 100 years (Stark, 2007) with a central debate between two broad types of mechanisms: perseverations arise because the new input is not sufficiently activated (“failure to activate”) or because the previous target is not sufficiently inhibited (“failure to inhibit”).

Negative serial position effects are another example of inhibitory effect of item repetition: performance progressively deteriorates across repetitions of an item. Such effects have become a hallmark of “refractory/access” deficits in aphasia (Warrington & McCarthy, 1983, 1987; McNeil, Cipolotti, & Warrington, 1994; Forde & Humphreys, 1995; Warrington & Cipolotti, 1996; McCarthy & Kartsounis, 2000; Crutch & Warrington, 2008).

In the present study we aimed to shed new light on these dynamics by examining the facilitative and inhibitory consequences of different kinds of item repetition in two individuals with aphasia. Critically, the two individuals had deficits at different levels of processing: one with a phonological deficit and spared semantic processing, the other with a semantic deficit and spared phonological processing. We chose a simple spoken word-to-picture matching task because this task has minimal working memory and cognitive control demands and we manipulated whether the repeated item was the target or a distractor in order to assess both the facilitative and inhibitory effects of item repetition. The results indicated that inhibitory effects emerge specifically when a level-specific deficit weakens processing of critical input. This account was implemented in a simple computational model and tested with concrete simulations that provide an existence proof that the proposed principles are sufficient to account for the observed data. We conclude with discussion of how these results inform theories of typical and impaired word comprehension.

2. Experiment

2.1 Methods

2.1.1 Participants—Sixteen (10 female, 6 male) neurologically intact adults from the greater Philadelphia area completed the study. Their ages ranged from 35 to 78, with a mean age of 59. All participants were native English speakers who reported having normal or corrected-to-normal vision and hearing. They had no history of neurological events or conditions, and all scored 27 or above on the Mini Mental State Examination (Folstein, Folstein, & McHugh, 1975).

Two individuals with aphasia (MR1626 and MR2374) were selected from the Moss Neurocognitive Rehabilitation Research Registry (Schwartz et al, 2005) based on their

differing patterns of performance on background psycholinguistic testing (Table 1). Background test scores were obtained through the Moss Psycholinguistics Project Database (www.mappd.org; Mirman et al, 2010). These two participants were approximately matched on overall aphasia severity (Western Aphasia Battery [Kertesz, 1982] Aphasia Quotient), lexical processing (Philadelphia Picture Naming Test [Roach et al, 1996] and PALPA auditory lexical decision [Kay, Lesser, & Coltheart, 1992]), and lesion size. In addition, because the left inferior frontal gyrus (LIFG) has been hypothesized to be involved in resolving the competition produced by item repetition (e.g., Schnur et al, 2009), the participants were matched with respect to the lesion status of LIFG: for both participants the left inferior and middle frontal gyri were substantially lesioned (see Figure 1; lesion location was defined by an experienced neurologist). Both participants with aphasia also performed very well on word-to-picture matching using familiar words/concepts (Picture Name Verification Test), indicating that they would be unlikely to exhibit substantive differences in accuracy in our study.

MR1626 was a 74-year-old right-handed male with 11 years of education. In 2007 he suffered a left middle cerebral artery ischemic stroke involving primarily the left frontal lobe, including the inferior frontal gyrus and the motor strip, and extending along the interior of the Sylvian fissure. A phonological deficit was apparent in the preponderance of formal and nonword errors in picture naming, impaired word repetition and severely impaired nonword repetition, and modest impairment of speech perception (syllable discrimination). He performed normally on a test of non-verbal spatial short-term memory (a computerized version of the Corsi blocks task), suggesting that his poor performance on tests of verbal short-term memory also reflected a phonological deficit¹. High performance on tests of semantic association and low rates of semantic naming errors revealed largely intact semantic knowledge.

MR2374 was a 54-year-old right-handed male with a college education. He suffered a left middle cerebral artery ischemic stroke in 2010 resulting in a large inferior frontal gyrus lesion with smaller extension into the middle frontal gyrus. His overall pattern of performance on background tests suggested a semantic impairment with marked deficits on verbal and nonverbal tests of semantic association, primarily semantic errors in picture naming, and at-chance performance on a two-item semantic category discrimination task². In contrast, spatial and phonological short-term memory were relatively spared, as were speech perception and word and nonword repetition.

All participants were tested during the summer or fall of 2011 and were paid \$15/hour for their participation.

2.1.2 Stimuli—Picture stimuli consisted of 180 items from Rossion and Pourtois (2004) or additional pictures of a similar style. The 180 items were divided into 30 sets of six items. Because semantic blocking and semantic relatedness have played an important role in studies of inhibitory item repetition effects (e.g., Belke, Meyer, & Damian, 2005; Damian, Vigliocco, & Levelt, 2001; Howard et al, 2006; Hsiao et al, 2009; Oppenheim, Dell, &

¹This participant appeared to have both phonological *input* and *output* deficits, either because the impairment affected a shared phonological processing level, or happened to affect both independently. In either case, for this participant, the distinction between phonological input and output deficits is not relevant.

²This task is meant to test semantic short term memory by using progressively longer lists of items followed by a probe item with the participant asked to indicate whether the probe item matches the semantic category of any of the items in the list (adapted from Freedman & Martin, 2001; for descriptions of all battery tests see Mirman et al, 2010). MR2374's failure to progress beyond list length 1, which is just AX semantic category discrimination, combined with his relatively normal performance on phonological and spatial short-term memory task (also probe tasks with an analogous structure), suggests that this was due to a severe semantic deficit and not a general short term memory deficit.

Schwartz, 2010; Schnur et al, 2006, 2009), semantic relatedness was also manipulated: half of the item sets were semantically related, and half were unrelated. The semantic categories included for the related sets were fruit, vegetables, large animals, small animals, sea animals, sports equipment, tools, bugs, vehicles, clothing, musical instruments, body parts, birds, furniture, and kitchen appliances. All items within a set were phonologically unrelated. The full list of stimuli is in Supplementary Materials Table S1.

Target words were recorded by a female native speaker of Standard American English in the context of a carrier phrase (“Find the...”). The target word was then taken out of the carrier phrase to be presented as a single word. The average duration of the target words was 700 msec. Neither related and unrelated items nor item conditions differed significantly in duration. The intensity of all final sounds files was normalized. There was no overall difference in length or word frequency between items in related sets and unrelated sets.

2.1.3 Design—On each trial, participants had to pick which of four object images matched a spoken word. The trials were grouped into pairs such that exactly two objects from the first trial in a pair were present on screen during the second trial in the pair. There were three conditions reflecting the role of the Trial 2 target in Trial 1. In the “Repeat” condition, the second trial’s target was the same as the first trial’s target and the distractors were one non-target item repeated from the first trial and two new items. In the “Distractor” condition, the second trial’s target was one of the distractors from the first trial – an item that was present on screen during the first trial, but was not the target; the second trial distractors were the first trial’s target and two new items. In the “New” condition, the second trial’s target was one of the new items and the distractors were the target from the first trial, one non-target item from the first trial, and one other new item. Figure 2 shows an example of all trials from one item set. There were a total of 6 trials for each item set (3 conditions × 2 trials) and each item in a set appeared an equal number of times across the six trials. Five of the six items in each set were used as targets; one appeared as the target twice (in the Repeat condition), and one never appeared as the target. The trial pairs were arranged into three blocks such that each block contained an equal number of trials from each condition and an equal number of related and unrelated trials. Each item set appeared only once in each block and in all three conditions across blocks. Block order was randomized across participants. The individual items in each role across item sets were matched in length and word frequency.

2.1.4 Procedure—Ten practice trials with accuracy feedback were followed by the 180 experimental trials: 30 pairs of trials in each of the three conditions. Participants used an ergonomic mouse to facilitate clicking on the target picture. MR2374 used his right hand, MR1626 used his left hand due to limited use of his (previously dominant) right hand. To start each trial, participants clicked on a central fixation cross, after which four pictures appeared on the screen in the arrangement shown in Figure 2. Which picture appeared in which screen corner screen was randomly assigned on each trial. After a 1000 msec preview period the target word was played over speakers at a comfortable listening volume and the trial ended when the participant clicked on one of the four images. Stimuli were presented and responses were recorded using E-Prime Professional 2.0. Participants’ eye movements were tracked using an SR Research EyeLink 1000 Desktop Mount eyetracker, but fixation data were not included in the analysis due to poor calibration for both of the participants with aphasia.

2.2 Results

Overall word comprehension accuracy was near ceiling for all participants (Control: 99.5%; MR1626: 95.6%; MR2374: 96.6%) and logistic regression revealed no statistically

significant effects (the difference between the two participants with aphasia and control group was marginal: both $p = 0.08$).

Response times (RT) were measured from word onset and participant condition means were used for incorrect response trials. To examine the effect of item repetition, for each trial pair, the critical measure was the response time difference between the first and second trial. Reaction time means and standard errors by trial number (first or second in a pair), condition (i.e., second trial target type: New, Distractor, Repeat), relatedness of objects in the display (Related, Unrelated), and participant group (Control, MR1626, MR2374) are provided in Supplementary Materials Table S2.

For the primary analysis these RT differences were computed for each trial pair for each participant type (i.e., single observations for the two participants with aphasia and the mean of the control group) and analyzed using a by-item ANOVA (i.e., target item as random effects) with Group (Control, MR1626, MR2374) as a within-item factor and Condition and Relation as between-item factors. This approach had the advantage of relying on a relatively standard analysis method (ANOVA), although the variance in the Control group was much smaller than for the participants with aphasia, thus ANOVA's assumption of equality of variances was violated. However, alternative analyses based on Crawford-Howell *t*-tests for comparing individual cases to small control groups (1998; see also Crawford & Garthwaite, 2012) and multilevel regression with crossed random effects of subject and item (Baayen et al, 2008; Barr et al., 2013) produced the same qualitative patterns of results, so we report the findings in the more familiar ANOVA format.

There was a main effect of Condition ($F(2, 84) = 20.65, p < 0.0001$) reflecting that the RT differences were larger in the Repeat condition than the other conditions (i.e., a repetition priming effect). There was a significant effect of Relation ($F(1, 84) = 4.01, p = 0.048$) reflecting that priming effects were larger (more positive) for semantically related item sets than for unrelated item sets. There was a marginal main effect of Group ($F(2, 168) = 2.46, p = 0.088$) reflecting overall larger priming effects for the two participants with aphasia than the control group³. Finally, and most importantly, there was a significant Group-by-Condition interaction ($F(4, 168) = 4.79, p = 0.001$) indicating that the groups differed in priming effects across conditions. There were no other significant main effects or interactions (all other $p > 0.3$). Figure 3 shows the pattern of priming effects for each group in each condition (the data are averaged over Relation because it did not interact with either Condition or Group; the full RT data are in Supplementary Materials Table S2).

A series of post-hoc pairwise comparisons were conducted to uncover to nature of the Group-by-Condition interaction. In the Distractor condition, there were no priming differences between groups ($p > 0.4$). In the Repeat condition, both participants with aphasia exhibited larger repetition priming effects than the controls did (both $t > 2.9, p < 0.01$) and did not differ from each other ($t < 1, p > 0.6$). In the New condition, participant MR1626 (who had the phonological deficit) showed an inhibitory effect that was significantly more negative compared to the controls ($t = 2.44, p < 0.05$) and the other participant with aphasia, MR2374 ($t = 2.12, p < 0.05$), but MR2374 did not differ from controls ($t < 1, p > 0.6$). In sum, both participants with aphasia exhibited increased repetition priming effects relative to controls and the participant with the phonological deficit uniquely exhibited an inhibitory effect when the second trial target was a new item.

³The same pattern of results was obtained using reaction time ratios (Trial 1/Trial 2), so this is not simply due to slower responses by participants with aphasia producing larger priming effects. RT difference distributions were closer to normal than RT ratio distributions were, so we report the analyses of RT differences since these involve less violation of the ANOVA assumptions.

2.3 Discussion

There were two key findings in the behavioral study: (1) Both participants with aphasia exhibited equally increased repetition priming effects relative to a group of matched neurologically-intact controls. (2) The participant with a phonological deficit exhibited an inhibitory effect for new items, but neither the control group nor the participant with a semantic deficit exhibited this effect.

These results indicate that the facilitative effect of item repetition is enhanced when either of the relevant processing levels is impaired, but only a phonological deficit brings out the inhibitory effect of item repetition. The first effect could emerge if either deficit slows word comprehension, thus making the normal amount of facilitation functionally more consequential. In other words, the same amount of facilitation has a functionally greater impact on impaired word comprehension than unimpaired word comprehension. The second effect could be due to the fact that spoken word-to-picture matching is a phonologically-driven task because the phonological input (spoken word) uniquely specifies the target but the semantic input (displayed pictures) is equally strong for each of the four alternatives. As a result, residual phonological activation of the previous trial's target would have a stronger inhibitory effect when phonological processing of the new input is impaired. This account is in the same vein as “failure to activate” accounts of perseveration (e.g., Cohen & Dehaene, 1998; Dell, Burger, & Svec, 1997; Martin & Dell, 2007; and for a historical perspective see Stark, 2007), which will be discussed in greater detail in the General Discussion, but first it is important to show that this proposal can, in fact, account for the behavioral data. To that end, the core processing principles were instantiated in a simple computational model of word-to-picture matching and simulations were conducted under control, phonological deficit, and semantic deficit conditions.

3. Simulations

The purpose of these simulations was to evaluate whether the proposed account is sufficient to explain the observed data. To that end, we abstracted away from the complexities of speech perception, spoken word recognition, and semantic cognition, and modeled the spoken word-to-picture matching task in its most basic form. That is, the model was meant to implement the following principles and constraints in the simplest possible form:

1. Two simultaneous sources of external input: phonological input that specifies the target word and semantic input that specifies the four possible response alternatives.
2. A lexical selection layer integrates these two sources of input through a multiple constraint satisfaction process and a response is generated when any unit in this layer exceeds the response threshold.
3. Unit activations decay over time when there is no external input or if the input is too weak.
4. The task is self-paced: the next trial is initiated when all unit activations have decayed below a trial-initiation threshold.
5. Phonological and semantic deficits correspond to reductions in the strength of phonological or semantic input⁴, respectively. For comparison, an alternative

⁴For the purposes of these simulations we use “reduced input” to mean reduced input from the semantic or phonological layer to the lexical selection layer, which can be implemented either as reduction of the strength of external input to the semantic or phonological layers or as reduction of connection weight strengths between those layers and the lexical selection layer. The input to the lexical layer is the product of semantic or phonological unit activations and their weights, so reducing either one has the same effect on lexical processing.

account was implemented by reducing the level-specific decay rates and the strength of input was unchanged.

3.1 Model Implementation

The model architecture is shown in Figure 4 and is similar in spirit to the recurrent normalization model of visual search (Spivey, 2007; see also Lupyan & Spivey, 2008; Reali et al, 2006; and for a related model of word production see Howard et al, 2006). All units were modeled as leaky-integrator neurons (e.g., Thelen et al, 2001; Usher & McClelland, 2001) with activation driven by net input and decay according to:

$$\frac{da_i}{dt} = -\lambda a_i + N_i \quad (1)$$

where a_i is the activation of unit i , N_i is the net input to unit i , and the decay rate (1.0) reflects leakage of the activation. There were three processing layers, each consisting of six units corresponding to the six concepts in a set of items from an experimental trial pair. The “Semantic” and “Phonological” layers corresponded to the different input sources. On each trial, external input was provided to a single Phonological layer unit corresponding to the target word and to the four Semantic layer units corresponding to the four objects on the screen. This activation then spread to the “Lexical” or selection layer, where the input from the Semantic and Phonological layers was integrated and units competed through lateral inhibition (for a more detailed implementation of the spoken word-to-picture matching task see Chen & Mirman, under review). The lexical units followed the same leaky-integrator activation function, which can be written as follows, with the net input portion expanded to show separately the contribution of semantic, phonological, self-recurrent, and lateral interactions:

$$\frac{da_i}{dt} = -\lambda a_i + \sum_{j=1}^N w_{sj} s_j + \sum_{j=1}^N w_{pj} p_j + \alpha a_i - \beta \sum_{j \neq i}^N a_j \quad (2)$$

where w_s and w_p are connection weights from unit j in the semantic and phonological layer, respectively, to unit i in the lexical layer and s_j and p_j are the activations of unit j in the semantic and phonological layers, respectively (w_s and w_p were both set to 0.5); α is the recurrent self-activation connection weight (0.1) and β is the lateral inhibitory connection weight (0.1) from other units in the lexical layer.

A simulated trial pair began with all unit activations set to 0 followed by presentation of external input to the target word unit in the phonological layer and the four displayed object units in the semantic layer. Activation was then allowed to propagate to the lexical layer until a lexical unit reached the decision threshold (0.5). Once the decision threshold was reached, the Trial 1 “response time” was recorded (i.e., the number of processing cycles required to reach the decision threshold), the external input was removed, and the unit activations were allowed to decay. In the behavioral task, participants clicked on the screen to initiate each trial. To model this self-paced aspect of the task, unit activations were required to decay until all activations were below a “trial-initiation” threshold (0.2) indicating that the model was ready for the next trial. At this point, the next trial would begin by presenting external input to the appropriate units (without resetting the unit activations). In the semantic layer, the external input was presented to the first trial's target, a non-target item from the first trial, and two new items (as in the behavioral experiment). For a “Repeat” condition trial, the same phonological target unit received external input; for a “New” condition trial, the phonological unit corresponding to one of the new items received

external input. For brevity, because there were no group differences in the “Distractor” condition, this condition was not simulated. Similarly, because there were no group differences across semantically related and unrelated trials, we only report simulations of unrelated trials (consistent with the behavioral data, simulation of related trials produced the same pattern of results).

For Control simulations, all external inputs were set to 2.0. To model a phonological deficit (e.g., MR1626) the external input to the phonological layer was reduced to 0.5. To model a semantic deficit (e.g., MR2372) the external inputs to the semantic layer were reduced to 0.5. This implementation is a concrete form of the hypothesis that deficits correspond to difficulty activating the new input. For comparison, we considered an alternative hypothesis that deficits correspond to increased persistent activation of previously active representations. This hypothesis was implemented by reducing the decay rate for the affected level from 1.0 to 0.5 without changing the input strength. That is, a phonological deficit was implemented by reducing the phonological layer decay rate, thus producing more persistent phonological activation and a semantic deficit was implemented by reducing the semantic layer decay rate, thus producing more persistent semantic activation⁵. These two implementations correspond to “failure to activate” and “failure to inhibit” accounts of perseveration errors (for discussion see, e.g., Fischer-Baum & Rapp, 2012; Stark, 2007).

3.2 Results and Discussion

When the level-specific deficits were implemented as reductions in input strength, the simulation produced the key aspects of the observed behavioral pattern: both the phonological and semantic deficits increased repetition priming in the Repeat condition and only the phonological deficit increased the negative perseveration effect in the New condition (Figure 5, left panel). The phonological and semantic deficits each slowed down processing because there was less external input. When residual activation from the first trial corresponded to the second trial's target (i.e., the Repeat condition), this boost was relatively more facilitative for the deficit simulations because the overall processing was slower.

When the residual activation was mismatching, strong phonological input could quickly overcome this residual activation even if the semantic input was weak, but weak phonological input required more time to overcome the residual activation, thus producing the inhibitory effect. This asymmetry between phonological and semantic deficits arose because the external phonological input was the critical input that uniquely specified the target (i.e., it was a phonologically-driven task). Consider the network dynamics on the second trial in a New condition trial pair: there is residual activation in the trial 1 target, there is phonological input to the new target, and there is semantic input to all four displayed candidates, including both the new target and the trial 1 target. Thus, when the phonological input is reduced (phonological deficit) the network takes longer for this weaker input to overcome the residual activation; when the semantic input is decreased (semantic deficit), there is less input to both the current trial's target and the residually-active distractor that was the previous trial's target, so this decrease is as much beneficial (less input to the residually active distractor) as it is detrimental (less input to the new target).

In contrast, when level-specific deficits were implemented as reduced decay (increased persistent activation), the simulation (Figure 5, right panel) produced the observed increased repetition priming for both the phonological and semantic deficits, but for the New condition the simulation results were opposite to the observed behavioral data: the semantic deficit

⁵Also, the start of trial 2 was fixed at cycle number 420, which was the average start point for trial 2 in the reduced-input simulations. This change was necessary because allowing activations to decay to a trial-initiation threshold would essentially eliminate the persistent activation: slower decay would simply become longer inter-trial-intervals.

caused slower response to the new target and there was no effect of phonological deficit. In the Repeat condition, slower decay meant greater residual activation of the target (in either the phonological or semantic layer), so when it was repeated, the recognition was faster. In the New condition, reducing the phonological decay rate (phonological deficit) produced greater residual activation in the phonological layer, but that was quickly overcome by the strong phonological input to the new target, so there was no additional delay in target recognition. However, because semantic input was applied to all candidate objects, the trial 2 semantic input was applied to both the new target and the residually active trial 1 target, which was a distractor on trial 2 (and the other two distractors). This reactivation of the trial 1 target exacerbated the increased residual activation, making it more difficult for the new target to overcome this distractor and producing the inhibitory effect. Indeed, any further decrease in the semantic decay rate caused the model to make overt perseveration errors (i.e., the trial 1 target passed the decision threshold before the trial 2 target did).

4. General Discussion

4.1 Summary of Findings

A behavioral experiment examined the consequences of deficits at particular levels of processing on the facilitative and inhibitory dynamics of item repetition in a simple spoken word-to-picture matching paradigm. On each trial, participants had to select the named object from four pictures on a computer screen. In each critical trial pair, two pictures from the first trial – the target and one distractor – were re-presented on the screen during the second trial. The key manipulation was whether the target on the second trial in a pair was the same as the target on the first trial or one of the new pictures. In addition to a control group of older neurologically-intact adults, two participants with aphasia completed the study. The two participants with aphasia were approximately matched on overall severity, but differed in the nature of their deficit: one had a phonological deficit and spared semantic processing; the other had a semantic deficit and spared phonological processing. Both participants with aphasia exhibited equally increased repetition priming effects relative to the control group and only the participant with a phonological deficit exhibited an inhibitory effect (i.e., second trial RT slower than first trial RT) when the target was new.

This pattern was taken to indicate that facilitative repetition priming effects arise due to normal residual activation that are more functionally significant for individuals with slowed overall lexical processing (e.g., Plaut & Booth, 2000) and that deficits correspond to weakened (slowed) processing within the impaired level of processing. This account was implemented in a simple computational model and simulations of the model provided evidence that it is sufficient to account for the observed behavioral data. An alternative implementation based on the hypothesis that deficits correspond to abnormally large residual activation in the impaired level failed to account for the observed behavioral data. However, since this was a study of two individual cases, we cannot rule out the possibility that other individuals with aphasia will exhibit a pattern consistent with a failure to inhibit deficit (for evidence that participants with aphasia can exhibit both patterns see, e.g., Fischer-Baum & Rapp, 2012).

These results suggest a dynamic interplay of facilitative and inhibitory influences such that concordant partial activation can facilitate processing, but if it is too strong relative to new input, the net result becomes an inhibitory effect. Related phenomena have been studied extensively in the domain of lexical neighborhood effects where strong competitors exert net inhibitory effects and weak competitors exert net facilitative effects (Chen & Mirman, 2012, under review; see also Mirman, 2011; Mirman & Magnuson, 2008).

The present data leave open (at least) two further questions about the facilitative and inhibitory dynamics involved in item repetition. First, on the second trial in a pair, is the activation of the previous trial's target due to residual activation (as we have described) or incremental learning (e.g., Oppenheim et al, 2010)? Both can account for the present data, though they may make different predictions about the dynamics of the competition process. Second, the IFG has been implicated in resolving competition among partially active representations (e.g., Schnur et al, 2009), but both participants with aphasia in this study had IFG lesions and yet exhibited different behavioral patterns. We have explained this in terms of differences in the level of processing that was impaired. If this is a sufficient account, then what is the role of IFG? In other words, are level-specific deficits sufficient or is there an additional contribution of domain-general cognitive control processes and deficits? A combination of behavioral experiments and computational model simulations, as in the present study, is a promising approach for answering these questions.

4.2 Relation to Perseveration Errors

One of the key findings in the present study was that a phonological deficit slowed word recognition in the presence of an item that was the target on the previous trial. This pattern can be considered a response time version of perseveration – although the previous target did not elicit an incorrect perseverative response, it was a stronger competitor. As mentioned in the Introduction, perseveration errors could arise because the new input was not sufficiently activated (“failure to activate”) or because the previous target was not sufficiently inhibited (“failure to inhibit”).

A key difference between the accounts is that “failure to inhibit” has typically been framed as a relatively general deficit of cognitive control (e.g., Gotts & Plaut, 2002; Schnur et al, 2009; Warrington & McCarthy, 1983; see also Jefferies & Lambon Ralph, 2006). In contrast, “failure to activate” has typically been connected to other deficits in aphasia, often at particular levels of processing (e.g., Cohen & Dehaene, 1998; Dell et al, 1997; and Stark, 2007, shows that this was recognized early on as well). Dell and colleagues (Martin & Dell, 2007; Dell et al, 1997) have argued based on computational modeling and behavioral data that failure to activate the new target correctly accounts for both perseverative and non-perseverative errors in word production, so proposing an additional deficit of inhibition is unnecessary and unparsimonious (though see Fischer-Baum & Rapp, 2012, for evidence that both deficits are necessary to account for the full range of perseveration deficit patterns). Cohen and Dehaene similarly argued that perseverations arise from normal residual activation combined with deficits in a particular level of processing. On their view, the deficit undermines the ability of new inputs to overcome normal residual activation specifically at the affected level of processing. They provided key evidence for this account by examining the nature of picture naming perseveration errors produced by three patients with deficits at different levels of processing: lexical, phonological, and visual.

The present behavioral and computational results are consistent with the level-specific failure to activate hypothesis and extend it in several ways. First, we examined both the facilitative and detrimental effects of item repetition – repetition priming and perseveration – and showed that this proposal correctly accounts for contrasting patterns in the two contexts. Second, we tested word comprehension rather than word production and participants who did not make overt perseveration errors. This is important because the alternative accounts of perseveration make claims about the underlying dynamics of processing (deficits of activation vs. inhibition), but previous studies have almost exclusively focused on perseveration errors in word production, which are only the most extreme outcome. Finally, we used simulations of a computational model to go beyond a verbal description of the data. The simulations concretely demonstrated that a level-specific reduction in activation was sufficient to account for both of the critical patterns in the

behavioral data and that a level-specific reduction in decay (i.e., failure to inhibit) could not account for the behavioral data.

The computational model also draws attention to an oft-neglected aspect of perseveration effects: the detailed demands and dynamics of the task. One such aspect is that spoken word comprehension is a phonologically-driven task, so a semantic failure to activate deficit does not induce perseverations when there is effective processing of phonological input. For a semantically-driven task, the complementary pattern of results is predicted: slower performance on new items in the presence of repeated items for MR2374, but not for MR1626⁶.

Another key aspect is that the task was self-paced: participants initiated trials when they felt ready and trials were terminated when the participant produced a response. Further, response accuracy was near ceiling (and only correct response RTs were included in the analysis). That is, at the end of each trial, each participant had successfully activated the correct target item. This means that, although lexical activation was slowed for the participants with aphasia relative to the controls, it reached a functionally equivalent level because they produced the correct response with (nearly) equal probability. As a result, under the proposed account and as implemented in the computational failure to activate model, there was equivalent residual activation at the start of the subsequent trial for each participant group.

4.3 Relation to Refractory/Access Deficits

Like accounts of perseveration, accounts of “refractory/access” deficits in aphasia have wrestled with whether the deficits are due to impaired activation processes or impaired inhibition processes (e.g., Campanella & Shallice, 2011). In this case, the debate has been between accounts that propose excessive residual activation (i.e., failure to inhibit) leading to increased competition with new inputs (Campanella & Shallice, 2011; Howard et al, 2006; Oppenheim et al, 2010; Schnur et al., 2006, 2009), and those that propose excessive inhibition leading to difficulty re-activating a repeated target (e.g., Forde & Humphreys, 1995; Gotts & Plaut, 2002; McCarthy & Kartsounis, 2000; Warrington & McCarthy, 1983). In part, this debate continues because much of the key data comes from tasks in which a set of items is repeated multiple times (e.g., blocked cyclic naming); thus, errors could arise either from over-activation of (repeated) competitors or over-inhibition of the (repeated) target.

As we have argued, the results of our study are consistent with a variant of the over-activation view (Cohen & Dehaene, 1998): impaired input processing makes normal residual activation more difficult for the new input to overcome. More importantly, the present experimental paradigm may provide an avenue for resolving this debate. By limiting repetition to pairs of trials and specifically manipulating the repetition status of the second trial's target and distractors, we were able to disentangle facilitative and inhibitory effects of residual activation and to show how they are differentially affected by level-specific deficits. This general experimental paradigm may help to disentangle the predictions of excessive activation and excessive inhibition accounts of refractory/access deficits.

Comparing individuals with deficits at different levels of processing, as we have done, may also help to distinguish accounts of refractory/access deficits. Some studies have suggested that such deficits can be limited to particular modalities or semantic categories (e.g., Crutch & Warrington, 2001; McCarthy & Kartsounis, 2000; McNeil et al, 1994), but accounts tend

⁶We were unable to test this prediction because MR2374 was no longer available for testing.

to be framed in terms of general processing dynamics (excessive activation and inhibition) and with a particular focus on semantic processing despite evidence that such effects occur in other domains (e.g., Mulatti et al., 2012). Studies of perseveration errors have made important discoveries by examining how individual participants' perseveration errors relate to their broader deficit pattern (e.g., Cohen & Dehaene, 1998; Fischer-Baum & Rapp, 2012; Martin & Dell, 2007). Similarly, studies of refractory/access deficits may be able to make new progress by examining the relationship between specific deficits and refractory/access phenomena.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights for “Effects of phonological and semantic deficits on facilitative and inhibitory consequences of item repetition in spoken word comprehension”

- Tested effects of repetition on spoken word recognition in two people with aphasia
- Compared to controls, both participants exhibited increased repetition priming
- Only the phonological deficit participant showed an inhibitory effect for new words
- Tested reduced-activation and reduced-inhibition accounts using computational model
- Simulations showed that level-specific reduced activation can account for the data

Figure 1.
Three illustrative coronal slices from CT scans for participant MR1626 (top row; $z = 0, 9, 18$) and MR2374 (bottom row; $z = -3, 6, 15$).

Figure 2.
Example of all trials from all conditions for a semantically related item set.

Figure 3. Reaction time differences between first and second trial for each Group in each Condition. Error bars indicate $\pm$ SE, * indicates $p < 0.05$, ** indicates $p < 0.01$ for pairwise comparisons.

Figure 4. Schematic diagram of model. Filled units in the Phonological and Semantic input layers indicate the units that are receiving external activation. In the Lexical layer, the unit filled with black is the target, which receives input from both the Phonological and Semantic layer, the units filled with gray are the distractors, which receive input only from the Semantic layer.

Figure 5.

Simulations results for two different implementations of level-specific deficits: reduced input (left) and reduced decay (right). RT was measured as the number of processing cycles required to reach the decision threshold.

Table 1

Background test performance for the two participants with aphasia.

	MR1626	MR2374	Control Norms*
Aphasia Subtype	Broca's	Transcortical	-
		Motor	
WAB Aphasia Quotient	67.8	75.5	-
Auditory Lexical Decision (PALPA, <i>d'</i>)	2.28	2.55	-
Words (% Correct)	93	84	-
Nonwords (% Correct)	79	94/	-
Picture Naming (PNT, % Correct)	51	59	97.2 (2.7)
% Semantic Errors	2.3	9.7	-
% Mixed Errors	1.1	8.6	-
% Nonword Errors	22.3	4	-
% Formal Errors	12.0	4	-
Primary Perseverations	0.6	1.1	-
Word Repetition (PRT, % Correct)	68	96	-
Nonword Repetition (% Correct)	8	70	82.6 (10.5)
Phoneme Discrimination			
No Delay (% Correct)	80	90	97.45 (3.0)
Delay (% Correct)	78	93	95.55 (3.7)
Short-Term Memory			
Immediate Serial Recall (Words)	1.4	4.8	4.8 (0.3)
Span			
Semantic STM Span	1.67	0.5	5.39 (1.3)
Phonological STM Span	1.29	6.27	6.45 (1.6)
Spatial STM Span	5.0	5.3	5.31 (1.5)
Semantic Processing			
Pyramids and Palms (% Correct)	96	85	-
Camels and Cactus (% Correct)	81	31	89.8 (5.6)
Synonymy Triplets (% Correct)	90	67	97.4 (5.4)
Peabody Picture Vocabulary Test (Standard Score, $M = 100$, $SD = 15$)	90	46	100 (15)
Picture Name Verification Test (PNT, % Correct)	97	95	98.6 (0.9)
Lesion volume (cc)	77.3	88.8	-

* Note: Mean (SD) control norms were collected from various sources and are presented here for general information only; the neurologically intact control group from the present study did not complete this test battery.