Psychological Review
2010, Vol. 117, No. 1, 256–272

0033-295X/10/$12.00 DOI: 10.1037/a0015948

COMMENTS

Computational Modeling of Reading in Semantic Dementia: Comment on Woollams, Lambon Ralph, Plaut, and Patterson (2007)

Max Coltheart
Macquarie University

Jeremy J. Tree
Swansea University

Steven J. Saunders
Macquarie University

Woollams, Lambon Ralph, Plaut, and Patterson (2007) reported detailed data on reading in 51 cases of semantic dementia. They simulated some aspects of these data using a connectionist parallel distributed processing (PDP) triangle model of reading. We argue here that a different model of reading, the dual route cascaded (DRC) model of Coltheart, Rastle, Perry, Langdon, and Ziegler (2001), not only provides a more accurate simulation of these aspects of reading in semantic dementia than does the PDP model but also provides highly accurate simulations of other aspects of reading in this disorder that the PDP approach has not simulated. We conclude that our findings add to evidence both from simulations of normal skilled reading and from simulations of other kinds of acquired dyslexia that the nonconnectionist DRC model of reading offers a better account of normal and disordered reading than the connectionist PDP models of reading.

Keywords: reading, semantic dementia, dual route model, computational modeling

Semantic dementia (Hodges, Patterson, Oxbury, & Funnell, 1992; Snowden, Goulding, & Neary, 1989) is a progressive brain pathology particularly involving focal degeneration of the temporal lobes, especially the left temporal lobe. Its initial cognitive symptom is an impairment in the comprehension of written words, spoken words, and pictures. At this initial stage, reading aloud can be fully preserved even though reading comprehension is impaired. As the disease progresses, specific impairments in reading aloud begin to emerge. The disorder has recently excited the interest of reading theorists, in particular those concerned with building computational models of reading, because the patterns of preserved and impaired reading abilities seen in these patients and how these change as the disease progresses over time provide challenging sets of data for theories to explain and models to simulate.

By far the most extensive study to date of reading in semantic dementia has been that of Woollams, Lambon Ralph, Plaut, and Patterson (2007), who reported detailed individual data on reading and other tasks from 51 patients with semantic dementia. Some of these patients were assessed on more than one occasion (so as to follow the course of the disease), and hence, some patients yielded more than one data set; the total number of data sets reported in Woollams et al. was 100.[1] For all 100 data sets, there are measures of the accuracy of reading aloud for high- and low-frequency regular and exception words. For 34 of the data sets, there is also a measure of accuracy of reading aloud for nonwords.

Woollams et al. (2007) used one of the parallel distributed processing (PDP) triangle models of reading (Simulation 4 of Plaut, McClelland, Seidenberg, & Patterson, 1996) to simulate some aspects of the reading data collected from these patients. Our aim in this article is to evaluate this triangle model's account of the data provided by Woollams et al. and to compare the triangle model and the dual route cascaded (DRC) model (Coltheart, Rastle, Perry, Langdon, & Ziegler, 2001) with respect to how well each model can account for the reading symptoms seen in people with semantic dementia.

The Effect of Semantic Dementia on Reading Comprehension and Reading Aloud

So as to characterize what it is that computational models of reading[2] have to simulate as far as reading aloud in semantic

Max Coltheart and Steven J. Saunders, Macquarie Centre for Cognitive Science, Macquarie University, Sydney, Australia; Jeremy J. Tree, Department of Psychology, Swansea University, Swansea, United Kingdom.

We thank Anne Castles for her comments on an earlier version of this article and Anna M. Woollams for much valuable discussion of the article by Woollams, Lambon Ralph, Plaut, and Patterson (2007).

Correspondence concerning this article should be addressed to Max Coltheart, Macquarie Centre for Cognitive Science, Macquarie University, Sydney NSW 2109, Australia. E-mail: max@maccs.mq.edu.au

[1] We are grateful to Anna M. Woollams for providing us with the reading data from all of these data sets.

[2] This is a shorthand for *computational models of reading aloud.* Of course, there is much more to reading than reading aloud—reading comprehension, for example—but we are concerned here only with reading aloud.

dementia is concerned, we begin with a description of the effects that this condition has on reading abilities.

The Three Phases of Semantic Dementia

Blazely, Coltheart, and Casey (2005) offered a description of the stages of reading deterioration in semantic dementia. Thanks to the study by Woollams et al. (2007), a rather more detailed picture of the course of this deterioration can now be proposed. We suggest that, in any patient with semantic dementia, a sequence of three phases may be discerned in the patient's reading as the disease progresses.

Phase 1: Impaired reading comprehension with intact reading aloud. Patients must have impaired reading comprehension if they are to meet the diagnostic criteria for semantic dementia. At the onset of the disorder, however, this is the only form of impaired reading they show: Their reading aloud of exception words (even those of low frequency), regular words, and nonwords is within normal limits.

Woollams et al. (2007) identified 5 of their 100 data sets in which the patient performed within the normal range on accuracy of the reading aloud of low-frequency exception words (they defined *normal* as within two standard deviations of the mean of a normal control group: The cutoff value here was 87% correct). These 5 data sets are shown in Table 1.

Here, we have five cases of semantic dementia in which reading aloud, at least for words (we do not know about nonword reading because it was not tested with these five data sets), was fully preserved despite the presence of semantic impairment. Three of these patients were classified by Woollams et al. (2007) in the mild category of impairment and the other two in the mild–moderate category. Numerous other patients with semantic impairments but normal accuracy in reading aloud even for low-frequency exception words have been reported. Some of these suffered from semantic dementia (Blazely et al., 2005; Cipolotti & Warrington, 1995; McKay, Castles, Davis, & Savage, 2007; Schwartz, Saffran, & Marin, 1980), and we take these cases as examples of patients who were at our Phase 1 of semantic dementia when tested. Others were patients with Alzheimer's disease (Lambon Ralph, Ellis, & Franklin, 1995; Noble, Glosser, & Grossman, 2000) or stroke (Gerhand, 2001); such patients are relevant to any model of reading according to which there is an intimate connection between access to semantics and exception word reading, even though these were not patients with semantic dementia.

Phase 2: Impaired reading comprehension with pure surface dyslexia. Somewhat later in the course of the disorder, the patient begins to show a frequency-sensitive impairment in reading aloud exception words (low-frequency exception words less accurate than high-frequency exception words; performance can be within normal limits for the latter). This is surface dyslexia, and in this phase, it is pure surface dyslexia in the specific sense that the reading aloud of nonwords is within normal limits even though there is an impairment in the reading aloud of exception words (at least those of low frequency).

Since, in 5 of the 100 data sets of Woollams et al. (2007), reading accuracy of low-frequency exception words was within the normal range, it follows that in 95 of these data sets reading of low-frequency exception words was impaired. How many of these 95 data sets represent cases of pure surface dyslexia—that is, how many had preserved nonword reading? Of the 100 data sets, there were 34 that included nonword reading data, so we can only ask this question in relation to these 34 of the 100 data sets. Control data for the Woollams et al. nonword reading test showed normal performance (performance within two standard deviations of the mean performance of normal control readers) to be 94% correct or better. Nonword reading accuracy was 94% or better in 10 of these 34 (29%). These 10 data sets are shown in Table 2. (In the patient identifiers, the number refers to the order of the testing session. Thus, e.g., G.C.1 and G.C.2 refer to the same patient: G.C.1 identifies data from the first testing session of the patient, and G.C.2 identifies data from the second testing session of the same patient.)

In 66 of the 100 data sets, nonword reading data were not collected. However, for 19 of these 66 cases (29%), reading accuracy was within normal limits on low-frequency regular words (i.e., 91% correct or more) but impaired for low-frequency exception words (less than 87% correct). These 19 cases are arguably also instances of pure surface dyslexia, though nonword reading data would be needed and would need to be in the normal range for this to be definitively claimed.

Phase 3: Impaired reading comprehension with generalized impairment of reading aloud. Still later in the disorder, reading accuracy for nonwords also begins to decline, so that accuracy for both exception words and nonwords (and often for regular words too) is below normal limits. This was the case in the remaining 24 of the 34 data sets from Woollams et al. (2007) that included data on nonword reading: All showed impairments both on word reading and on nonword reading. There were no cases where nonword reading was impaired but exception word reading was intact, even though there were numerous cases where exception word reading was impaired and nonword reading was not (see Table 2). Hence, in this sample of 34 data sets, impaired nonword reading did not begin to be apparent until after impaired exception word reading

Table 1
Percentage Correct Word Reading Accuracy in Five Cases of Semantic Dementia With Intact Reading Aloud of Words, Including Low-Frequency Exception Words

Patient	High-frequency regular words	Low-frequency regular words	High-frequency exception words	Low-frequency exception words	Nonwords
B.C.1	100	97.62	100	92.86	Not tested
G.C.1	100	100	100	92.86	Not tested
M.A.1	100	100	100	100	Not tested
E.B.1	100	100	100	100	Not tested
M.G.1	100	100	95.24	92.86	Not tested

Table 2
Percentage Correct Reading Accuracy for Nonwords and Low-Frequency Exception Words in 10 Cases of Semantic Dementia With Pure Surface Dyslexia

Patient	Low-frequency exception words (control cutoff 87%)	Nonword reading (control cutoff 94%)
G.C.2	76.19	95.0
A.M.3	69.05	95.0
J.H.4	26.19	95.0
S.L.1	66.67	97.5
A.M.1	64.29	97.5
B.M.2	57.14	97.5
G.C.6	47.62	97.5
P.S.M.1	78.57	100
B.M.1	69.05	100
A.M.4	50.00	100

had already been established. In other words, semantic dementia is not accompanied by pure phonological dyslexia (impaired nonword reading with intact exception word reading).

The task confronted by computational models of reading is thus to offer an explanation of the progression through these stages of reading impairment and of why these stages take the form they do, as well as also to attempt to make quantitative predictions of the reading data from individual patients with semantic dementia. The degree to which a model succeeds in these tasks is a measure of its worth as a description of the human reading system.

Computational Modeling of Reading

The Triangle Models Approach to Modeling Reading

The general form of the triangle models of reading aloud is shown in Figure 1 of Woollams et al. (2007). We use the plural in referring to these models because there have actually been seven different triangle models proposed, and these, while all adhering to the general structure depicted in this figure, do differ from each other in substantive ways.

For example, in one of the triangle models, the units in the orthographic system represent *wickelgraphs* (random three-letter sequences); in others, they represent single letters; and in still others, they represent orthographic onsets, vowels, and codas. Similarly, in one of the triangle models, the units in the phonological system represent *wickelphones* (random three-phoneme sequences); in others, they represent single phonemes; and in still others, they represent phonetic features. Thus, the general triangle model approach does not make any proposals about the specific ways in which orthography and phonology are actually represented in the human reading system.

A more critical fact about differences between the various triangle models has to do with how well the direct orthography-to-phonology (O→P) pathway of each triangle model can read words with atypical or exceptional correspondences between spelling and sound (exception words). Seidenberg and McClelland (1989) took pains to emphasize that this single route in their model could read exception words perfectly and yet did well on reading nonwords, which they considered as contradicting earlier claims that two distinct reading routes are needed to handle these two different reading tasks. These latter claims were also based on data from patients with pure surface dyslexia (e.g., the patients of Bub, Cancelliere, & Kertesz, 1985, and McCarthy & Warrington, 1986) in whom, after brain damage, reading of exception words, especially when these are of low frequency, is impaired, while nonword reading accuracy remains intact. A problem for the triangle models is that it is not obvious how one could selectively damage an O→P pathway that, when intact, was perfect at reading exception words and nonwords, in such a way as to harm exception word reading while sparing nonword reading and regular word reading. Indeed, attempts to lesion a triangle model in this way to simulate surface dyslexia (Patterson, 1990; Patterson, Seidenberg, & McClelland, 1989) did not succeed: A computational lesion of the O→P pathway severe enough to mimic the low level of performance with low-frequency exception words shown by the patient of McCarthy and Warrington (1986) also harmed the triangle model's ability to read nonwords aloud, and yet, this patient was normal at nonword reading.

Thus, Plaut et al. (1996) tried a different approach to triangle model simulation of surface dyslexia, by creating a version of the triangle model in which reading of exception words by the direct O→P pathway was not perfect: They referred to this model as Simulation 4 (S4), and it was trained in such a way that after completion of training, its direct O→P pathway still made errors, particularly when reading low-frequency exception words. As S4 was being trained, input from the semantic system (S) to the phonological system (P) was gradually introduced. Woollams et al. (2007) referred to this as "S"→P input because the S system is not implemented in Model S4, so the input had to be artificially introduced by the modeler. The strength of this input was programmed to increase monotonically over training epochs and to be greater for high-frequency than for low-frequency words (for details, see Plaut et al., 1996; Woollams et al., 2007). The upshot was that Model S4 could read virtually all regular words and nonwords just using the O→P pathway but relied on "S"→P input for correct reading of many exception words, especially those low in frequency; the provision of this input during training prevented the O→P pathway from learning to read all low-frequency exception words correctly (which it otherwise would have been able to do). Thus, if, after training, the model is lesioned by depriving it of the "S"→P input, it is worse at reading exception words than regular words: It is surface dyslexic.

So, the triangle model proposal here is that surface dyslexia is caused by semantic impairment, and indeed, these two symptoms do very frequently co-occur (as in the data of Woollams et al., 2007). However, this claim would predict that all patients with semantic impairment will be surface dyslexic, that is, will be abnormal at reading exception words (at least those of low frequency). This is not so; as we mentioned above, numerous patients with semantic impairments but normal accuracy in reading aloud even for low-frequency exception words have been reported (including five such patients reported by Woollams et al., 2007; see Table 1). How can such patients be reconciled with the idea that semantic impairment causes surface dyslexia?

Plaut et al. (1996) and Woollams et al. (2007) addressed this issue by supposing that, in intact readers, there are individual differences in the degree to which the reading of low-frequency exception words requires the support of input from the semantic system to phonology. Those individuals whose premorbid reading

of low-frequency exception words required such input will be surface dyslexic if they subsequently suffer a semantic impairment; those who premorbidly did not require any input from semantics to read any exception word will remain normal at exception word reading after suffering a semantic impairment. This somewhat complex proposal is in fact too simple, as Woollams et al. pointed out, because all the patients with semantic dementia they reported who had intact exception word reading at a stage of their disease when a semantic impairment was clearly demonstrable did subsequently become surface dyslexic as the disease progressed further. Hence, it was suggested that "as these individuals did not rely on S→P activation as extensively as most during reading aloud, exception word reading did not begin to suffer until a greater decline in semantic knowledge had occurred" (Woollams et al., 2007, p. 332). So, Woollams et al. varied two model parameters in their simulations: Individuals were assumed to vary premorbidly in the strength of the semantic contribution to phonology and postmorbidly in the degree of semantic impairment (which impacted both the strength and the variability of the semantic contribution according to a fixed equation).

Since no method was offered by which one might retrospectively assess the degree to which any patient would have been relying, premorbidly, on semantics to support exception word reading, Woollams et al. (2007, p. 332) commented, "We acknowledge that this hypothesis regarding individual differences in division of labor is difficult to test in the absence of premorbid estimates of semantic reliance during reading aloud".

Triangle Model S4 is one of two triangle models in which the direct O→P pathway is less than perfect at exception word reading, that is, in which there is a division of labor between the two routes with respect to exception word reading (the other is Harm & Seidenberg, 2004), and is the only one applied to the simulation of surface dyslexia. Model S4 is the version of the triangle model used in the simulations by Woollams et al. (2007), and so, that is the version of the triangle model that we consider in this article.

The method by which reading in semantic dementia was simulated by Woollams et al. (2007) was by training the S4 triangle model with steadily increasing "S"→P input and then, after completion of training, lesioning the trained model by making the putative contribution of semantics to phonology both (a) weaker and (b) more noisy: This noise was frequency dependent in the sense that the lower the frequency of the word presented to the model for reading, the noisier the computation of its phonology (the larger the standard deviation of the Gaussian distribution from which the noise was sampled).

The DRC Approach to Modeling Reading

The DRC model has been described in detail elsewhere (Coltheart et al., 2001), as has its ability to accurately simulate a variety of results obtained from studies of normal and disordered reading (Blazely et al., 2005; Castles, Bates, & Coltheart, 2006; Coltheart, 2005, 2006; Coltheart et al., 2001; Nickels, Biedermann, Coltheart, Saunders, & Tree, 2008; Rastle & Coltheart, 2006).

The DRC model has two routes from print to speech. One is a lexical nonsemantic route that receives input in parallel from all the letters in a visually presented letter string: This parallel input activates frequency-sensitive word entries in an orthographic lexicon, and these in turn activate their corresponding frequency-sensitive entries in a phonological lexicon, from which the phonemes of the phonological lexical entry are activated in the phoneme level. As this is happening, a letter-sound translation process is sweeping from left to right across the activated letter units, using a stored set of grapheme–phoneme correspondence (GPC) rules to translate the letters to phonemes and so to build up activation in phonemes (from left to right) at the phoneme level; this is how the nonlexical route of the model works.

Exception words (those that disobey the GPC rules) will be wrongly translated to phonology by the nonlexical route but correctly translated by the lexical route. Nonwords (which are not represented in the lexicons) will be correctly translated to phonology by the nonlexical route but will yield fragmentary[3] or no output to the phoneme level from the lexical route. Both routes will activate all the correct phonemes of a letter string when this letter string is a regular word (a word that obeys the GPC rules).

Simulating the Data Reported by Woollams et al. (2007)

We first consider just those aspects of the Woollams et al. (2007) data that those authors simulated using the triangle model approach, so as to directly compare the ability of the triangle and DRC models to simulate these results. After that, we consider aspects of the patient data that were not simulated by the triangle model and evaluate how well the DRC model can simulate these.

The DRC Account of Semantic Dementia

According to the DRC account of reading in semantic dementia, the reading system can be compromised in three ways. The disorder begins with just the semantic system compromised. Because, in the DRC model, reading aloud accuracy is perfect without the use of the semantic system, at this stage in the disorder, reading accuracy is intact (as it was in the patients with semantic dementia listed in Table 1). When impaired reading aloud first appears during the progression of the disease, this is due to an impairment of the lexical nonsemantic reading route; at that point, the nonlexical reading route is still intact. Here, the patients will have a frequency-sensitive impairment of reading of exception words, with nonword reading and regular word reading still intact (i.e., pure surface dyslexia). At a later stage of the disease, the nonlexical route also begins to be impaired, so that nonword and regular word reading will be abnormal as well as exception word reading. Thus, two lesions of the DRC model will be needed to fully capture the range of reading aloud impairments here. Noble et al. (2000) expressed views about the neuropathology of semantic dementia that are congruent with our ideas about the nature of the progressive reading deterioration seen in semantic dementia; Woollams et al. (2007, pp. 333–334) expressed doubts about just how consistent these ideas actually are with what is known about the neuropathology of the disease.

[3] Depending on the particular parameters of the model being used, a nonword's word neighbors may be activated in the orthographic lexicon and so in turn in the phonological lexicon, and this can result in some fragmentary activation at the phoneme level, though the set of phonemes thus (weakly) activated will never correspond to the correct pronunciation of the nonword.

These are not predictions from the DRC model. The model claims nothing about the neuroanatomical locations of its processing components, so cannot make such predictions. Instead, this is just a description in DRC terms of what we think happens in semantic dementia. We presume that the reason the disorder first affects the semantic system, then affects the lexical nonsemantic route for reading, and only later affects the nonlexical route for reading has to do with a combination of (a) which are the particular brain regions through which the disorder spreads over time, with more and more regions being affected over time, and (b) which of these regions are important for the semantic system, which are important for lexical nonsemantic reading, and which are important for nonlexical reading.

A region that appears important for the lexical nonsemantic reading route is the left fusiform gyrus, proposed by McCandliss, Cohen, and Dehaene (2003) as the site of the visual word form system. We might expect abnormality of this region to result in surface dyslexia. Bright, Moss, Stamatakis, and Tyler (2008) reported neuroimaging data from two of the patients studied by Woollams et al. (2007), BS and EK. Both patients were scanned three times over a 3-year period. For both patients, the first scan revealed no abnormality in the region of the left fusiform gyrus, whereas atrophy of this region was evident in the second and third scans for both patients. When these patients were tested by Woollams et al., both were surface dyslexic. Nestor, Fryer, and Hodges (2006) also reported hypometabolism of left fusiform gyrus in a group of people with semantic dementia.

Lesioning the DRC Model to Simulate Reading in Semantic Dementia

To capture the frequency sensitivity of the impairment of the lexical nonsemantic route, we lesioned that route in the DRC model[4] by deleting the *x*% least frequent words in the model's orthographic lexicon; the deleted words were then effectively nonwords. Any deleted word that was regular would still be read correctly because the intact nonlexical route can do this. Any deleted word that was irregular would be regularized. So, this lesion made the model surface dyslexic; the larger *x* was, the more severe the model's pure surface dyslexia.

The nonlexical route uses GPC rules to read aloud. The DRC model has 234 such rules. We lesioned this route by deleting the least frequently used *y*% of the GPC rules (the measure of GPC frequency was simply the number of words in which that particular GPC occurs). This made the model impaired at nonword reading; the larger *y* was, the more severe this impairment.

These deletions of orthographic lexical entries and GPC rules were probabilistic in the following way. Suppose the cutoff for being in the bottom *x*% of words with respect to frequency was a frequency of 150. Then, the probability of a word being deleted from the lexicon was a function of the difference between its frequency and the cutoff frequency, 150. This probability was normally distributed with a mean equal to the difference between the cutoff frequency and the word's frequency and a standard deviation of .20. Hence, the probability of deletion was .50 for a word right at the cutoff (a word with frequency 150), increased as the word's frequency decreased from this cutoff value, and decreased as the word's frequency increased above the cutoff value. The same probabilistic scheme was used in lesioning the GPC route.

We constructed a large set of lesioned DRC models by varying the lexical lesion severity *x* from 0% to 99.5% in 0.5% steps while varying the nonlexical lesion severity *y* from 0% to 100% in 0.5% steps. This generated 40,200 different versions of DRC, each corresponding to a different combination of lesioned lexical route and lesioned nonlexical route, that is, each corresponding to a different hypothetical patient with acquired dyslexia. Then, we submitted to each lesioned model the high- and low-frequency regular and irregular words and the nonwords[5] administered to their cases by Woollams et al. (2007), and the model's response to each item was scored as correct or incorrect. This yielded a word and nonword reading accuracy profile for each of the 40,200 lesioned models.

Thus, we varied two model parameters to simulate the reading impairments in these data sets; as mentioned earlier, Woollams et al. (2007) also varied two model parameters in their simulations.

If the DRC model is capable of simulating the full range of reading impairments seen in semantic dementia, then for every one of the 100 data sets we are considering, there must be a profile in this multidimensional space that corresponds to that data set's profile. We must emphasize that this is a far from vacuous prediction. This is so because there is an infinite number of reading profiles that are logically possible but that are not in this space: For example, whenever the reading accuracy of regular words is no higher than the reading accuracy of irregular words, reading accuracy for nonwords must be zero, and any profile where this is not so cannot be generated by any lesioning of DRC and therefore will be absent from the profile space, even though the observation of such a profile is logically possible. So, it could be the case that not a single one of the 100 patient data sets was at all close to any of the 40,200 lesioned DRC score profiles.

We can express in another way this point about it being possible for these simulations to falsify the DRC model. The model lesion technique varies two theoretically motivated parameters (how impaired the lexical route is, how impaired the nonlexical route is), but for each data set, the number of observations to be simulated is not two: It is five for some data sets and four for others. Given that, for each data set, the number of observations to be simulated is greater than the number of parameters to be varied, if the simulations fit the observations well, this is not a negligible finding.

For each of the 100 data sets, we identified the model profile that was closest to that patient data set profile by calculating the absolute difference between the patient's percentage correct and each lesioned DRC model's percentage correct for each of the different categories of stimuli (four categories—high-frequency exception, low-frequency exception, high-frequency regular, and low-frequency regular words—for the 66 cases where there were no nonword reading data, and a fifth category as well, nonwords,

[4] The model we used can be downloaded from http://www.maccs.mq.edu.au/~ssaunder/SemanticDementia/

[5] We had to discard two words, *trial* and *sour*, because in DRC's dialect, these are disyllabic and therefore not in DRC's vocabulary, and we replaced the nonword *gamp* with *gomp* because *gamp* is a word in DRC's vocabulary (it means umbrella).

for the 34 cases where such data were collected), averaging these absolute discrepancies, and then finding for each patient that lesioned DRC model for which the averaged absolute discrepancy between patient and lesioned model was minimized.

The median value of this average of absolute discrepancies between simulated and obtained percentage correct across the 34 data sets for which nonword reading was available was 1.4 (range: 0.0–8.0), indicating that each of these patient data sets was well fitted by some lesioned version of the DRC model. Table 3 shows the group patient data and the group simulation data; the fit of model to data is clearly satisfactory.

For the remaining 66 data sets, those for which nonword reading data were not available, the fits to individual patients were again satisfactory. The median value of this average of absolute differences between simulated and obtained percentages correct across the 66 data sets was 0.8 (range: 0.0–5.7), indicating that each of these patient data sets was also well fitted by some lesioned version of the DRC model. Table 3 shows the group patient data and simulation data; again, the fit of model to data is clearly satisfactory for both groups.

At the risk of belaboring the point about whether this method of lesioned DRC simulation is too powerful, we did a kind of Monte Carlo study. If it were the case that any possible pattern of accuracy on the five types of stimuli could be well matched by one of the 40,200 lesioned DRC models from which the best match could be chosen, then we would find good matches between lesioned DRC performance and obtained scores even when the latter were derived randomly rather than from testing patients. So, we created 100 random data sets by assigning random numbers between 0 and 100 for the percentage accuracy with the five item types and then searched the full space of 40,200 lesioned DRC models for each random data set's best match. If DRC's success in capturing the patient patterns was because it can capture any pattern, then it would capture the random patterns just as well as it captured the patient patterns. However, as Figure 1 shows, this was not so.

The median value of this average of absolute differences between simulated and obtained percentage correct across the 100 random data sets was 16.0 (range: 2.8–33.3). For the 100 patient simulations, this median was 1.0 (range: 0.0–8.0). That shows that the range of possible patterns is far larger than the range of patterns that the set of lesioned DRCs could accurately capture, yet the 100 patterns that were actually seen in the patient data set were all captured well by a lesioned DRC model.

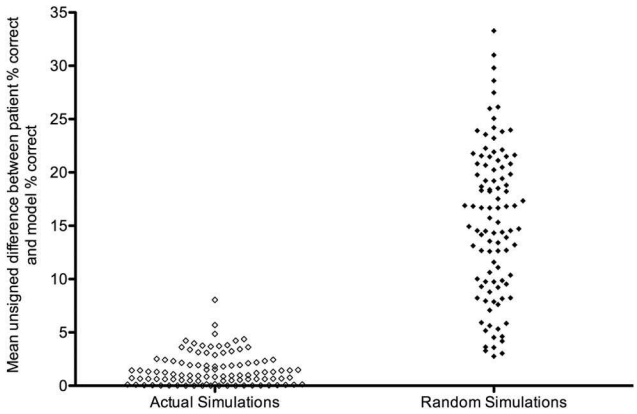

Figure 1. Best dual route cascaded model fits to the 100 patient data sets (left) and 100 random data sets (right).

Comparing Triangle Model Simulations With DRC Simulations

We can now turn to the results obtained by Woollams et al. (2007) that were simulated in their article using the triangle models approach (these are results only for word reading; as discussed below, the triangle model did not simulate the impaired nonword reading shown by some of the patients), so as to consider how well the DRC model fares, in comparison to the triangle model approach, when attempting such simulations. Here, we consider every result reported by Woollams et al. for reading of words, the triangle model simulation of that result, and the DRC model simulation of that result.

One of the variables studied by Woollams et al. (2007) was severity. For the patient data, *severity* means severity of the semantic deficit, as measured by a composite score derived from picture-naming and spoken-word-to-picture-matching scores. For the triangle model simulations, the severity of the semantic deficit was represented by the degree to which the "S"→P input in the model was weakened and made noisier. For the DRC model, of course, there was no semantic deficit to vary in severity since lesioning of a semantic system was not involved in the DRC simulations. That is because, on the DRC account of reading in semantic dementia, the association between a patient's semantic deficit and his or her reading accuracy is just that—an association, not a causal relationship.

Table 3
Reading Accuracy in the Two Groups of Patients and Their DRC Simulations

	34 data sets with nonword data		66 data sets without nonword data	
Category	Patients	DRC	Patients	DRC
High-frequency regular words	92.50	94.76	94.00	94.45
Low-frequency regular words	83.56	84.21	81.53	81.41
High-frequency exception words	86.69	89.14	83.45	85.17
Low-frequency exception words	60.75	59.87	53.79	52.84
Nonwords	78.90	78.30		

Note. DRC = dual route cascaded model.

Nevertheless, severity effects can be simulated by the DRC model. For each particular patient data set, our simulations yielded a specific best fitting lesioned DRC model (i.e., a model with a particular combination of a lexical lesion of a certain degree with a nonlexical lesion of a certain degree) and, hence, a particular accuracy score for each different stimulus type for that lesioned model and, therefore, for that patient data set simulation. Therefore, for each lesioned DRC model, one can define *severity* as the severity score of the particular patient whose data set that lesioned model simulates.

That allows one to plot accuracy of reading of each of the four word types as a function of severity not only for reading by the patients but also for reading by the DRC model, and the patient and model plots can be compared as a way of investigating how well the simulations match the patient data. This is done in Figure 2. For all four word types, the relationship between reading accuracy and severity seen in the patient plots is very well captured in the lesioned DRC plots.

The plots in Figure 3 may be directly compared to the plots in Figure 6 of Woollams et al. (2007), which present the same patient data plots along with triangle model plots. Comparisons between these two figures show clearly that the relationship between severity and accuracy of reading of each of the four word types as seen in the patients is captured more accurately by the DRC model than by the triangle model.

Woollams et al. (2007) reported the results of regression analyses to investigate severity effects. These analyses indicated that the relationship between severity and reading accuracy was stronger for low-frequency than for high-frequency words and stronger for exception words than for regular words for both the patient data and the triangle model simulations. Table 4 shows the results of these regression analyses plus the results of the regression analyses for the DRC simulations.

For the four sets of word data (we discuss the nonword data later), the DRC R^2 values are much more similar to the patient R^2 values (average difference: 0.005) than the triangle model R^2 values are (average difference: −0.24), and the DRC intercept values are more similar to the patient R^2 values (average difference: −0.85) than the triangle model intercept values are (average difference: 1.50). Hence the effect of severity on word reading accuracy in the patients is captured more accurately by the DRC model than by the triangle model, confirming the impression gained by comparing our Figure 2 with Figure 6 of Woollams et al. (2007).

Word Reading Accuracy

For the patients and for the triangle model simulations, reading accuracy was significantly affected by regularity (exception word reading worse than regular word reading) and frequency (high-frequency words read more accurately than low-frequency words). For both the patient data and the triangle model data, these two variables interacted, reflecting the larger effect of regularity on low-frequency words. The interaction between regularity and frequency was independent of level of severity.

All of this was also true for the DRC simulations: An analysis of covariance with regularity and frequency as factors and patient severity score as a covariate yielded significant effects of regularity, $F(1, 98) = 157.5$, $p < .0001$, and frequency, $F(1, 98) = 178.9$, $p < .0001$, and a significant interaction between these variables, $F(1, 98) = 27.3$, $p < .001$. The interaction among frequency, regularity, and severity was not significant ($p = .490$). Hence, the pattern of results in the patient data was completely captured by the DRC simulations (as it was by the triangle model simulations).

LARC Errors

A legitimate alternative reading of components (LARC) error is "a response in which the orthographic components of the stimulus are pronounced in accordance with correspondences contained in another existing monosyllabic word" (Woollams et al., 2007, p. 325). All regularization errors are by definition LARC errors, but LARC errors can also occur with regular words (e.g., reading the regular word *food* to rhyme with *blood* or *good*).

In both the patient data and in the triangle model simulations (see Woollams et al., 2007, Figure 7), percentage LARC errors (calculated as a percentage of total responses, correct or incorrect) showed the same pattern of effects of regularity and frequency as overall error rates (i.e., main effects of both frequency and regularity and an interaction between these two factors, with this interaction independent of severity) The same results were obtained with the DRC simulations: The proportion of incorrect responses by the DRC model that were LARC errors showed strong effects of regularity, $F(1, 98) = 158.3$, $p < .001$, and frequency, $F(1, 98) = 77.4$, $p < .001$, and a Frequency × Regularity interaction, $F(1, 98) = 70.25$, $p < .001$. The interaction among frequency, regularity, and severity was not significant ($p = .356$). Hence, the pattern of results in the patient data was completely captured by the DRC simulations (as it was by the triangle model simulations).

Figure 3 plots the percentage of DRC's LARC errors with each of the four word types as a function of patient severity. Figure 7 of Woollams et al. (2007) provided such plots for the patient data and for the triangle model simulations. Inspection of these two figures reveals that the DRC simulation plot is much more similar to the patient data plot than the triangle model plot is.

We conclude that all of the features of the data reported by Woollams et al. (2007) that have been simulated using the triangle model approach can also be simulated by the DRC model and, moreover, that these DRC simulations are superior to the triangle model simulations: The patient plots of accuracy of reading for the different word types as a function of severity are more accurately reproduced in the DRC simulations than in the triangle model simulations, the same is true of the patient plots of percentage LARC errors for the different word types as a function of severity, and in the regression analyses summarized in Table 3, the fit of simulation to patient data is much better for the DRC model than for the triangle model.

DRC Simulations of Aspects of Reading in Semantic Dementia That Were Not Simulated by the Triangle Model Approach

Nonword Reading

As noted earlier, it is not just exception word reading that is impaired in some cases of semantic dementia: Nonword reading impairment is impaired in some cases too (e.g., in 71% of the 34

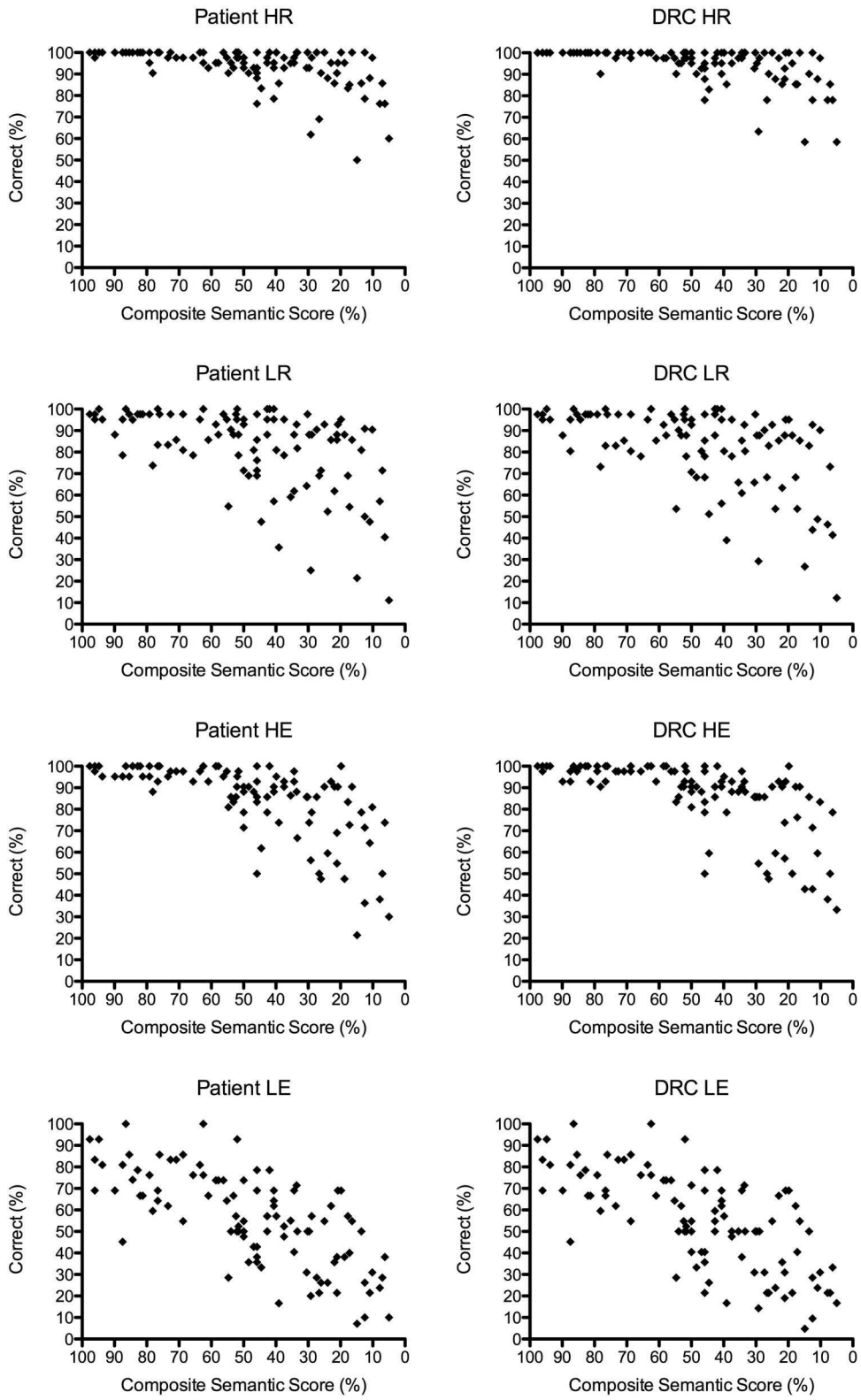

Figure 2. Effect of severity on reading of regular and irregular high- and low-frequency words in patients with semantic dementia and their dual route cascaded model simulations. HE = high-frequency exception words; HR = high-frequency regular words; LE = low-frequency exception words; LR = low-frequency regular words.

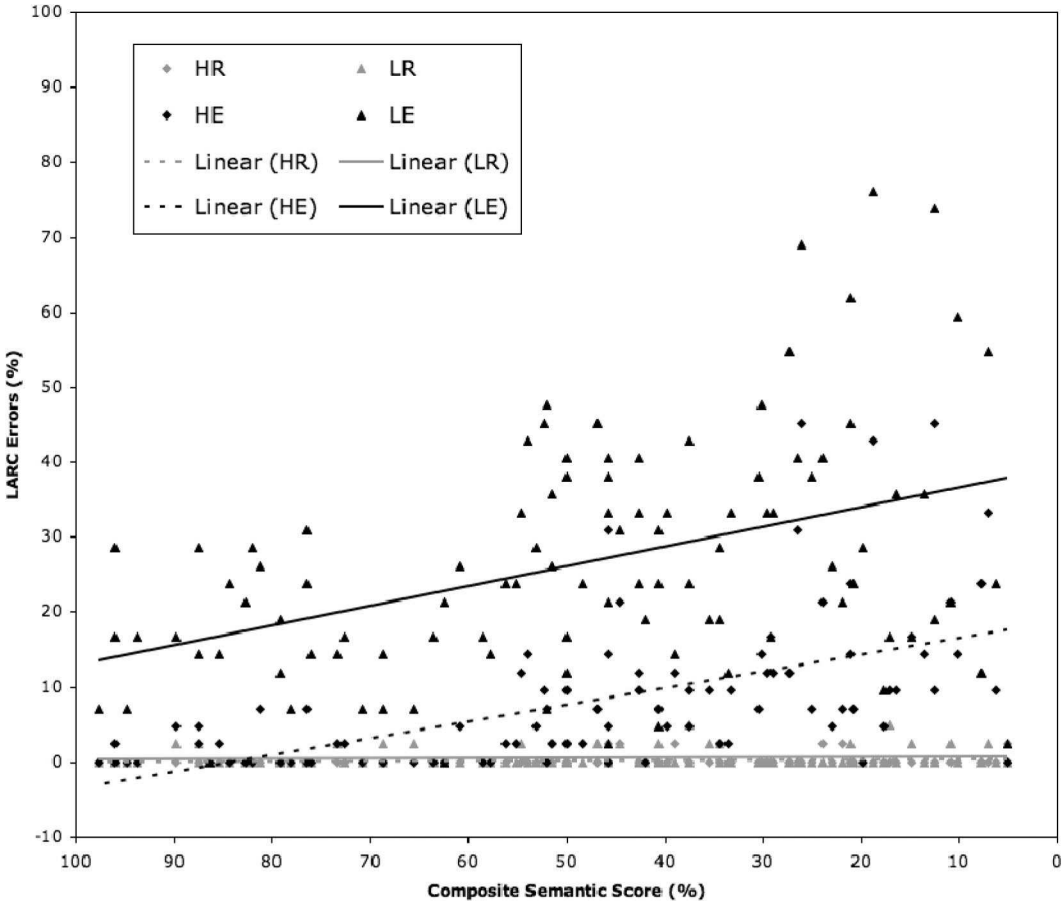

Figure 3. Percentage of lesioned dual route cascaded model responses that were LARC errors, as a function of severity, for regular and irregular high- and low-frequency words. HE = high-frequency exception words; HR = high-frequency regular words; LARC = legitimate alternative reading of components; LE = low-frequency exception words; LR = low-frequency regular words.

data sets of Woollams et al., 2007, where nonword reading was assessed). This impairment was not captured by the triangle model: Its nonword reading accuracy was exactly the same when it was lesioned as when it was intact. In contrast, we have already presented data showing that the DRC model can successfully do this: Table 2 reports that the mean nonword reading accuracy of the 34 lesioned DRC models simulating the 34 data sets that included nonword reading data was 78.30%, which compares well with the mean patient score of 78.89%.

These simulations of nonword reading are in fact remarkably accurate, as Figure 4 illustrates. The correlation between patient and lesioned DRC accuracy here is .997 ($p < .001$). It is important to appreciate that this accuracy was achieved in the presence of the constraint that accurate simulation of reading accuracy for high- and low-frequency regular and exception words was also required using the same lesion values as used to produce the results shown in Figure 4.

Relationship Between Severity and Nonword Reading Accuracy

Although Woollams et al. (2007) did not successfully model the fact that nonword reading is impaired in some cases of semantic dementia, they did (as mentioned above) collect nonword reading data for 34 of the 100 data sets they reported, and they observed that in this group, nonword reading performance "was somewhat impaired, with a mean accuracy of 78.53% (*SD* = 22.51). In contrast to all word conditions, however, nonword reading performance was not significantly predicted by level of semantic knowledge" (Woollams et al., 2007, pp. 328–329). This was also true of the individual-patient lesioned DRC simulations for this group. Nonword reading by this group of 34 lesioned DRCs was somewhat impaired, with a mean accuracy of 78.30% (*SD* = 22.54).

Woollams et al. (2007) also noted that that the degree of nonword reading impairment in this patient group was unrelated to severity. In the patient data, the regression of nonword reading accuracy on severity of semantic impairment (as measured by the composite semantic score) did not approach significance; this was also true of the DRC data.

In sum, it is clear that the nonword reading performance in this group of 34 cases of semantic dementia and its independence from severity of semantic impairment are modeled by DRC with a high degree of fidelity.

Yet there is a puzzle here. If, as our account claims, the impairment of exception word reading appears relatively early in the

disorder and the impairment of nonword reading relatively late, one would expect nonword reading to be better in a group of patients who are relatively early in the disorder—in some of these patients, nonword reading would not impaired at all—than in a group of patients who are late in the progression of the disorder. More generally, at the earliest stage of the disorder, reading aloud of all types of items is intact, whereas at the latest stages, reading aloud of all types of items is impaired. So, there must surely be an association between the severity of the semantic impairment and the severity of the nonword reading disorder. Why, then, is no such association observable in the data of Woollams et al. (2007) and in the DRC simulations of these data?

The relevant data here are those of the 34 data sets from 20 different patients tested on nonword reading on between one and five occasions.

The decision as to whether or not to test a patient on nonword reading on any testing occasions (nonword reading was tested on 34 of the 100 testing sessions) was not made systematically,[6] so it is possible that the testing sessions where nonword reading was measured were with patients whose range of severity of semantic impairment was restricted compared to the range across the full 100 data sets. However, this is not so: The composite semantic score for those tested on nonword reading was 48.32 ($SD = 4.31$), and for those not tested on nonword reading, it was 47.71 ($SD = 3.09$).

We did discover, however, that the absence of any relationship between severity of semantic impairment and nonword reading in the 34 data sets from 20 patients was due just to the data from one patient, Patient M.A. When that one patient's data are removed (this involves removing 5 data sets from the 34 because M.A. was tested on nonword reading on five occasions[7]), the regression of nonword reading on severity of semantic impairment is significant in the human data ($R^2 = .122$, one-tailed $p = .03$) and in the DRC simulations ($R^2 = .146$, $p = .041$).

Table 4
Regressions of Patient and DRC Reading Performance on Patient Severity and Regressions of Triangle Model Reading Performance on Strength of "S"→P Contribution in the Model

Category	Intercept	R^2
High-frequency regular words		
Patient data	84.5	0.26
DRC model	86.0	0.27
Triangle model	87.7	0.43
Low-frequency regular words		
Patient data	63.7	0.26
DRC model	64.1	0.26
Triangle model	60.5	0.54
High-frequency exception words		
Patient data	62.9	0.42
DRC model	66.5	0.40
Triangle model	59.1	0.57
Low-frequency exception words		
Patient data	26.8	0.50
DRC model	24.7	0.49
Triangle model	24.6	0.84
Nonwords		
Patient data	71.4	0.03 (*ns*)
DRC model	69.6	0.04 (*ns*)
Triangle model		

Note. DRC = dual route cascaded model; "S"→P = semantics to phonology.

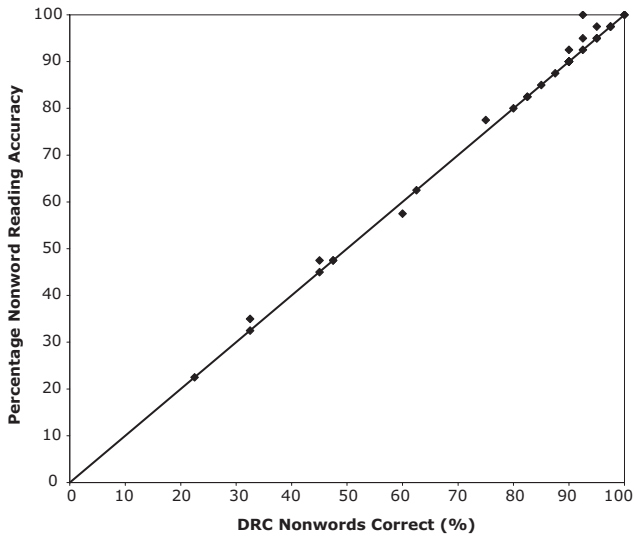

Figure 4. Nonword reading accuracy in 34 cases of semantic dementia and in dual route cascaded model (DRC) simulations of these cases.

To explore the way in which Patient M.A. was an outlier, Figure 5 plots nonword reading accuracy for patients and DRC simulations against severity of semantic impairment for each of the 34 data sets that contained nonword reading data. Here, it is clear that M.A. was quite different from all of the other patients: M.A.'s nonword reading impairment was present at a much earlier severity stage than is the case with all of the other patients. This is true also for the DRC simulations of M.A. All 5 of the other patients with very marked nonword reading impairments (<80% correct) were at late severity stages (all scoring <40% on the composite semantic test); again, this was also true for the DRC simulation data. We have nothing to say about what might explain why M.A.'s nonword reading impairment became apparent at an earlier severity stage than is characteristic of the other patients.

We note that even in M.A.'s data, there is a clear trend for nonword reading accuracy to decline as severity of semantic impairment increases, and since this effect was actually significant in this patient group when M.A.'s data were removed, we offer this as preliminary evidence that it is a general feature of semantic dementia that a nonword reading deficit is associated with the late stages of the disease.

The Dual Route Equation

Since, in the DRC account of the human reader, a correct translation of a nonword from print to speech by a human reader indicates correct operation of that reader's nonlexical route, one can estimate the integrity of the nonlexical route in any reader by measuring the accuracy with which nonwords are read. Similarly, since correct translation of an irregular word from print to speech

[6] Anna M. Woollams (personal communication, February 2008).

[7] M.A. was not tested on nonword reading on the first testing occasion, which is why there is no M.A.1 in Figure 5.

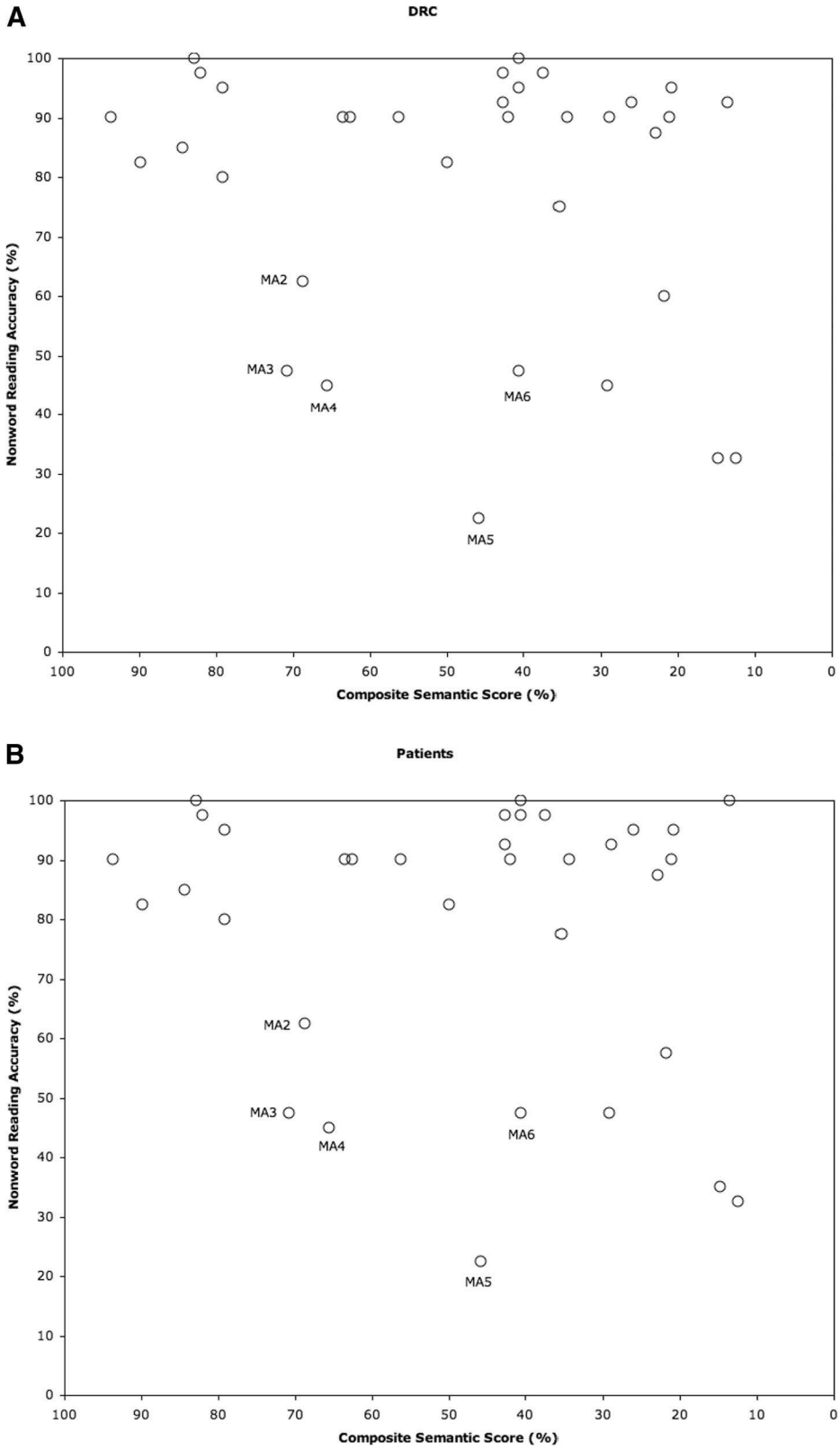

Figure 5. Nonword reading accuracy as a function of degree of semantic impairment, in patients (left) and lesioned dual route cascaded model (DRC) simulations (right). MA = Patient M.A.

indicates correct operation of the lexical route, one can estimate the integrity of the lexical route by measuring the accuracy with which exception words are read. Since a regular word will be read correctly whenever either route does its job correctly, the DRC model predicts that in any reader, one ought to be able to estimate the accuracy with which a set of regular words is read if one knows the accuracy with which the person reads exception words and nonwords, assuming that the regular and exception words are matched on relevant variables such as frequency and that the regular words and nonwords are matched on relevant variables such as length. The specific equation generated from these considerations is

$$\text{pr(regular word correct)} = \text{pr(exception word correct)} + (1 - \text{pr[exception word correct]}) \times \text{pr(nonword correct)}.$$

Coltheart et al. (2001, p. 247) reported results of the application of this equation to data from 1,488 children ages 7 to 15 years whose reading of regular words, exception words, and nonwords had been measured. For this sample, the correlation between obtained regular word accuracy and the accuracy predicted by the dual route equation was very high: .921. This was further explored by Castles et al. (2006), who analyzed data from nine separate samples[8] of children in all of whom reading of regular words, exception words, and nonwords had been measured. The nature of these samples (see Castles et al., 2006, for details) and the correlations between obtained regular word accuracy and the regular word accuracy predicted by this dual route equation are shown in Table 5.

These are all high correlations, indicating that the equation works well in predicting children's reading regardless of whether the children are normal readers, are dyslexic, or have suffered brain damage. Analyses of age effects showed that the accuracy of prediction by the equation is independent of age: The equation is just as accurate for children age 7 or 8 years (who can barely read) as it is for children age 14 or 15 years (most of whom are skilled adult-level readers). This is consistent with the view (Marshall, 1984) that the architecture of the reading system proposed in the DRC model is appropriate even for children who have only just begun to learn to read.

Table 5
Correlations Between Obtained Regular Word Reading Accuracy and Regular Word Reading Accuracy Predicted From the Dual Route Equation for Nine Samples of Children

Sample characteristics	Correlation of observed with predicted regular word reading accuracy
Children—normal readers ($N = 420$)	.829
Children—normal readers ($N = 56$)	.732
Children—normal readers ($N = 297$)	.736
Children—normal readers ($N = 242$)	.838
Children—normal readers ($N = 309$)	.814
Children—normal readers ($N = 812$)	.821
Children with dyslexia ($N = 53$)	.678
Children with dyslexia ($N = 40$)	.857
Children who had had strokes ($N = 17$)	.960

The reason this work was done with children (some normal reader samples, some disordered reader samples) is that adult normal readers would be at ceiling on all the word and nonword reading measures, and so, there would be no reading score variance for the equation to capture. That is of course not the case for adults with an acquired dyslexia—such as adults with semantic dementia. We therefore applied this equation to the data of the 34 cases of Woollams et al. (2007) for whom nonword reading data were collected (the equation cannot be applied unless data on regular words, exception words, and nonwords are available). For high-frequency words, the correlation between observed regular word reading accuracy and the prediction from the equation was .928 ($p < .001$); for low-frequency words, it was .924 ($p < .001$). Thus, the DRC equation makes rather accurate predictions, for individual patients with semantic dementia, of the patient's regular word reading accuracy given knowledge of the patient's exception word reading accuracy and nonword reading accuracy. We see no way in which any existing triangle model could be used to do this.

Pro Tem Summary: Conclusions Concerning Simulation of Reading in Semantic Dementia by the Triangle and DRC Models

We conclude that the DRC model is clearly the superior model here, our reasons for this conclusion being the following:

1. Every effect that the triangle model simulates is more accurately simulated by the DRC model;
2. The dual route equation that so accurately predicted regular word reading accuracy in the patients has no counterpart in the triangle model framework;
3. The triangle model was unable to simulate the nonword reading impairment in semantic dementia, whereas the DRC simulation of this impairment was highly accurate; and
4. The DRC model offers simulations at the individual-patient level, whereas the triangle model simulations do not.

This brings to an end our discussion of the data of Woollams et al. (2007) and its simulation by the triangle and DRC models. We conclude our article with a brief discussion of two other sets of data on acquired dyslexia and attempts to simulate these using the lesioned DRC method we have introduced in this article.

Two Other DRC Simulations of Acquired Dyslexia

The Study by Gold et al. (2005)

There were three groups of subjects in this study: 6 patients with semantic dementia, 10 patients with Alzheimer's disease, and 14 healthy controls. They were given 163 regular words varying in length from three to six letters to read aloud under speeded reading

[8] Some of these samples were included in the analyses of Coltheart et al. (2001).

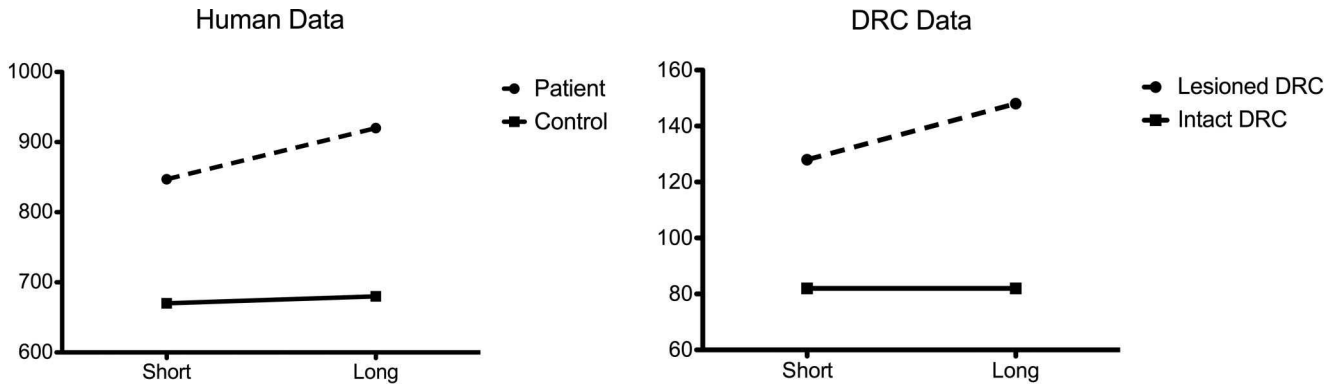

Figure 6. Effect of length on reaction time for reading aloud of regular words, for controls and patients with semantic dementia (left) and for intact and lesioned dual route cascaded (DRC) models (right).

conditions. The words were categorized as short (three to four letters) or long (five to six letters) and varied in frequency. Accuracy of reading these words was at ceiling for all three groups. All three groups of subjects showed an effect of word frequency on reading aloud latency.

According to the DRC analysis of reading in semantic dementia, if these patients with semantic dementia had been in Phase 3 of the disorder, they would have been less accurate than healthy controls on low-frequency regular word reading, which they were not. They were not in Phase 1 of the disorder either because they all had prominent rather than mild semantic impairments (Gold et al., 2005, Figure 1). Thus, these patients were all in Phase 2 of the disorder and hence would have been expected to be surface dyslexic, which they were: On the American National Adult Reading Test (Grober & Sliwinski, 1991) test of irregular word reading, all scored below the means of the healthy and Alzheimer's groups, with 5 showing particularly poor performance. Any patient in Phase 2 will be reading at least some regular words via the nonlexical route. Reading via the nonlexical route generates a substantial length effect that is minimal or absent in reading via the lexical route (Weekes, 1997), a result that is also true of the DRC model's reading (Coltheart et al., 2001) Therefore, the DRC analysis predicts that although all three groups of subjects were at ceiling on accuracy of reading regular words, the patients with semantic dementia should show a larger effect of length on reading aloud latencies for regular words than the other two groups. This is what was found by Gold and colleagues, who therefore concluded (Gold et al., 2005, pp. 842–843) that their data were better explained by the DRC approach than by the triangle models approach.

To computationally verify the DRC analysis here, we carried out a simulation study using the same short and long regular words as were used in the study by Gold and colleagues (2005).[9] These words were run through the intact DRC model (simulating control subjects) and through a DRC model with nonlexical route intact but lexical route lesioned by 94% (simulating severe pure surface dyslexia). Figure 6 shows the results. In the human data, the length effect for control readers was not significant, whereas the patients with semantic dementia showed a large and significant effect of length. The same contrast is seen between intact and lesioned DRC models. For the intact model, the effect of length was not significant ($F < 1$); for the lesioned model, it was highly significant, $F(1, 157) = 8.465$, $p = .004$.

The Study by Crisp and Lambon Ralph (2006)

This was a study of 12 heterogeneous stroke patients with acquired dyslexias of various sorts, the inclusion criterion being the presence of at least one of the three symptoms: (a) an imageability effect on word reading, (b) the occurrence of semantic paralexias in word reading, and (c) worse reading of nonwords than words—(in fact, all 12 patients showed nonword reading worse than normal, their percentage correct for nonword reading ranging from 0% to 70%). The matched materials relevant to our analyses that were administered to this patient group were as follows:

1. Thirty-six low-frequency monosyllabic regular words from Monaghan and Ellis (2002);

2. Thirty-six low-frequency monosyllabic exception words from Monaghan and Ellis (2002), matched to the regular words on frequency, age of acquisition, neighborhood size, and number of letters; and

3. Twenty-four monosyllabic nonwords from the PALPA battery (Kay, Lesser, & Coltheart, 1992).

Application of the DRC equation to the prediction of regular word reading accuracy from exception word reading accuracy and nonword reading accuracy with the data from these patients yielded a correlation between predicted and observed regular word reading accuracy of .973 ($p < .001$). Figure 7 plots observed against predicted regular word reading accuracy for the 12 patients.

The data of these 12 patients were also individually simulated using the lesioned DRC method described above. The 96 items were presented to every lesioned DRC, and for each patient, the lesioned DRC that yielded the minimum absolute average dis-

[9] We thank Brian Gold for providing us with these stimuli. We excluded one word (*fire*) because in DRC's dialect, it is disyllabic.

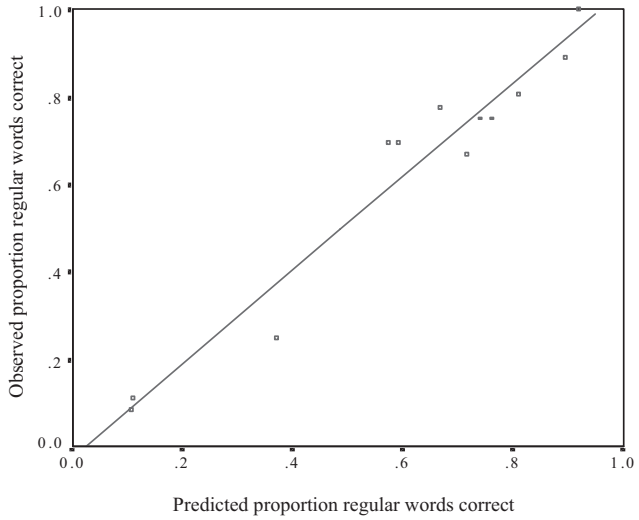

Figure 7. Relationship between obtained regular word accuracy and regular word accuracy predicted from the dual route equation for 12 patients with various forms of acquired dyslexia.

crepancy between the patient's proportions correct on the three types of items and the proportions correct of the lesioned DRC model was identified. The results are shown in Table 6. As can be seen, for 9 of the 12 patients, there existed a lesioned DRC whose data were identical to the data of the patient. For the remaining 3 patients, the mean unsigned discrepancy was not zero but was very small: .009. Let us point out again that this is not a vacuous finding: For each patient, two parameters are being used to fit three data points, so successful fitting is not guaranteed. The fit of DRC to individual patients was therefore excellent in this lesioning-of-DRC study.

General Conclusions

It was claimed in the first study reporting the full DRC model (Coltheart et al., 2001) that the DRC model was able to offer accounts of a much wider variety of facts about adult skilled visual word recognition and reading aloud than any other computational model of reading then in existence. More recently, Rastle and Coltheart (2006) focused on two specific questions about the nature of the human reading system about which the DRC and the PDP triangle model approaches disagree—"Is there any processing in the human reading system that is serial?" and "Are there representations in the human reading system that are local rather than distributed?"—and provided evidence that the answer to both questions is yes (as asserted by the DRC model) rather than no (as asserted by the PDP approach). In particular, Rastle and Coltheart summarized eight different lines of evidence that, they argued, show that there is serial processing in the human reading system and hence are inconsistent with any purely parallel processing model of that system. We therefore consider that the DRC model is currently superior to the PDP triangle model approach in its ability to account for skilled adult reading. We do not mean to claim that there are no findings with adult skilled readers that the triangle models can simulate and the DRC model cannot. There are some: consistency effects, for example, including consistency

Table 6
DRC Simulations of Accuracy of Reading Regular Words, Exception Words, and Nonwords in 12 Patients With Acquired Dyslexia

Patient	Regular words	Exception words	Nonwords	Lexical lesion	Nonlexical lesion	Mean absolute discrepancy between model and patient data
R.S.	.694	.361	.333	76.0%	68.0%	.009
DRC	.694	.333	.333			
P.G.	.750	.583	.375	53.5%	77.5%	.009
DRC	.750	.555	.375			
D.B.	1.000	.806	.583	31.5%	67.0%	.009
DRC	.972	.806	.583			
L.R.	.083	.028	.083	96.5%	93.5%	.000
DRC	.083	.028	.083			
M.M.	.111	.110	.000	84.5%	91.5%	.000
DRC	.111	.110	.000			
R.J.	.250	.278	.083	70.0%	96.0%	.000
DRC	.250	.278	.083			
A.B.	.806	.583	.542	55.0%	72.5%	.000
DRC	.806	.583	.542			
M.R.	.750	.639	.333	57.0%	87.0%	.000
DRC	.750	.639	.333			
B.N.	.667	.472	.458	62.5%	77.0%	.000
DRC	.667	.472	.458			
T.H.	.889	.861	.250	23.0%	76.0%	.000
DRC	.889	.861	.250			
N.S.	.778	.528	.292	51.5%	80.0%	.000
DRC	.778	.528	.292			
T.J.	.694	.556	.083	49.0%	85.5%	.000
DRC	.694	.556	.083			

Note. DRC = dual route cascaded model.

effects in acquired dyslexia (Patterson & Behrmann, 1997; Perry, Ziegler, & Zorzi, 2007). Yet these findings are few, whereas the number of effects that the DRC model has simulated but that the triangle models have not is large.

We also consider that the DRC approach is currently superior to the PDP approach in its ability to account for various forms of acquired and developmental dyslexia. This argument has been made by Coltheart (2006) in relation to acquired surface and phonological dyslexia and by Castles et al. (2006) in relation to developmental dyslexia. In this article, we have made this argument in relation to the patterns of acquired dyslexia seen in semantic dementia. We have compared the accounts of reading in semantic dementia offered by the DRC model and the PDP triangle model and have shown (a) that even those aspects of reading in semantic dementia that have been simulated using the triangle model are more accurately simulated by the DRC models and (b) that there are aspects of reading in semantic dementia that have not been simulated by the triangle model but are accurately simulated by the DRC model.

Hence, we conclude that the DRC model is currently to be preferred to triangle models based on the PDP approach as an account of how human readers recognize printed words and read aloud.

References

Blazely, A., Coltheart, M., & Casey, B. (2005). Semantic dementia with and without surface dyslexia. *Cognitive Neuropsychology, 22,* 695–717.

Bright, P., Moss, H. E., Stamatakis, E. A., & Tyler, L. K. (2008). Longitudinal studies of semantic dementia: The relationship between structural and functional changes over time. *Neuropsychologia, 46,* 2177–2188.

Bub, D., Cancelliere, A., & Kertesz, A. (1985). Whole word and analytic translation of spelling-to-sound in a non-semantic reader. In K. E. Patterson, M. Coltheart, & J. C. Marshall (Eds.), *Surface dyslexia: Neuropsychological and cognitive studies of phonological reading* (pp. 15–34). London, England: Erlbaum.

Castles, A., Bates, T., & Coltheart, M. (2006). John Marshall and the developmental dyslexias. *Aphasiology, 20,* 871–892.

Cipolotti, L., & Warrington, E. K. (1995). Semantic memory and reading abilities: A case report. *Journal of the International Neuropsychological Society, 1,* 104–110.

Coltheart, M. (2005). Modeling reading: The dual-route approach. In M. J. Snowling & C. Hulme (Eds.), *The science of reading: A handbook* (pp. 6–23). Oxford, England: Blackwell.

Coltheart, M. (2006). Acquired dyslexias and the computational modelling of reading. *Cognitive Neuropsychology, 23,* 96–109.

Coltheart, M., Rastle, K., Perry, C., Langdon, R., & Ziegler, J. (2001). DRC: A dual route cascaded model of visual word recognition and reading aloud. *Psychological Review, 108,* 204–256.

Crisp, J., & Lambon Ralph, M. (2006). Unlocking the nature of the phonological–deep dyslexia continuum: The keys to reading aloud are in phonology and semantics. *Journal of Cognitive Neuroscience, 18,* 348–362.

Gerhand, S. (2001). Routes to reading: A report of a non-semantic reader with equivalent performance on regular and exception words. *Neuropsychologia, 39,* 1473–1484.

Gold, B. T., Balota, D. A., Cortese, M. J., Sergent-Marshall, S. D., Snyder, A. Z., Salat, D. H., . . . Buckner, R. L. (2005). Differing neuropsychological and neuroanatomical correlates of abnormal reading in early-stage semantic dementia and dementia of the Alzheimer type. *Neuropsychologia, 43,* 833–846.

Grober, E., & Sliwinski, M. (1991). Development and validation of a model for estimating premorbid verbal intelligence in the elderly. *Journal of Clinical and Experimental Neuropsychology, 13,* 933–949.

Harm, M. W., & Seidenberg, M. S. (2004). Computing the meanings of words in reading: Cooperative division of labor between visual and phonological processes. *Psychological Review, 111,* 662–720.

Hodges, J. R., Patterson, K., Oxbury, S., & Funnell, E. (1992). Semantic dementia: Progressive fluent aphasia with temporal lobe atrophy. *Brain, 115,* 1783–1806.

Kay, J., Lesser, R., & Coltheart, M. (1992). *PALPA: Psycholinguistic Assessments of Language Processing in Aphasia.* Hove, England: Erlbaum.

Lambon Ralph, M. A., Ellis, A., & Franklin, S. (1995). Semantic loss without surface dyslexia. *Neurocase, 1,* 363–369.

Marshall, J. C. (1984). Toward a rational taxonomy of the developmental dyslexias. In R. N. Malatesha & H. A. Whitaker (Eds.), *Dyslexia: A global issue* (pp. 45–58). The Hague, the Netherlands: Martinus Nijhoff.

McCandliss, B. D., Cohen, L., & Dehaene, S. (2003). The visual word form area: Expertise for reading in the fusiform gyrus. *Trends in Cognitive Sciences, 7,* 293–299.

McCarthy, R. A., & Warrington, E. K. (1986). Phonological reading: Phenomena and paradoxes. *Cortex, 22,* 359–380.

McKay, A., Castles, A., Davis, C., & Savage, G. (2007). The impact of progressive semantic loss on reading aloud. *Cognitive Neuropsychology, 24,* 162–186.

Monaghan, J., & Ellis, A. W. (2002). What exactly interacts with spelling–sound consistency in word naming? *Journal of Experimental Psychology: Learning, Memory, and Cognition, 28,* 183–206.

Nestor, P. J., Fryer, T. D., & Hodges, J. (2006). Declarative memory impairments in Alzheimer's disease and semantic dementia. *NeuroImage, 30,* 1010–1020.

Nickels, L., Biedermann, B., Coltheart, M., Saunders, S., & Tree, J. (2008). Computational modelling of phonological dyslexia: How does the DRC model fare? *Cognitive Neuropsychology, 25,* 165–193.

Noble, K., Glosser, G., & Grossman, M. (2000). Oral reading and dementia. *Brain and Language, 74,* 48–69.

Patterson, K. (1990). Alexia and neural nets. *Japanese Journal of Neuropsychology, 6,* 90–99.

Patterson, K., & Behrmann, M. (1997). Frequency and consistency effects in a pure surface dyslexic patient. *Journal of Experimental Psychology: Human Perception and Performance, 23,* 1217–1231.

Patterson, K., Seidenberg, M. S., & McClelland, J. L. (1989). Connections and disconnections: Acquired dyslexia in a computational model of reading processes. In R. G. M. Morris (Ed.), *Parallel distributed processing: Implications for psychology and neurobiology* (pp. 131–181). London, England: Oxford University Press.

Perry, C., Ziegler, J. C., & Zorzi, M. (2007). Nested incremental modeling in the development of computational theories: The CDP+ model of reading aloud. *Psychological Review, 114,* 273–315.

Plaut, D., McClelland, J. M., Seidenberg, M. S., & Patterson, K. (1996). Understanding normal and impaired word reading: Computational principles in quasi-regular domains. *Psychological Review, 103,* 56–155.

Rastle, K., & Coltheart, M. (2006). Is there serial processing in the reading system; and are there local representations? In S. Andrews (Ed.), *From inkmarks to ideas: Current issues in lexical processing* (pp. 3–24). Hove, England: Psychology Press.

Schwartz, M. F., Saffran, E. M., & Marin, O. S. (1980). The word order problem in agrammatism: I. Comprehension. *Brain and Language, 10,* 249–262.

Seidenberg, M. S., & McClelland, J. L. (1989). A distributed, develop-

mental model of word recognition and naming. *Psychological Review, 96,* 523–568.

Snowden, J. S., Goulding, P. J., & Neary, D. (1989). Semantic dementia: A form of circumscribed cerebral atrophy. *Behavioural Neurology, 2,* 167–182.

Weekes, B. S. (1997). Differential effects of number of letters on word and non-word naming latency. *Quarterly Journal of Experimental Psychology: Human Experimental Psychology, 50*(A), 439–456.

Woollams, A. M., Lambon Ralph, M. A., Plaut, D. C., & Patterson, K. (2007). SD-squared: On the association between semantic dementia and surface dyslexia. *Psychological Review, 114,* 316–339.

Received March 17, 2008
Revision received March 10, 2009
Accepted March 11, 2009 ■

Postscript: Reading in Semantic Dementia—A Response to Woollams, Lambon Ralph, Plaut, and Patterson (2010)

Max Coltheart
Macquarie University

Jeremy J. Tree
Swansea University

Steven J. Saunders
Macquarie University

Let us first summarize what Woollams and her colleagues (Woollams, Lambon Ralph, Plaut, & Patterson, 2007, 2010) and we agree about, which is as follows. All patients with semantic dementia have impairments of semantic memory. All of them also show atrophy to the anterior temporal lobes, which spreads along the temporal pole to more posterior regions. Other regions (e.g., frontal lobes) may also eventually be affected, and the pattern of atrophy can differ between hemispheres. All patients with semantic dementia will show surface dyslexia at some point or another during the evolution of their condition. Across any group of patients, any measure of comprehension ability (e.g., the average of picture-naming and picture-to-word-matching performance) will be positively correlated with accuracy of reading of irregular words.

Our fundamental disagreement has to do with the interpretation of the last of these facts. Woollams and colleagues regard this correlation as involving a causal relationship: The semantic impairment is causing the reading impairment because, to a degree that differs from person to person and from exception word to exception word, correct reading of an exception word needs input from semantics to phonology. In contrast, we regard this correlation as representing what cognitive neuropsychologists sometimes refer to as a mere association, due to the influence of a third variable, which, in this case, is the severity of the atrophy. The more extensive this atrophy is, the worse comprehension performance will be and the worse exception word reading will be, but this is not telling us anything about the nature of the reading system or about the role of semantics in reading aloud because there is nothing causal about the observed correlation between these two variables. So in response to a question such as "Why would the ability of the patients to name pictures and match spoken words to be expected to have an effect on the ability of the DRC model to read aloud?", our answer would be that the ability of patients to name pictures and match spoken words is a measure of the extent of the patients' cortical atrophy and that the greater this atrophy is, the more damage there will be to nonsemantic components of the reading system. That is why a computational model of reading that has no semantic system (the dual route cascaded [DRC] model) can nevertheless very accurately simulate, as Coltheart, Tree, and Saunders (2010) demonstrated, the patterns of exception word, regular word, and nonword reading seen in individual patients with semantic dementia. Textbook treatments of cognitive neuropsychology standardly argue that inferences from dissociations between abilities are much more secure than inferences from associations between abilities (see, e.g., Ellis & Young, 1996, p. 6), a tenet that we take Woollams and colleagues to reject since they are making a theoretical inference on the basis of the association between semantic impairment and reading impairment.

A second tenet of cognitive neuropsychology to which we adhere but that, it appears, Woollams and colleagues also reject is that when studying any particular neuropsychological impairment of cognition, the real task is to explain the patterns of results seen in individual patients having that impairment, not just to explain the group means of a set of such patients, that is, to do single case studies rather than group studies (see, e.g., Ellis & Young, 1996, p. 9). That is why we have sought to investigate whether the DRC model can produce an accurate simulation of every one of the 100 data sets from patients with semantic dementia that were collected by Woollams et al. (2007). In contrast, the simulations using the triangle model reported by Woollams et al. (2007, 2010) do not involve any attempt to model the individual patterns of performance with exception words, regular words, and nonwords seen in data from individual patients. On the contrary, the impaired nonword reading seen in the majority of cases of semantic dementia (seen in 71% of the data sets of Woollams et al., 2007, that included nonword reading data) was not modeled by Woollams et al. (2007), and the new modeling reported by Woollams et al. (2010), where impaired nonword reading was produced in the triangle modeling by adding noise to the computation of phonology from semantics, aimed only at matching the model to the group mean nonword reading of the patients. There was no attempt here to explain why some patients were within the normal range on nonword reading and some very severely impaired. The worst performance of the triangle model on nonword reading was 49% correct (see Figure 2 of Woollams et al., 2010), whereas in the patient data, there were seven patients who scored less than 49%. The triangle model simulation produced a mean nonword reading level of 81.4%, with a range from 49% to 100%. In the patient data, the mean was 78.9%, with a range from 23% to 100%. In the DRC model simulation data, the mean was 78.3%, with a range from 23% to 100%, and across the 34 data sets, the correlation between the DRC-simulated nonword reading performance and the patient reading performance was .997. This is a clear demonstration that the triangle model simulations of nonword reading performance in semantic dementia done to date fit the patient data much less well than do the DRC model simulations.

We have failed to understand the discussion by Woollams and colleagues criticizing our view about the phases of reading impairment in semantic dementia. They and we agree (a) that there are patients with semantic dementia whose reading aloud is intact, (b) that there are patients with semantic dementia who are impaired at reading aloud exception words but normal at reading aloud nonwords, (c) that there are patients with semantic dementia who are impaired at both exception word reading and nonword reading, and (d) that there are no reports of patients with semantic dementia who are in the normal range of exception word reading but impaired at nonword reading. Surely, it follows from these four facts that in the course of semantic dementia, impaired exception word reading emerges only after semantic impairment has already emerged and that impaired nonword reading emerges only after both semantic impairments and then impaired exception word reading have both already emerged? That is what our claim is regarding the phases of reading impairment in semantic dementia. Do Woollams and colleagues dispute this claim? If so, on what basis? The data presented in Figure 1 of Woollams et al. (2010) do not bear on this claim. What would bear on this claim would be the discovery of patients with semantic dementia, intact exception word reading, and impaired nonword reading.

Concerning what Woollams et al. (2010) have to say about assessing model fit: What we want when we are seeking to computationally model any kind of acquired cognitive impairment is to show that the performance of the model when lesioned shows similarities to the performance of the relevant patients. The closer this similarity is, the stronger the patient data are as support for the model. So, in analyses aimed at assessing model fit, one variable has to be data from the model, and the other has to be data from the patients. We cannot understand why Woollams et al. followed Seidenberg and Plaut (2006) in referring to this as "comparing apples to oranges" (Woollams et al., 2010, p. 277), nor why they think that the appropriate way of assessing model fit is for both variables to be derived from the model. To do that is to carry out an investigation just of the model—an exercise in pure connectionism, a branch of mathematics rather than of cognitive science—not to seek to explain patient data in terms of a proposed model. We therefore are unable to see the point of the analyses reported in Tables 1 and 2 of Woollams et al.

We conclude with some remarks on the goals of the computational modeling of cognition, another topic on which our views differ greatly from those of Woollams and colleagues. In common with other computational modelers of cognition such as Jacobs and Grainger (1994) and Perry, Ziegler, and Zorzi (2007), we adhere to the practice of nested incremental modeling. What this means is that if one discovers some result that is inconsistent with one's current model—call this Model A—even if one is able to show that some new model—call this Model B—can account for this new result, the replacement of Model A by Model B does not count as making progress unless it is shown that all of the results that Model A could account for can also be accounted for by Model B, that is, the set of results that Model A can explain is nested inside the set of results that Model B can explain. As Woollams et al. (2010) noted, their views about the goals of computational modeling are as set out in Seidenberg and Plaut (2006), who clearly repudiated the principle of nested incremental modeling:

> The PDP [parallel distributed process] approach is frustrating to some because there is no single simulation that constitutes *the* model of the domain. The model seems like a moving target. . . . Each model shares something with all of the others, but each model differs as well. Where, then, is the integrative model that puts all the pieces together? The answer is, there is none and there is not likely to be one. (Seidenberg & Plaut, 2006, p. 42)

We must confess to having been flabbergasted when we read these words. If there is no fact of the matter about what the human reading system is like—about what its cognitive architecture actually is—then what is the point of the experimental psychology of reading, and what is it that computational modelers of reading are trying to model?

References

Coltheart, M., Tree, J. J., & Saunders, S. J. (2010). Computational modeling of reading in semantic dementia: Comment on Woollams, Lambon Ralph, Plaut, and Patterson (2007). *Psychological Review, 117,* 256–272.

Ellis, A. W., & Young, A. W. (1996). *Human cognitive neuropsychology* (augmented ed.). Hove, England: Psychology Press.

Jacobs, A. M., & Grainger, J. (1994). Models of visual word recognition: Sampling the state of the art. *Journal of Experimental Psychology: Human Perception and Performance, 20,* 1311–1334.

Perry, C., Ziegler, J. C., & Zorzi, M. (2007). Nested incremental modeling in the development of computational theories: The CDP+ model of reading aloud. *Psychological Review, 114,* 273–315.

Seidenberg, M. S., & Plaut, D. C. (2006). Progress in understanding word reading: Data fitting versus theory building. In S. Andrews (Ed.), *From inkmarks to ideas: Current issues in lexical processing* (pp. 25–49). Hove, England: Psychology Press.

Woollams, A. M., Lambon Ralph, M. A., Plaut, D. C., & Patterson, K. (2007). SD-squared: On the association between semantic dementia and surface dyslexia. *Psychological Review, 114,* 316–339.

Woollams, A. M., Lambon Ralph, M. A., Plaut, D. C., & Patterson, K. (2010). SD-squared revisited: Reply to Coltheart, Tree, and Saunders (2010). *Psychological Review, 117,* 273–283.