Simultaneous simulations of pure, surface and phonological acquired dyslexia within a full computational model of the primary systems hypothesis

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Abstract

According to the primary systems hypothesis, reading requires interactions of visual-orthographic, phonological and semantic systems. Damage to each primary system generates very different types of acquired dyslexia. Variants of the connectionist 'triangle' models of reading have been developed to investigate individual acquired dyslexia. However, only a few studies have investigated multiple acquired alexia within one framework. Importantly, there are no studies that simultaneously simulate both central dyslexia (e.g., surface and phonological dyslexia) and peripheral dyslexia (e.g., pure alexia). That is largely due to the lack of a visual component in the traditional reading models. To verify the predictions made by the primary systems hypothesis, we developed a connectionist 'deep' multi-layer triangle model of reading including visual, orthographic, phonological and semantic processing layers. We investigated whether damage to the model could produce the general behavioural patterns of impaired performance observed in patients with the corresponding reading deficits. Crucially, damage to the visual-orthographic, phonological or semantic components of the model resulted in the expected reading impairments associated with pure alexia, phonological dyslexia and surface dyslexia, respectively. The simulation results demonstrated for the first time that neurologically-impaired reading including both central and peripheral dyslexia could be addressed within a single triangle model of reading. The findings are consistent with the predictions made by the primary systems hypothesis.

Keywords: Computational Modelling; Word Reading; Pure Alexia; Phonological Dyslexia; Surface Dyslexia

Introduction

According to the primary systems hypothesis (Patterson & Lambon Ralph, 1999), reading requires interactions of visual, phonological and semantic systems. Damage to different systems generates very different types of acquired dyslexia. For example, disruption to the phonological system leads to phonological-related impairments as observed in phonological-deep dyslexia (Crisp, Howard, & Lambon Ralph, 2011; Crisp & Lambon Ralph, 2006). According to recent large studies (Woollams, Halai, & Lambon Ralph, 2018; Halai, Woollams, & Lambon Ralph, 2020), phonological-deep dyslexia occurs in approximately 80% to 90% of aphasic patients with damage to left peri-sylvian areas following middle cerebral artery (MCA) stroke. These patients have prominent phonological deficits as a part of their aphasia. By contrast, the vast majority of surface dyslexic cases in the literature are patients with semantic dementia (plus a handful of others with anterior temporal damage). Semantic dementia, the temporal lobe variant of frontotemporal dementia, arises from atrophy centred on the anterior temporal lobes bilaterally resulting in progressive degradation of the semantic system (Hodges & Patterson, 2007; Patterson et al., 2006; Woollams, Lambon Ralph, Plaut, & Patterson, 2007). These two types of dyslexia are considered *central dyslexia* (Shallice & Warrington, 1980).

Additionally, disruption to the visual-orthographic system underpins pure alexia (Arguin, Fiset, & Bub, 2002; Behrmann, Plaut, & Nelson, 1998; Damasio & Damasio, 1983; Roberts, Lambon Ralph, & Woollams, 2010) or neglect dyslexia (Primativo, Arduino, De Luca, Daini, & Martelli, 2013; Vallar, Burani, & Arduino, 2010), which are forms of *peripheral dyslexia* (Shallice & Warrington, 1980). Specifically, pure alexia arises after damage to the left ventral occipitotemporal (vOT) region, most commonly following a posterior cerebral artery (PCA) stroke, alongside a few cases of posterior ventral occipitotemporal (vOT) glioma (e.g., Rice et al. 2021; Roberts et al. 2013). Hence, surface, phonological, and pure alexia are distinct from each other in terms of aetiology, location of damage and the disordered reading phenotype (described in detail below), albeit certain acquired dyslexic features can sometimes overlap, transdiagnostically, across types.

Computational modelling has served as a powerful tool to advance our understanding of language processes and their disorders by making theoretical ideas testable and forcing the theories to be rigorously specified (Coltheart, Rastle, Perry, Langdon, & Ziegler, 2001; Harm & Seidenberg, 2004; Norris, 2006; Perry, Ziegler, & Zorzi, 2007; Plaut, McClelland, Seidenberg, & Patterson, 1996; Seidenberg & McClelland, 1989). Variants of the computational models of reading have been applied to investigate individual acquired dyslexia including phonological dyslexia (Coltheart et al. 2001; Plaut et al., 1996; Welbourne & Lambon Ralph, 2005, 2007; Welbourne, Woollams, Crisp, & Lambon Ralph, 2011), surface dyslexia (Coltheart et al. 2001; Plaut et al., 1996; Woollams et al., 2007), deep dyslexia (Plaut & Shallice, 1993) as well as multiple acquired alexia within

one framework (Welbourne et al., 2011). These advanced computational studies of acquired dyslexia have provided valuable insights into the functional locus of impairments, their consequences on behaviours, and the mechanisms of reading processes. However, most models have focused on simulating different types of dyslexia individually. As far as we are aware, there is no computational model that has simulated both central and peripheral dyslexia within one framework. For models developed within the connectionist triangle modelling framework (Plaut et al., 1996; Welbourne & Lambon Ralph, 2005, 2007; Welbourne et al., 2011), that is largely due to a lack of a visual component in the traditional modelling reading framework through which the model would be presented with the visual form of words and would convert these into internal “orthographic” intermediate representations that precede semantic and phonological activation. Consequently, the primary systems hypothesis has not yet been properly tested for its adequacy in computational modelling of acquired reading disorders.

Additionally, it remains unclear whether the model would learn and behave as expected when all the core reading processing components (visual-orthographic, phonological, and semantic) are included in the same system. These were the overarching goals of the present study. Specifically, we investigated whether damage to different locations in a ‘deep’ variant of the triangle model would result in patterns of impaired performance mimicking those observed in patients with functionally corresponding reading deficits - namely pure alexia, phonological dyslexia, and surface dyslexia as predicted by the primary systems hypothesis (Patterson &

Lambon Ralph, 1999). Below we provided a brief review of pure alexia, phonological dyslexia, and surface dyslexia and their diagnostic symptoms in word reading.

Pure alexia (PA) is a neuropsychological deficit generally caused by lesions in the left ventral occipitotemporal region (Damasio & Damasio, 1983). PA patients generally show strong word-length effects on reading times, and it is thought by many researchers to result from damage to visual processing (Arguin et al., 2002; Behrmann et al., 1998; Fiset, Arguin, & McCabe, 2006; Rice et al. 2021; Roberts et al., 2010; Roberts et al., 2013). In addition, some PA patients' word naming performance is sensitive to lexical variables such as frequency (Behrmann et al., 1998; Johnson & Rayner, 2007; Montant & Behrmann, 2001), regularity (Behrmann et al., 1998), orthographic neighbourhood size (Arguin et al., 2002; Fiset et al., 2006; Montant & Behrmann, 2001), age of acquisition (Cushman & Johnson, 2011) and imageability (Behrmann et al., 1998).

Patients with phonological dyslexia (PD) are characterised by a relative impairment of nonword reading in the context of better word reading accuracy (Beauvois & Dérouesné, 1979; Patterson & Kay, 1982). Many studies suggest that the functional locus of phonological dyslexia is a disturbance to generalised phonological processing because the patients' reading performance is strongly correlated with, and mirrored by their non-reading phonological deficits, and they exhibit the same qualitative performance characteristics on reading and non-reading tasks, including lexicality and imageability effects (Crisp & Lambon Ralph, 2006; Patterson & Marcel, 1992; Rapcsak et al., 2009).

Most cases of surface dyslexia come from patients with semantic dementia (SD: Jefferies, Lambon Ralph, Jones, Bateman, & Patterson, 2004; McCarthy & Warrington, 1986; Patterson & Hodges, 1992; Woollams et al., 2007), which is characterised by progressive degradation of conceptual knowledge associated with atrophy centred on the ventrolateral and polar temporal lobe (Hodges, Patterson, Oxbury, & Funnell, 1992; Nestor, Fryer, & Hodges, 2006). Indeed, the link between surface dyslexia and semantic dementia is long-lived and prominent, with early descriptions proposing the alternative name of “semantic dyslexia” for this entity (Shallice & Warrington, 1980; Shallice, Warrington, & McCarthy, 1983). Importantly, from a large-scale patient study and an accompanying model (Woollams et al. 2007), it is evident that semantic impairment almost inevitably leads to surface dyslexia; with some minor individual variation in terms of the level of semantic impairment needed before the surface pattern inevitably emerges. The findings are further supported by fMRI (Hoffman et al. 2015) and TMS (Woollams et al. 2016) studies with healthy participants. Behaviourally, patients with surface dyslexia show greater difficulty in reading aloud words with inconsistent spelling-to-sound mappings, particularly for low frequency items, whereas their nonword reading abilities are relatively preserved (see Table 1 in Woollams et al. 2007). There are a handful of mild SD patients who initially have good reading performance (Blazely, Coltheart, & Casey, 2005; Cipolotti & Warrington, 1995) but often these patients exhibit surface dyslexia as their semantic impairment progresses (Schwartz, Marin, & Saffran, 1979; Schwartz, Saffran, & Marin, 1980) perhaps reflecting variations in premorbid

individual differences in the reliance on semantics for reading aloud (Hoffman, Lambon Ralph, & Woollams, 2015; Woollams et al., 2007; Woollams, Madrid, & Lambon Ralph, 2017).

Building on the existing triangle models of reading, this study developed a deep variant of the triangle model of reading aloud including visual, orthographic, phonological and semantic processing layers, illustrated in Figure 1. Somewhat like deep neural networks for visual recognition, our model was presented with the raw visual image of words and nonwords on the input layer. This contrasts with previous computational models of reading that utilised a pre-defined orthographic coding approach (Harm & Seidenberg, 2004; Plaut et al., 1996). To illustrate, in the Plaut et al.'s study (1996), the word *foot* underwent parsing into onset /f/, vowel /oo/, and coda /t/ clusters of graphemes. However, employing such a pre-defined orthographic coding scheme hinders the model's ability to discern variations in letter lengths (Chang, Furber & Welbourne, 2012a). Moreover, the use of a predefined orthographic input does now allow us to explain how pure alexia arises from a more generalised visual impairment (Behrmann et al. 1998; Roberts et al. 2013). Thus, instead, the current model was required to transform the raw visual image of input through a series of layers, such that the deepest "visual layer" with its learned internal representations served as input to a "triangle" model framework with parallel pathways to semantic and phonological representations (as well as connection between the two). Following training and assessment of the baseline undamaged model, we investigated whether damage to the model could produce the general

behavioural patterns of impaired performance observed in patients with the corresponding functional reading deficits. Specifically, relative to the model that was not damaged, we predicted that: (a) all of the patient models would show a strong frequency effect. Critically, the PA model would show the key diagnostic symptom of a strong word-length effect as observed in Roberts et al. (2013); the SD model would show the key diagnostic symptom of a strong consistency effect as observed in Woollams et al. (2014); (b) for nonword reading, the performance of the PD model would show the key diagnostic symptom of impaired nonword reading performance, reflecting in lexicality effects (Welbourne & Lambon Ralph, 2007).

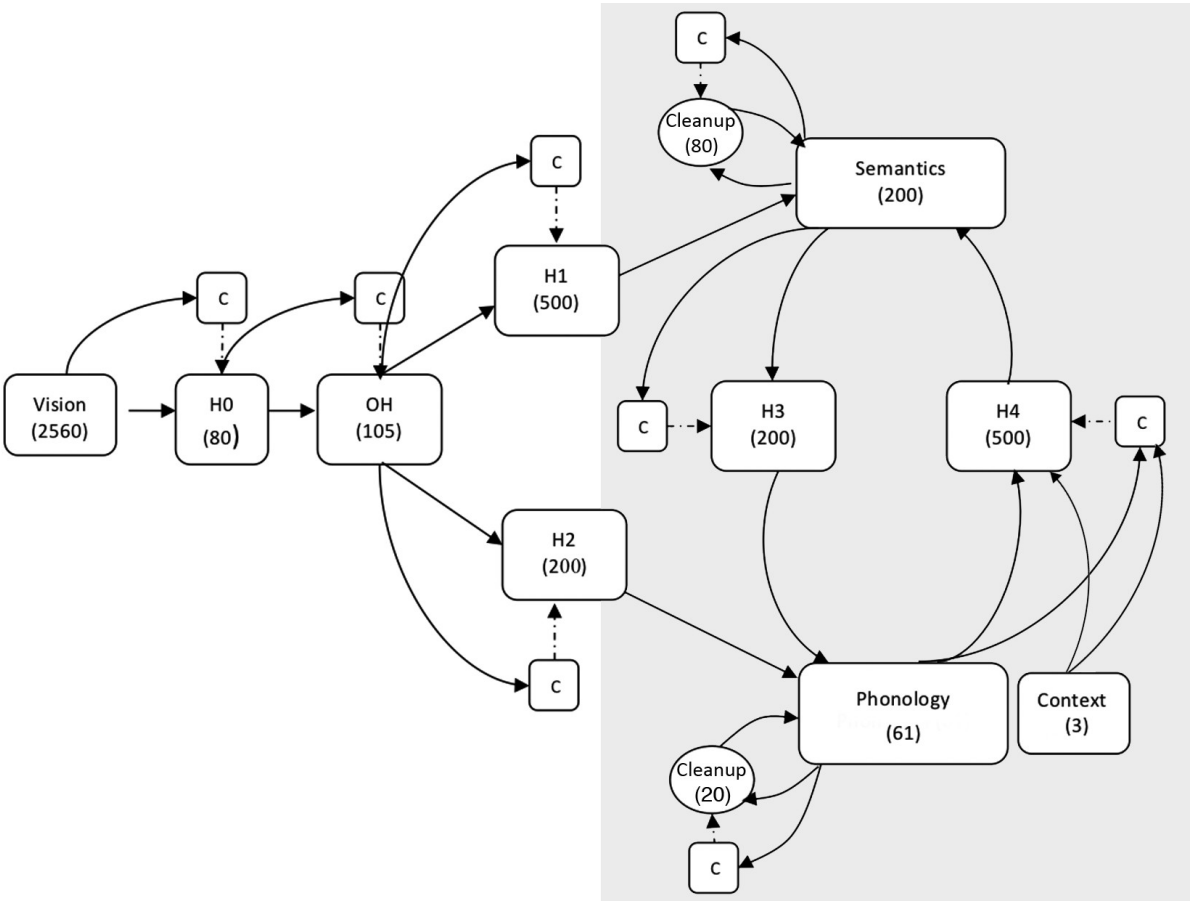

Figure 1. The architecture of the model. It consists of visual, orthographic (OH), phonological and semantic layers connected by five intermediate hidden layers. The two output layers have clean-up units to stabilise the representations. There is a local control unit for each layer except input and

output layers. Each local control unit receives the same inputs as the layer that it controls, and connects to that layer via inhibitory only connections (dashed lines). The numbers in the brackets indicate the number of units. H: Hidden; c: Control.

Simulations of multiple acquired dyslexia

Before testing the model after damage to different locations in the model, we confirmed through a series of detailed simulations and analyses that the undamaged model was able to learn word production and word comprehension tasks both orally and visually. The undamaged model also showed good generalisation in its ability to read nonwords and produced a range of standard reading effects including frequency, consistency, word length and the interactions between frequency and consistency, and between frequency and word length as observed in behavioural studies (Taraban & McClelland, 1979; Weekes, 1997; Balota et al. 2004). As the present study focused on the investigations of the damaged model, to keep the manuscript as concise as possible, we briefly described training methods for the undamaged model in the Method section and the detailed examination of the undamaged model are reported in the Supplementary Materials.

Based on this successful undamaged model, we then investigated whether damage to different locations in the model would result in the patterns associated with pure alexia, phonological dyslexia, and surface dyslexia as predicted by the primary systems hypothesis (Patterson & Lambon Ralph, 1999).

Method

The model of word reading

The simulation was based on a ‘deep’ multi-layer computational model of word reading reflecting the general triangle modelling framework (Harm & Seidenberg, 2004; Plaut et al., 1996; Seidenberg & McClelland, 1989; Welbourne & Lambon Ralph, 2007; Welbourne, Woollams, Crisp, & Lambon Ralph, 2011). Crucially the model started reading from the raw visual image of words and nonwords on the input layer without pre-defined orthographic representations. The model was a recurrent neural network model, and it was implemented using the Light Efficient Network Simulator (LENS) software (Rohde, 1999).

Model Architecture

The architecture of the model is shown in Figure 1. The model had two separate pathways for recognising words from visual input: a phonological pathway and a semantic pathway. The visual layer was connected to the OH layer via a hidden layer. The OH layer was equivalent to the single orthographic layer in traditional triangle models, though the representations were learned through the course of training rather than being supplied as predefined inputs. The OH layer was connected to both the phonological and semantic layers via two more hidden layers. Both the phonological and semantic layers were connected to their own set of clean-up units, which helped stabilise the representations. Additionally, there were three context units that provided contextual information for

disambiguating homophones. Phonological and semantic units were connected to each other via two hidden layers[1].

To effectively regulate the activation of different layers in the model, control units were added to each layer except input and output layers. These control units received the same inputs as the layer they were connected to, and all their outgoing connections were inhibitory. That allowed them to control the activation of all the units in a layer simultaneously. The control units were free to learn to regulate the activity of the units they controlled in whatever fashion reduces error. It was anticipated that for a control unit connected to other units in the deep layers, it would learn to exert a strong inhibition early in training which gradually decreased with the time course of training. The control mechanism enabled activations in the model to propagate forward from visual layers, allowing units to remain in proximity to their equilibrium states before the propagating activation reached the deep layers of the model. The effects of control units on model performance are reported in the Supplementary Materials.

The training corpus consisted of 2,971 monosyllabic words used in previous modelling studies (Plaut et al. 1996; Seidenberg & McClelland, 1989), covering word stimuli in various empirical reading studies (e.g., Taraban & McClelland, 1987).

[1] The number of hidden units in the model was determined by pilot studies to ensure its capacity to support pre-literate language learning and reading. Due to the mappings between vision, orthography, phonology and semantics varying in nature (some arbitrary mappings and others quasi-regular and componential), the number of hidden units varied accordingly to support learning.

Representations

Following Chang, Furber, and Welbourne (2012a), the visual input of the model was fed with bitmap images of words in Arial 12-point lower case font. Each word image consisted of ten letter slots with each slot comprising 16x16 pixels. Thus, there were in total 2,560 visual input units. Each word was positioned with its vowel aligned on the central slot of the image. For example, *rote* is represented as _ _ _ *r o t e* _ _ _. For words that have a second vowel, the second vowel was placed right next to the first vowel, for example, *root* is represented as _ _ _ *r o o t* _ _ _.

This scheme of phonological representations was the same as that used in Plaut et al.'s (1996, Table 2) model. Each word was represented 61 phoneme units which were parsed into onset, vowel and coda clusters of phonemes with specific units used to represent each possible phoneme in each cluster. For example, *sprint* is represented as */sprint/*, and *sprout* is represented as /sprWt/. For the context representations, three context units were used. For non-homophones, the context units were set to zero. Within the same homophone family, different context units were assigned, one for each. The maximum number of meanings corresponding to a given pronunciation in the training corpus is four.

The semantic representations were taken from a semantic space system based on co-occurrence statistics (Chang, Furber, & Welbourne, 2012b). Each semantic representation was composed of 200 semantic units. The key feature of this semantic representation is that each semantic vector for an item contains information about the presence (e.g., dogs have four legs) and absence (e.g., doges never fly) of its semantic features.

Representations derived from this semantic system have validity in reflecting human judgments on semantic categories (see Chang, Furber, & Welbourne, 2012b for details).

Training Procedures

The training procedure was separated into two phases. In phase 1 the links between phonology and semantics were trained (shown in grey in Figure 1) in order to simulate pre-literate language learning in children. In phase 2 the full reading model was trained. All output layers in the model were given a fixed negative bias of -2 to encourage sparse representations. In phase 1, the phonology-semantics model was subdivided into two parts in which the production model learned the mappings from semantics to phonology, and the comprehension model learned the mappings from phonology to semantics. Both the production and comprehension models were trained on the entire corpus. The probability of each word being presented to the model was determined by its logarithmic frequency[2]. Slightly different learning rates and weight decays were used to train the two models because of the nature of the difficulty of the tasks. The production model was trained with a learning rate of 0.2 and a weight decay of 1E-7. The comprehension model was trained with a learning rate of 0.05 and weight decay of zero. Each example was presented for six intervals of

[22] This use of logarithmic frequency was to enhance training efficiency, especially for the model to learn words with very low frequency. As demonstrated by Plaut et al. (1996), reading models trained with raw frequency or compressed frequency behave very similarly, with the exception that the model trained with raw frequency generally show stronger frequency effects.

network time and each interval of time was divided into three ticks[3]. In each presentation, the input pattern was clamped onto the appropriate units for six intervals of time. For the last two intervals, the activations of the target units were tested. All the units in the model used a sigmoid activation function. Error score, the difference between the actual activation and its target activation, was used to calculate weight changes between units according to a back-propagation through time (BPTT) algorithm (Pearlmutter, 1989, 1995). If the output unit's activation was within 0.1 of its target, no error signals were computed, and the weight connections were not updated. After the separate training, the two models were combined and there was a short period of interleaved training to fine-tune the model.

After the phase 1 training, the weights were loaded into the reading model and frozen so that during reading training, the model was able to utilise the pre-trained knowledge of the mappings between phonology and semantics mimicking children's oral language skills prior to learning to read. In phase 2, the model was trained on the reading tasks with a learning rate of 0.1, a weight decay of 1E-8 and a momentum of 0.9. Each visual representation was presented for ten intervals of network time (again each interval of time was broken into three time ticks). The model was required

[33] According to Rohde (1999), intervals and ticks in Lens (the neural network simulator that we are using) represent abstract units of time in the network's environment, serving as measures of continuous time. A time interval in Lens can be thought of as a second or tenth of a second of real time, which can be digitalised into ticks. The more ticks per interval, the finer the gain at which the continuous network is simulated, however, it also extends the duration of training required.

to produce correct phonological and semantic patterns. For the last two intervals, the output activations were compared with their target phonological or semantic representations and errors were computed. No error was computed when the output units' activation and target were within 0.001. Again, logarithmic frequency was used to determine the probability with which word was presented to the model. To preclude any possibility that simulation result could be generated from one particular set of initial weights, 20 models with different initial weights were trained. According to Bishop (2006, Section 5.5.2, p.259), in neural networks, the error measured with respect to independent data (e.g., an unseen dataset) often shows a decrease at first, followed by an increase as the network starts overfitting. Training can therefore be stopped at the point of smallest error with respect to the unseen data set in order to obtain a network having good generalization performance. Thus, in the present study, the accuracy rate on regular nonword pronunciation was used to determine the endpoint of training.

Testing Procedures

The testing procedures for both training phases were exactly the same. The semantic accuracy was based on the Euclidean distances between the activations of the semantic units and each of the semantic representations in the training corpus (Monaghan, Chang, Welbourne, & Brysbaert, 2017; Monaghan, Shillcock, & McDonald, 2004). Specifically, the semantic representation which was the closest to the activation of the semantic units was taken as the semantic output. If the output was the same as the target representation, it was a correct response. Error score was

measured as the sum of the squared differences over the semantic layer. The procedure for the generation of the phonological output was the same as that used in Plaut et al.'s (1996, *p*.15) study. Briefly, for the vowel units, the most activated vowel unit was selected as output. Onset and coda units were divided into groups of mutually exclusive units and the highest active unit above 0.5 was taken as the output for each group. If no unit was active above 0.5 then the group did not contribute to the output. For some affricate-like units such as /ks/, /ts/ or /ps/, if one of these units was active along with their components then the order of the components in the response was reversed. The phonological accuracy was determined by whether, for each phoneme unit, the closest phoneme to the model's actual production was the same as its target phoneme. Error score was measured as the sum of the squared differences between the activation of each input unit and its target activation.

Simulations of Acquired Dyslexia

The approach to simulate PA and PD patient types was similar with the only difference being the location of the damage to the model. Whereas SD is unlike the other two deficits as it is the result of a progressive disorder (Hodges et al., 1992). The location of the damage to the model for simulating PA, PD and SD patients is illustrated in Figure 2. For PA damage, 90% of the links connecting to or from the H0 layer coupled with 90% of the links into or out of the connected control units were randomly removed. The model was retrained for 400,000 training times with data sampled every 10,000 epochs for the last 50,000 training times. Damage of 40% and 60% of the links to the model were also conducted to simulate relatively mild and

moderate PA patients respectively. For PD damage, the model was damaged by randomly removing 90% of the links into and out of the phonological layer together with 90% of the links into or out of the connected control units. Again, the model was retrained for 400,000 training times with data sampled from every 10,000 training times for the last 50,000 training times. For SD damage, following Welbourne and colleagues (2007, 2011), we simulated it by repeatedly interleaving very mild damage and retraining. We randomly removed 0.8% of the links into or out of the semantic layer together with the links into or out of the connected control units and then trained the network for 20 times. This process was repeated with data sampled from every 20 training times for the last 100 training times. For the two damage-plus-recovery (PA and PD) models, the amount of retraining was chosen for the model to recover to a stable performance level and all three models were matched to the overall performance reported in patient studies (Roberts et al., 2013; Welbourne & Lambon Ralph, 2007; Woollams et al., 2014).

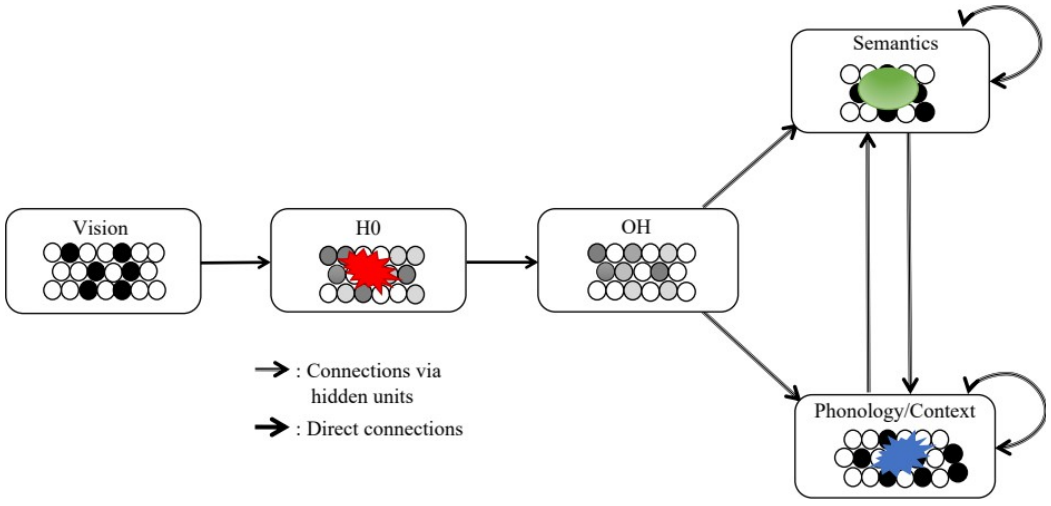

Figure 2. The specific areas of damage in the intact reading model, used to simulate pure alexia (PA), phonological dyslexia (PD), and semantic dementia (SD), are as follows: the visual-orthographic processing layer for

PA (red), the phonological processing layer for PD (blue), and progressive degradation of the semantic layer for SD (green).

The damaged models were tested on the effect of frequency and consistency (Taraban & McClelland, 1987), the effect of word-length (Chang, 2012), and nonword reading (Glushko, 1979) as was done for the intact model (see stimulus details in Supplementary). Critically, the key investigation was to see if the damaged models could simulate the diagnostic symptoms of different types of acquired dyslexia. For the PA, we investigated whether the PA model (damage to the visual system) could simulate word-length effects for patients with different levels of severity as reported by Roberts et al. (2013), in which more severe patients show stronger word-length effects in word naming. For the PD, we investigated whether the PD model (damage to the phonological system) could simulate relatively preserved reading performance but worse nonword reading performance as reported by Welbourne and Lambon Ralph (2007). Lastly, for the SD, we investigated whether the SD model (progressive degradation of the semantic system) could simulate a strong consistency effect in word naming as reported by Woollams et al. (2014). For each of the target effects, we conducted an ANOVA analysis for each model separately for the comparisons with the patient data. Then, to further investigate whether the effect was different across the models, the model data were combined, and additional ANOVAs were conducted with model type (Intact, PA, PD, and SD) as a between-group variable.

Results

In line with the patient studies (Patterson & Kay, 1982; Rice et al. 2021; Roberts et al. 2010; Woollams et al., 2007), the ANOVA analyses of word naming and nonword reading were conducted on the accuracy rates except for the word length effects, in which the analyses were conducted on error scores as a proxy for behavioural response times. As the accuracy rates were bounded between 0 and 100, and that violated the assumptions of ANOVA, we transformed them into unbounded empirical logit scores prior to the analyses. We used the following formula to compute the empirical logit (Donnelly and Verkuilen, 2017).

where n refers to the number of observations used to compute y. In the present study, n was 20 because there were 20 models for each model type.

All the descriptive and individual statistical results of the PA, PD, SD, and the intact models are summarised in Tables 1 and 2 respectively. For the PA model, a two-way ANOVA analysis on the word-length effect was conducted with word-length as a within-group variable, with severity as a between-group variable and with error scores as a dependent variable. The result showed both word-length, $F(2, 114) = 20.75$, $p < .001$ and severity, $F(2, 57) = 20.06$, $p < .001$, were significant predictors. Critically, the interaction between word-length and severity was also significant, $F(4, 114) = 12.48$, $p < .001$. As can be seen in Figure 3, the simulation result from the PA model (based on the “error” score metric – the difference between the target and activated output pattern) is consistent with the word-length effects observed in the PA patients (as measured in terms of reading times

to correctly named items) at different levels of severity by Roberts et al. (2013).

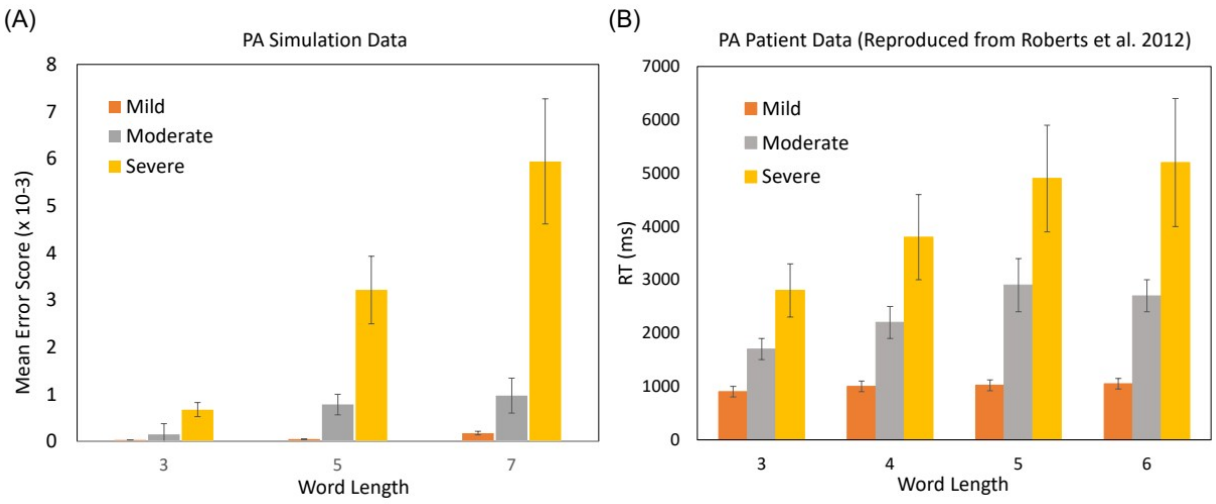

Figure 3. The word-length effects for different levels of severity produced by (A) the PA model and (B) the PA patient data (Roberts et al., 2013).

For the PD model, both the average reading accuracy across all words in the frequency and consistency task and the lexicality effect size produced were computed. Lexicality effect size is defined as the difference in accuracy between reading high-frequency regular words and reading regular nonwords (Welbourne & Lambon Ralph, 2007). The result was illustrated in Figure 4. As can be seen, the PD model had a relatively preserved reading accuracy compared to nonword reading, consistent with the PD patient data reported in Welbourne and Lambon Ralph (2007).

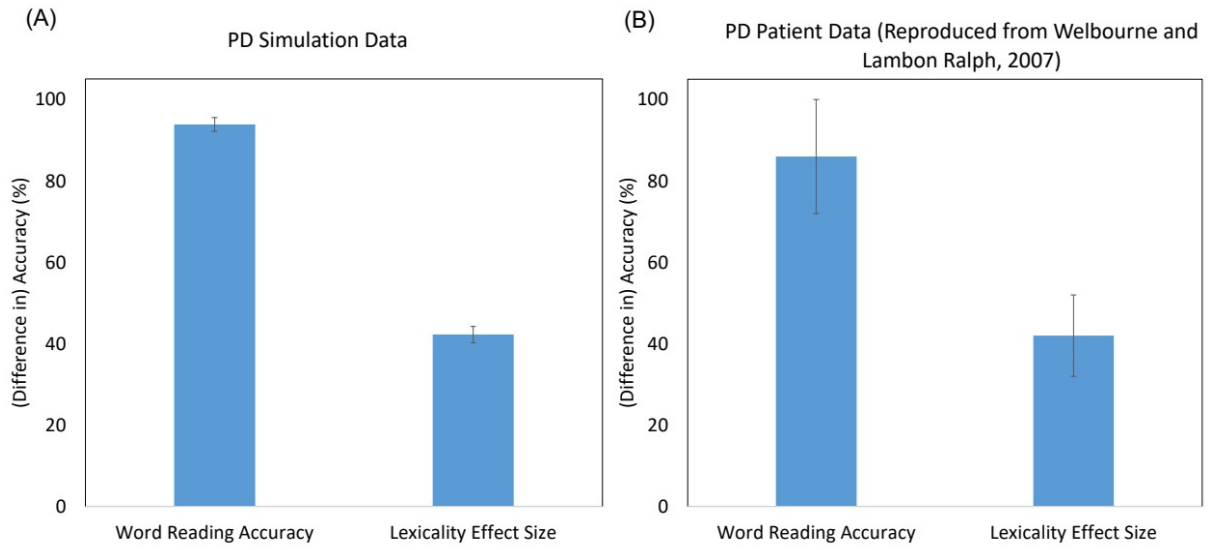

Figure 4. The reading accuracy and lexicality effect size (the difference in accuracy between reading high-frequency regular words and reading regular nonword) produced by (A) the PD model and (B) the PD patient data reproduced from Welbourne and Lambon Ralph (2007).

For the SD model, the ANOVA analysis on the frequency and consistency task showed both frequency and consistency effects were significant, $F(1, 19) = 12.3$, $p < .001$, and $F(1, 19) = 215.6$, $p < .001$. The interaction between frequency and consistency was not significant. The consistency effect was observed for both high and low frequency words, in which the worst performance was observed for low frequency inconsistent words. As illustrated in Figure 5, the simulation result is in accordance with the SD patients reported in Woollams et al. (2014).

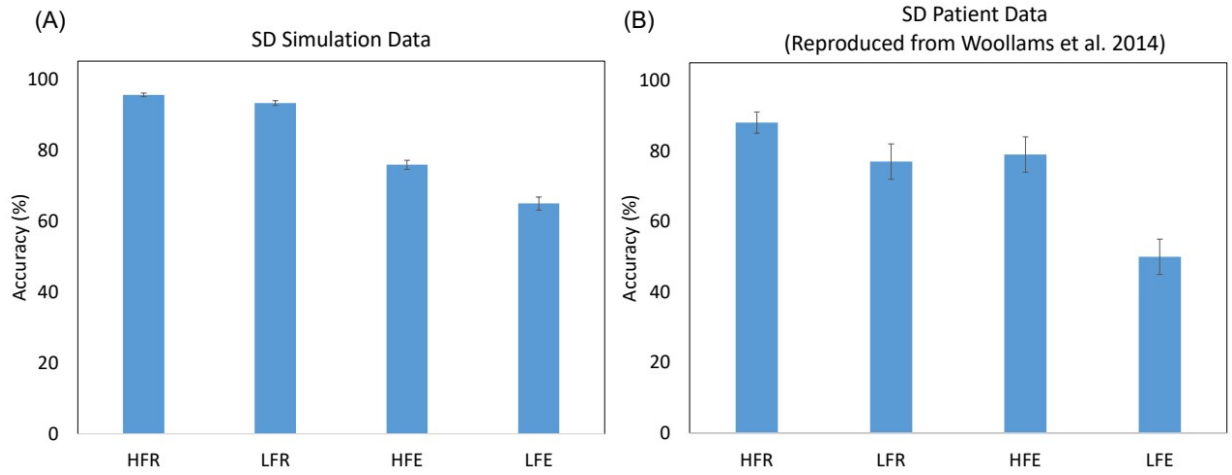

Figure 5. The frequency and consistency effect produced by (A) the SD model and (B) the SD patient data reproduced from Woollams et al. (2014). HFR: High Frequency Regular; LFR: Low Frequency Regular; HFE: High Frequency Exception; LFE: Low Frequency Exception.

Table 1. *Descriptive results of the intact (undamaged) and the damaged models on the three reading tests in word naming*

	Intact model	PA model	PD model	SD model
		Frequency & Consistency (Taraban & McClallend, 1987)		
High Frequency/ Consistent	100 (0)	99.9 (0.1)	99.5 (0.1)	95.0 (1.0)
High Frequency/ Inconsistent	99 (0.5)	97.0 (0.6)	98.2 (0.5)	75.4 (2.2)
Low Frequency/ Consistent	100 (0)	99.1 (0.3)	94.6 (0.7)	92.7 (1.2)
Low Frequency/ Inconsistent	98.5 (0.5)	91.5 (1.5)	83.1 (1.5)	61.0 (2.6)
		Word Length (Chang, 2012)		
WL3	0.0017	0.0007	0.0114	0.002
WL5	0.002	0.0032	0.0164	0.003
WL7	0.0039	0.0059	0.0344	0.013
		Nonword (Glushko, 1979)		
Nonword	92.6 (0.96)	77.0 (1.41)	57.3 (2.0)	65.7 (1.3)

Note: Means and standard errors of variables in brackets. Word naming data on frequency and consistency was based on accuracy rates while the data on word length was based on error scores.

Table 2. *Statistical results of the intact and the damaged models on the two reading tests in word naming*

	Intact	PA model	PD model	SD model
Frequency & Consistency (Taraban & McClallend, 1987)				
Frequency, F(1, 19)	*n.s.*	24.3 $p < .001$	298.8 $p < .001$	12.3 $p < .001$
Consistency, F(1, 19)	11 $p < .01$	56.4 $p < .001$	67.7 $p < .001$	215.5 $p < .001$
Interaction, F(1, 19)	*n.s.*	9.5 $p < .01$	30.2 $p < .001$	1.8 *n.s.*
Word Length (Chang, 2012)				
Word length F(2,	13.4	18.9	42	68.22

Note: *n.s.*: non-significant. Word naming data on frequency and consistency was based on the empirical logit transformation of accuracy rates while the data on word length was based on error scores.

The simulation results reported thus far confirmed that the damaged models were able to simulate the corresponding behavioural reading patterns as observed in patients with acquired dyslexia. It remained unclear whether these effects were different across the models: i.e., as expected by the Primary System Hypothesis, were the paradigmatic reading symptoms of each acquired dyslexia related to the location of damage? To investigate this issue, four sets of model data including intact, PA, PD and SD were entered into a series of ANOVAs, with model type (Intact, PA, PD, and SD) as a between-group variable.

For the frequency and consistency task, all three patient models demonstrated a significant frequency effect while the intact model did not. The combined ANOVA showed a significant difference in the frequency effect across the models, $F(3, 190) = 19.29$, $p < 0.001$. As illustrated in

Figure 6A, the effect was the relatively strong for the PD and the SD models compared to the PA and the intact models. In addition, all the models were sensitive to consistency. The combined ANOVA showed a significant difference in the consistency effect across the models, $F(3, 190) = 57.55$, $p < 0.001$. Critically, Figure 6B shows that the SD model had the strongest consistency effect, followed by the PD model, then the PA model, and the intact model.

For the word-length effect, all the models demonstrated a significant effect, $ps < 0.001$. To directly compare the slopes of the word-length effect across the models, we normalised error scores by the value obtained from the 3-letter length condition for the combined ANOVA analysis. Figure 7A showed a significant interaction pattern between word-length and model type, $F(2, 152) = 3.6$, $p < .01$, in which the performance of the PA model was the most strongly modulated by word length. Interestingly, the SD model also showed some length effects which have been reported in previous neuropsychological examinations of SD patients (Cumming, Patterson, Verfaellie & Graham, 2006).

For nonword reading, the PD model could only pronounce 57.3% of nonwords correctly, which was much worse than that of the intact model 92.6%, the PA model 77% and the SD model 65.7%, as illustrated in Figure

7B, and the differences were significant, $F(3, 57) = 121.4$, $p < .001$.

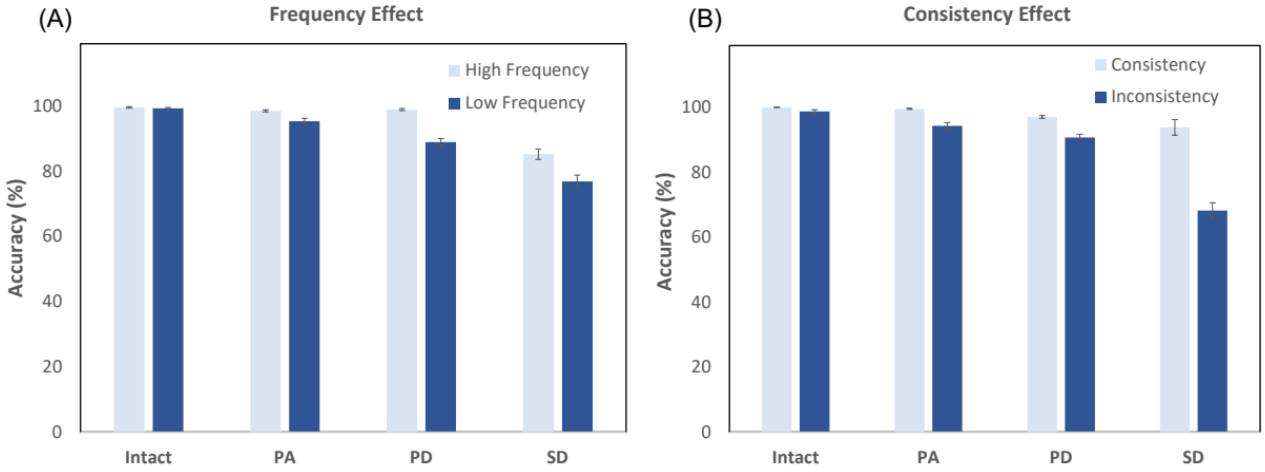

Figure 6. The effects of the frequency (A) and consistency (B) produced by the intact, PA, PD, and SD models in word naming. Error bars represent ± 1SEM.

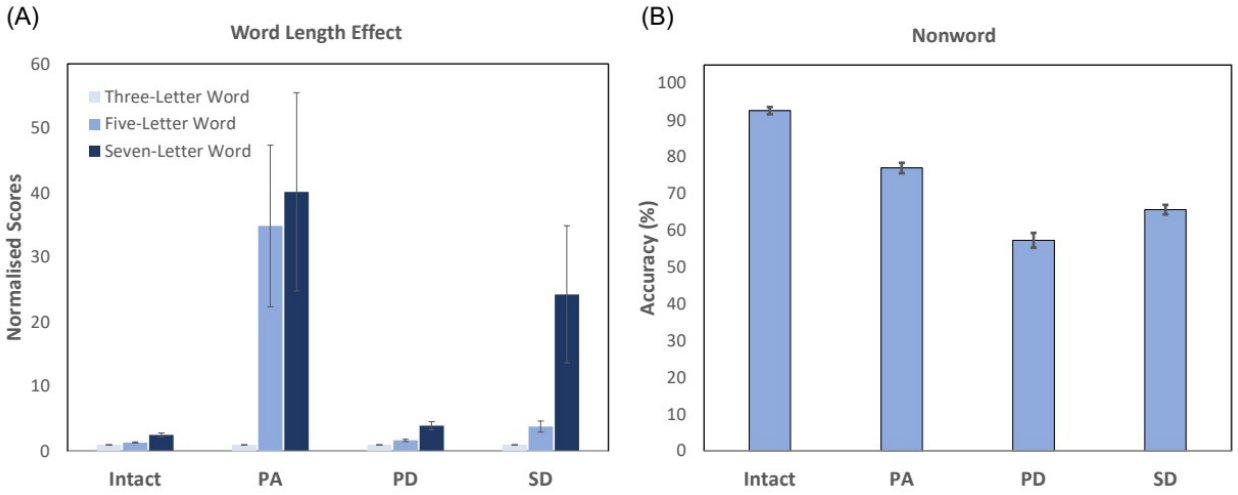

Figure 7. The effect of the word length (A) in word naming and the performance of nonword reading (B) produced by the intact, PA, PD, and SD models. Error bars represent ± 1SEM.

General Discussion

The primary aim of this paper was to simultaneously simulate multiple acquired dyslexia within a single reading framework. Building on the existing modelling work, our simulation results went beyond to demonstrate that a “deep”, multi-layer triangle model of reading with an additional visual

processing component (with ‘raw’ visual input of words and nonwords) was able to achieve good performance on a range of standard reading effects including frequency and consistency effects, and word-length effects in word naming, and nonword reading. Critically for testing the Primary Systems Hypothesis, when a specific location was damaged (visual-orthographic, phonological or semantic), the model produced the key diagnostic reading behaviours as observed in patients (Roberts et al. 2013; Welbourne & Lambon Ralph, 2007; Woollams et al. 2014) who have the corresponding functional impairment. Together, the simulation results demonstrated that both typical and impaired reading (i.e., pure alexia, phonological dyslexia, and surface dyslexia) could be simulated in a fully implemented triangle model of reading, consistent with the primary systems hypothesis (Patterson & Lambon Ralph, 1999).

Most previous computational models of reading based on triangle modelling framework have been applied to address typical and atypical reading separately. While these models have been successful in addressing reading effects relevant to phonological and semantic processing, they are relatively silent on the role of visual-orthographic processing in reading. Because reading starts from visual processing of printed words, the lack of a visual system in past reading models prevents them from addressing reading effects associated with visual-orthographic processing and its disorders. The present model went beyond previous work by implementing a visual system that allowed the model to learn mappings between vision, phonology, and semantics, in which internal visual (‘orthographic’) representations emerged as a consequence of learning. The implementation

of the visual system also enabled the model to be sensitive to letter length in words as in human readers. By contrast, if the model started the processes with pre-defined orthographic representations, it would not be able to properly address length-related effects as is evident in previous simulation work (Chang et al. 2012a; Chang, Monaghan, & Welbourne, 2019).

Another important key advantage of the present model with a visual processing component along with phonological and semantic processing components is that it provides an opportunity for the model to simulate neurologically-impaired behavioural patterns observed in both central dyslexia and peripheral dyslexia. The Primary Systems Hypothesis (Patterson & Lambon Ralph, 1999) proposes that damage to different generalised (i.e., not reading specific) systems (vision, phonology, semantics) are the core cause of the corresponding types of acquired dyslexic patterns. This is exactly what we observed in the simulation results. The simulations of pure alexia demonstrated that, following damage to the visual system, the PA model was strongly sensitive to word length in word naming, consistent with the key diagnostic symptom of the patients with pure alexia (Arguin et al., 2002; Behrmann et al., 1998; Fiset, Arguin, & McCabe, 2006; Roberts et al., 2010, 2013). In addition, relative to the intact model, the performance of the PA model was more sensitive to frequency. This is consistent with the partial activation account (Behrmann et al., 1998) suggesting that the reading performance of the PA patients may still be supported by the partially activated reading system.

The model with damage to the phonological processing layer produced poor nonword reading performance while it was better able to read words. This is in accordance with the hallmark feature of patients with phonological dyslexia (Beauvois & Dérouesné, 1979; Patterson & Kay, 1982; Welbourne & Lambon Ralph, 2007). Lastly, the simulations of semantic dementia demonstrated strong frequency and consistency effects in reading, consistent with the findings in the SD patients (Jefferies et al., 2004; Patterson et al., 2006; Woollams et al., 2014). These results provide further support to the view of a strong association between semantic dementia and surface dyslexia (Woollams et al., 2007).

In addition to investigating whether the damaged model could simulate the diagnostic symptoms of different types of acquired dyslexia, we also compared these behavioural effects across the damaged models. Although such comparisons are not commonly done in the neuropsychological studies where each patient group tends to be reported singly, it can potentially provide useful insights into the associated processing with functions. For example, as shown in Figure 6A, all the damaged models showed a frequency effect but to a different degree (i.e., the PD model ≧ the SD model > the PA model). It could suggest that the functional locus of the frequency effect is more associated with both phonological and semantic processing than visual-orthographic processing. Similarly, although nonword reading is not a key diagnostic test for PA and SD, from Figure 7B, we could see that damage also had different impact on performance. Thus, the outcome from the comparisons across the damaged

model can be seen as modelling prediction for future behavioural investigations.

Limitations and future directions

The present model has demonstrated that both typical and impaired reading could be simulated within the same modelling framework. There are, however, some limitations to this model. First, for good interpretability, the implementation of visual processing in the present model was deliberately designed to be simple compared with much more complex approaches used in the field of computer vision. For instance, VGGNet (Simonyan, & Zisserman, 2015) has 16-19 deep layers and is trained with about 1.3 million images. Such a complex model can generally achieve very high accuracy on object recognition; however, the model is difficult to interpret especially with respect to the function of the layers in the model. Moreover, for learning efficiency and performance, at the output layers, most complex models use localist one-hot encoding[4] instead of more biologically plausible distributed representations. Although the visual system in the present model was simple, the visual input is flexible and could be readily extended to learn different types of visual stimuli such as objects and faces, and different spatial transformations. This would be an interesting direction to explore in the future. Secondly, the model has simulated a specific set of reading effects in word naming, which were selected for capturing the defining key symptoms of patients with three different types of acquired dyslexia. We note though that the research in

[44] For localist one-hot encoding, the dimension of the representational vector exactly matches the number of training exemplars, with one for each.

word reading is vast so the specific tasks chosen here are neither comprehensive nor exhaustive. However, the key processing components involved in reading have been implemented in the model. Thus, future research can extend the present model to simulate various tasks of interest in word reading especially for the effects related to orthographic processing. Thirdly, whilst each of these three acquired dyslexia are distinct from each other, there are some individual variations within each type. Future research can explore the sources of these individual variations. There are at least three types of sources. The first is probably the most dominant and the one that we have explored here, namely the level/severity of impairment to each of the primary systems - this is simulated in the model by the level of damage to each primary system and is supported by the patient literature which has shown that each type of acquired dyslexia is related to the level of impairment to each of the primary systems (e.g. semantic impairment→surface dyslexia; phonological impairment→phonological-deep dyslexia; and visual impairment→pure alexia) (Behrmann et al., 1998; Crisp & Lambon Ralph, 2006; Rice et al., 2021; Roberts et al., 2010; Woollams et al., 2007). The second might relate to individual differences in the partial yet variable recovery that patients make after acute brain damage – which may, in turn, relate to both neural and environmental factors. Progress in this area will require future large-scale longitudinal studies of reading during this recovery process alongside simulations of partial recovery in the models (Welbourne et al. 2011; Chang & Lambon Ralph, 2020). Thirdly and finally, there are possible sources of premorbid individual differences that influence the pattern of acquired

dyslexia after brain damage. Numerous studies have shown individuals differ with regard to reading experience and vocabulary knowledge as a consequence of great variations in reading effects of skilled readers (Adelman, Sabatos-DeVito, Marquis, & Estes, 2014; Andrews & Hersch, 2010; Davies, Arnell, Birchenough, Grimmond, & Houlson, 2017; Yap, Balota, Sibley, & Ratcliff, 2012). However, by definition, premorbid individual differences are hard to study because it is not possible to roll back time and assess the patients' reading system prior to the brain damage. Previous research has used other methods to explore this possibility, specifically on the relationship of individual differences in the level of semantic involvement in reading aloud. Thus very large-scale studies of semantic dementia (Woollams et al. 2007) have shown that there is a strong coupling of semantic impairment to surface dyslexia but that this is modulated by individual differences such that some patients' reading is somewhat better or worse than the group pattern – which implies that their premorbid system may have been differentially weighted with respect to the contribution of semantics. This idea was explored and supported in subsequent investigations of healthy readers using fMRI and rTMS (Hoffman et al. 2015; Woollams et al. 2017). Thus, the level of engagement by the semantic hub in the anterior temporal lobe (ATL) (which is the centre of atrophy in semantic dementia) varied across participants and correct predicted the level of transient reading impairment following ATL rTMS.

Conclusion

To conclude, we developed the complete triangle model of reading incorporating vision, orthography, phonology, and semantics to validate the

primary systems hypothesis of reading. The simulation results demonstrated for the first time that both typical and neurologically-impaired reading including pure alexia, phonological dyslexia, and surface dyslexia could be simultaneously simulated within the same triangle model of reading.

Data Availability

The modelling data can be downloaded from the GitHub using the following link:

https://github.com/yaningchang/Chang_Welbourne_Furber_LambonRalph_Reading_Paper.git

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