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Semantic priming in deep dyslexia: Investigating the integrity of the semantic system.

Bruce A. King
University of Windsor

SEMANTIC PRIMING IN DEEP DYSLEXIA:

INVESTIGATING THE INTEGRITY OF THE SEMANTIC SYSTEM

by

Bruce A. King

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ABSTRACT

In contrast to the orthodox position, failure of inhibition theory (Buchanan, McEwen, Westbury, & Libben, 2003) posits an intact semantic system in deep dyslexia. An alternate logically possible model consistent with the extant empirical data that incorporates a functional impairment within the semantic network is proposed. The present findings of deep dyslexic direct and mediated semantic priming effects falling within the normative range failed to support this alternate account's central tenet of compromised implicit semantic access in deep dyslexia. The results of the current investigation are used to frame an argument that failure of inhibition theory, operating upon the parsimonious assumption that the functional aetiology of deep dyslexia is confined to a selection impairment within the phonological output lexicon, can accommodate not only those aspects of the syndrome traditionally attributed to semantic system impairment, namely the concreteness and part-of-speech effects, but also the often overlooked deep dyslexic reading comprehension deficit.

In memory of my father

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CHAPTER I

INTRODUCTION

The act of reading entails the use of a complex, uniquely human skill set, requiring the extraction of orthographic information from a word's written form, transformation of that information into a mental representation, and subsequent derivation of the word's pronunciation and meaning. Many investigators have attempted to gain a better understanding of the mechanisms that underlie these processes by studying individuals who acquire a reading disorder as a result of brain pathology. Indeed, in the last thirty years there has been a substantial and productive interaction between the study of patients with acquired reading deficits and the development, evaluation, and refinement of information-processing models of the functional architecture supporting normal word recognition and production.

Several different forms of acquired reading impairment (or dyslexia) have been delineated. A primary distinction is made between *peripheral dyslexias*, in which the patients' ability to analyse the visual attributes of written words is affected, and *central dyslexias*, in which there is impairment at later stages of word processing (Shallice & Warrington, 1980). The peripheral dyslexias include spelling dyslexia, dyslexia consequent upon neglect, and attentional dyslexia.

In *spelling dyslexia* or *pure alexia*, patients read or attempt to read letter by letter. The term pure alexia refers to the fact that individuals with this disorder often have preserved writing ability despite their impaired reading (Jackson & Coltheart, 2001). In those cases whose letter naming is intact their residual reading capacities are typically mediated by a strategy of spelling each stimulus word aloud with subsequent

reconstruction; for example, a patient may say, "b-o-o-k spells 'book'" (hence the term spelling dyslexia, McCarthy & Warrington, 1990). As a result of this slow and inefficient letter-by-letter procedure the time taken to read a word grows monotonically as the length of the word increases. The anatomic basis of pure alexia has been extensively investigated. The disorder is usually associated with a lesion of the left occipital lobe, invariably accompanied by damage to white matter tracts such as the splenium of the corpus callosum or the forceps major (Behrmann, 1999; Coslett, 2000).

Neglect dyslexia is characterized by a failure to explicitly identify the initial (left neglect) or terminal (right neglect) portion of letter strings (typically the former). Errors may result from the omission (e.g., FEVER → "*ever*"), addition (e.g., RAIN → "*brain*") or substitution (e.g., DREAM → "*cream*") of letters (Ellis, Flude, & Young, 1987).[1] In all cases damage to the parietal lobe has been documented (McCarthy & Warrington, 1990). In patients who have suffered lesions in the right cerebral hemisphere, neglect dyslexia involves the contralateral side of the word and is always observed in the context of a more general left visuospatial neglect syndrome affecting a wide range of stimuli in addition to words. In contrast, in patients with lesions in the left hemisphere, neglect dyslexia may affect letters in either the contralateral or ipsilateral space, and may occur in isolation, with no additional signs of visuospatial neglect (Berndt, Haendiges, & Mitchum, 2005; Cubelli & Beschin, 2005).

Only a handful of cases of *attentional dyslexia* have been identified. The primary feature of this disorder is a deficit in the recognition of visual stimuli when more than one

[1] The following notational conventions are used in this paper. Printed stimuli are shown in small capital letters with oral responses given in italics enclosed in double quotation marks. The phonological forms of letters and letter clusters are coded in International Phonetic Alphabet notation between slashes. Bracketed regular type is employed to designate mental representations, such as semantic concepts. Pictorial representations of words are denoted in regular type within single quotation marks.

stimulus of the same type is present in the visual field. Thus, letters can be read in isolation but not when presented in a row. At a higher level, single words can be read in isolation but not when flanked by other words that have to be ignored (Warrington, Cipolotti, & McNeil, 1993). Patients with attentional dyslexia also exhibit difficulties identifying the constituent letters of words, even though the words themselves are read correctly (Parkin, 1996), and tend to make letter migration errors when pairs of words are presented (e.g., LED BIT → "*bed lit*", Mayall & Humphreys, 2002). All cases of this syndrome to date have had lesions located in the left parietal cortex (Shallice & Warrington, 1977; Warrington, Cipolotti, & McNeil, 1993; Mayall & Humphreys, 2002). However, the critical anatomical regions involved remain unknown.[2]

As noted above, the central dyslexic disorders are believed to result from dysfunctional processing operations which follow the initial visual analysis of a printed word. Three main subtypes of central dyslexia have been identified: surface dyslexia, phonological dyslexia, and deep dyslexia.

Individuals with *surface dyslexia* are unable to employ whole word (or lexical) information to retrieve pronunciation. Rather, aloud reading occurs via the application of preserved rules for print-to-sound correspondences (Marshall & Newcombe, 1973). Consequently, surface dyslexics can read aloud *regular words* (i.e., words which obey the predominant spelling-to-sound mappings of a language's orthography, such as GAVE, SAVE, and WAVE) and *nonwords* (i.e., pronounceable nonsense letter strings, such as FRIP) with near normal accuracy. However, these patients exhibit an inability to correctly pronounce *exception words* given the atypical grapheme-to-phoneme correspondences of

[2] A discussion of the various theoretical accounts of the peripheral dyslexias is beyond the scope of this paper. For reviews, see Parkin (1996) and Coslett (2000).

these letter strings (e.g., HAVE). The Procrustean use of rules for common pronunciations by surface dyslexics results in their regularisation of exception words (Buchanan, Hildebrandt, & MacKinnon, 1999). For example, PINT is read aloud with the short /i/ of HINT; the vowel digraph of BROAD is realized as /ou/, its normal pronunciation in words such as LOAD and TOAD (Marshall, 1984). In their review of the anatomic correlates of seven cases of surface dyslexia Vanier and Caplan (1985) tentatively concluded that, although there was significant variance in the precise locus of the lesions, "involvement of posterior structures, especially temporal lobe structures is necessary" (p. 521).

First described by Beauvois and Dérouesné (1979), *phonological dyslexia* appears to be attributable to a selective deficit in the sublexical procedure mediating grapheme-to-phoneme translation with reading achieved exclusively via a whole-word mechanism. Thus, phonological dyslexics typically pronounce regular and exception words with equal facility but display a substantial impairment in the oral reading of nonwords (Buchanan et al., 1999; Coslett, 2000). Phonological dyslexia has been observed in association with lesions in a number of locations in the left perisylvian cortex and, on occasion, with lesions of the right hemisphere. In the majority of patients with this disorder prominent damage to the superior temporal lobe and the angular and supramarginal gyri is found (Coslett, 2000).

The final subtype in the taxonomy of acquired central reading impairments is *deep dyslexia*. Patients with this disorder resemble phonological dyslexics in that they too are unable to read nonwords aloud. However, the reading profile of deep dyslexics is also characterized by impaired naming of words (Marshall & Newcombe, 1973). *Paralexias* or aloud reading errors observed in deep dyslexia include visual errors,

derivational errors, and most importantly, in terms of classification, semantic errors (see discussion below; Buchanan et al., 1999). The disorder is typically associated with large lesions in the dominant perisylvian cortex extending into the frontal lobe. Supramarginal gyrus involvement is observed in the majority of cases (Marin, 1980; Coslett, 2000).

The term "deep dyslexia" reflects Marshall and Newcombe's (1973) original conceptualisation of the disorder as partially consequent upon an impairment within a component of the reading system more central than either orthography or phonology, namely semantics (Buchanan et al., 1999). Given that the functional integrity of the semantic system in deep dyslexia remains an issue of contention across competing theoretical models, this thesis focuses upon this syndrome of reading impairment with the particular aim of investigating the possibility of compromised semantic processing at the implicit level as a proximal cause of the disorder. Specifically, the hypothesis of spurious activation of conceptual representations within the semantic system secondary to attenuation of inhibitory connections will be empirically tested.

CHAPTER II

LITERATURE REVIEW

The Symptom-Complex of Deep Dyslexia

Given that the majority of individuals with deep dyslexia exhibit a relatively homogeneous set of co-occurring deficits during word naming, the disorder is considered a syndrome (Coltheart, 1980a; Coltheart, Patterson, & Marshall, 1987).[3] The production of *semantic paralexias* during oral reading of single words is the *sine qua non* of the deep dyslexic symptom-complex. Semantic paralexias are defined as incorrect naming responses related to the target stimulus only in terms of meaning (e.g., HEART → "*blood*"). In addition to semantic errors, deep dyslexic patients typically have several other manifestations of reading impairment. These include an inability to read aloud orthographically legal nonwords and the production of *visual paralexias* (responses that share at least 50 percent of the letters in the target stimulus, e.g., MOUTH → "*month*"), *derivational* or *morphological paralexias* (responses that differ from targets in terms of a bound morpheme, e.g., SLEEP → "*sleeping*"), and *function-word substitutions* (e.g., FOR → "*and*") in single-word reading. Patients also invariably demonstrate greater difficulties naming abstract words (e.g., DESTINY) as compared with high-imageability words with concrete referents (e.g., DOG), a word class effect whereby open-class words (e.g., nouns, verbs, etc.) are read with greater ease than functors (e.g., prepositions, pronouns, conjunctions, interrogatives, etc.), and a syntactic category effect such that nouns are read more reliably than modifiers (adjectives and adverbs), which are, in turn, read more accurately than verbs (Coltheart, 1980a).

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[3] The syndrome classification is not without contention. For discussion, see Plaut & Shallice (1993).

"Orthodox" Models of Deep Dyslexia

Following the seminal work of Marshall and Newcombe (1973), several models have been developed in an attempt to account for the distinct oral reading pattern observed in deep dyslexia. Buchanan and colleagues (Buchanan et al., 1999) note that, although these theoretical accounts differ in many respects, they are all share the assumption that a uniform loss of sublexical phonological processing capacity (i.e., the ability to map letters and letter clusters to their corresponding sounds) represents a core deficit in the deep dyslexic reading system (e.g., Coltheart, 1980b; Glosser & Friedman, 1990; Morton & Patterson, 1980; Plaut & Shallice, 1993).

In this section several of the most prominent models of deep dyslexia incorporating the hypothesized inability to process subword phonology will be briefly reviewed.

The Morton and Patterson Account

A number of different cognitive models of normal reading have been proposed (for a review, see Besner, 1999). According to the *dual-route theory* (e.g., Beaton, 2004; Jackson & Coltheart, 2001) successful visual word recognition and production is achieved via a reading system in which there are two routes from print to speech: the *assembled* and *addressed routines* (see Figure 1). Only when both of these procedures are intact is an individual able to read all forms of text correctly.

The assembled or sublexical routine (pathway A) identifies subword orthographic segments (graphemes), activates their corresponding subword phonological segments (phonemes), and combines these to produce a complete phonological code, i.e., a pronunciation for the letter string. Since this grapheme-phoneme conversion route is

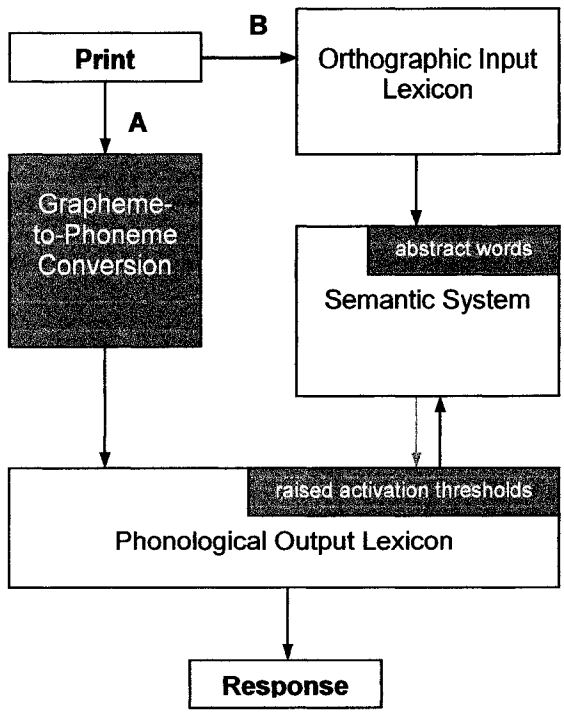

Figure 1. A version of the dual-route model of reading. Shaded regions indicate loci of impairment in the Morton and Patterson (1980) account of deep dyslexia. (Adapted from Buchanan et al., 1999).

rule-based, it provides correct pronunciations for both words and nonwords that feature typical spelling-sound correspondences. Exception words (e.g., COLONEL, YACHT, etc.), which do not conform to these correspondences, cannot be read via this pathway.

The addressed or lexical routine (pathway B) is comprised of pathways linking two *lexicons* (i.e., mental dictionaries) via the semantic system. The *orthographic input lexicon* contains distinct written symbol descriptions for each word in the reader's sight vocabulary. The *semantic system* holds representations for the meanings of words, while the *phonological output lexicon* consists of specifications for the sound structure of letter strings. It is here that selection of a particular entry supports the production of an oral response. Employment of the addressed routine entails visual recognition of the physical characteristics of print and subsequent activation of whole-word representations in the

orthographic input lexicon, allowing for the reading of both regular and exception words. However, since the existence of a particular representation in the orthographic input lexicon is contingent upon previous exposure to the corresponding letter string, this routine cannot be used to read aloud nonwords (Buchanan et al., 1999). Thus, in this pathway, visual analysis of a familiar printed word leads to activation of an orthographic representation, which accesses or "addresses" the corresponding node in the semantic system that, in turn, activates the appropriate phonological representation in the phonological output lexicon.

Operating within the dual-route framework, Morton and Patterson (1980) propose a multiple co-occurring deficit theory of deep dyslexia. According to this account, the deep dyslexic patient's inability to read aloud nonwords is secondary to the complete eradication of the assembled routine. Thus, it is assumed that individuals with the disorder are incapable of assembling phonology.

In order to accommodate the production of paralexic errors in deep dyslexia it is also postulated that some components of the addressed routine are damaged. Specifically, first, it is assumed that the semantic representations for particular abstract words are degraded. When the access codes for these words sent from the orthographic input lexicon to the semantic system are unable to activate semantic entries a second attempt of the addressed routine results in the closest orthographic neighbour of the target stimulus being selected as an eventual response. This accounts for visual paralexic errors. Semantic errors that are synonyms of the target word or that are made in response to targets that are highly abstract purportedly result from the inability of the semantic code operating on its own (due to the absence of the assembled reading route) to fully

specify the correct representation in the phonological output lexicon. Finally, in an attempt to provide an explanation for other forms of semantic paralexia observed in the syndrome, Morton and Patterson (1980) suggest that either representations for a set of words in the phonological lexicon have raised activation thresholds, leading to selection of the phonological entry nearest to threshold activation, or, alternatively, the transmission of semantic code to the phonological system is disrupted. The multiple loci of damage within the dual-route reading model posited by the Morton and Patterson (1980) account are indicated in Figure 1.

The Continuum Model

Glosser and Friedman (1990) contend that deep dyslexia represents the endpoint on a continuum of reading disability with less severe impairment of the reading system resulting in various forms of phonological dyslexia (see also Laine, Niemi, & Marttila, 1990; Sartori, Barry, & Job, 1984). The catalyst for the development of this continuum model were reports of several patients whose acquired reading disorders evolved with the passage of time from deep to phonological dyslexia. For example, Glosser and Friedman (1990) describe a closed head injury patient (GR) who at one month post-onset met the diagnostic criteria for deep dyslexia; his aloud reading performance was marked by a significant impairment of nonword reading capacity and semantic, orthographic, and derivational paralexic errors. Both a concreteness and part-of-speech effect were also evident. At 15 month follow-up, GR no longer produced semantic or morphological errors, and the concreteness and part-of-speech effects had disappeared. Nonword reading, however, remained impaired.

Like Morton and Patterson (1980), Glosser and Friedman (1990) assume that acquired dyslexia reflects the operation of a restricted subset of the processes that subserve normal reading. However, their continuum account of deep and phonological dyslexia is not formulated within the framework of dual-route theory but rather a lexical analogy model of word recognition (see Glushko, 1979; Henderson, 1982 and Kay & Marcel, 1981 for full descriptions of lexical analogy models). As in the dual-route model, the analogy model adopted by Friedman and colleagues (Friedman, 1996; Glosser & Friedman, 1990; Marchand & Friedman, 2005) contains a semantic and a non-semantic reading route. The non-semantic route, however, derives pronunciations for novel letter strings (i.e., nonwords and unknown words) by an analogical mapping of the subword orthographic units of these letter strings to orthographically similar lexical items contained within the semantic route. This is in contrast to the non-semantic pathway of the dual-route model in which, as noted earlier, systematic grapheme-phoneme correspondence rules are employed to assemble pronunciations (Buchanan et al., 1999).

Glosser and Friedman (1990) propound that phonological and deep dyslexic patients share one and sometimes two impairments in the lexical analogy reading system. Both forms of acquired dyslexia feature a disturbance of the direct orthography to phonology connections that constitute the non-semantic reading route, leading to disruption of oral nonword reading. Since bound morphemes and functor words are hypothesized to have semantic representations that are too "weak" to address a sufficiently specific phonological entry within the semantic reading route, this deficit in the non-semantic reading route is also assumed responsible for the production of derivational paralexias and impaired reading of closed-class words. A mild semantic

processing impairment within the semantic reading pathway in certain cases of phonological dyslexia results in concreteness and part-of-speech effects. A more significant deficit in semantic processing leads to the production of semantic paralexic errors in oral reading and, in turn, a deep dyslexia classification (Friedman, 1996; Glosser & Friedman, 1990).[4] According to this account, the evolution from deep to phonological dyslexia is secondary to gradual recovery of the semantic reading route and thus, recession and ultimately disappearance of semantic paralexias. The nonword reading impairment persists due to permanent damage to the phonological route (Friedman, 1996).

The Connectionist Approach: A Computational Model

Computational modelling of a cognitive activity entails the development and implementation of a computer program that simulates the activity in question utilizing formally specified processing mechanisms that hypothetically correspond to those employed in the human brain (Christiansen & Chater, 2001; Coltheart, 2006). *Connectionism* or *parallel distributed processing* is a particular form of computational modelling in which the architecture developed consists of a large number of densely interconnected neuron-like processing units or nodes (Farah, 2000). The intensity of excitatory or inhibitory signals sent from one node to another is contingent upon both the activation level of the transmitting node and the strength or "weight" of the internode connection. Each part of the network functions locally and in parallel with the other components such that at each moment during processing multiple nodes are simultaneously activated. Each representation (e.g., a word meaning) consists of the

[4] The functional dynamics of the impairment within the semantic system are not specified within the continuum model.

pattern of activation distributed over a specific computational node subpopulation (Horgan, 1997; Plunket 2001).[5] An important aspect of connectionist networks is their ability to learn from experience. This is achieved via systematic alteration of the weights between nodes so that given a set of input activation patterns during a training regimen the system ends up in the desired activation state (Farah, 2000; Horgan, 1997).

Shallice and colleagues (Hinton & Shallice, 1991; Plaut & Shallice, 1993) have attempted to model deep dyslexia via the introduction of lesions within various connectionist networks. Hinton and Shallice (1991) trained a connectionist architecture to produce semantic representations for a set of 40 words given their printed orthography as input. The grapheme-to-*sememe* (a term employed by Hinton and Shallice to refer to the distributed units or features of semantic representation) mapping in this network is executed with the aid of hidden units, with the sememes both interconnected and linked to a final layer of semantic representation that connects, recurrently, back to the sememe level. This pattern of connectivity within the semantic layers allows for a critical aspect of the Hinton and Shallice (1991) architecture; the capacity of the correct semantic representation of a word to serve as an *attractor*. Interactions among sememes results in a pattern of activated semantic nodes or features that resembles a word meaning known by the network to be gradually modified and, in turn, pulled toward the correct (i.e., known) semantic pattern over the course of settling. The region in the semantic space corresponding to the collection of initial sememe activation patterns that are drawn to a given attractor is termed a *basin* of attraction.

Hinton and Shallice (1991) reproduced the co-occurrence of semantic and visual

[5] The distributed units employed in connectionist systems lie in contrast to the holistic nodes of representation used in the dual-route model of reading.

errors in deep dyslexia by damaging the above network through the removal of certain connections or semantic units. This damage resulted in the outward expansion of attraction basins (see Figure 2) such that the initial activated sememe set would occasionally fall within a neighbouring basin, giving rise to an error response. These errors were often semantic in nature (e.g., [cat] → "*dog*") since the attractors of words with similar meanings lie in close proximity of each other in the modelled semantic space. The damage also led to visual errors (e.g., [bog] → "*dog*") since an inherent bias within the system resulted in words of similar orthography producing similar initial semantic patterns (Plaut, 1999).

Plaut and Shallice (1993) extended the work of Hinton and Shallice (1991) by implementing an architecture with a full semantic reading pathway, mapping orthography

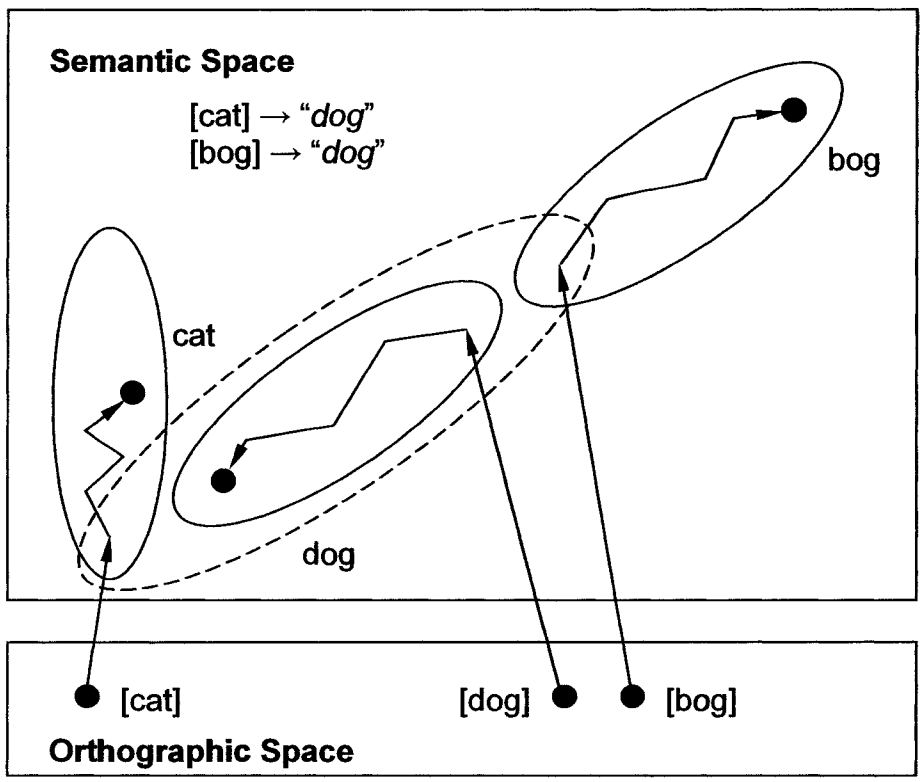

Figure 2. How damage to semantic attractors in the Hinton and Shallice (1991) connectionist model can give rise to semantic and visual errors. The solid ovals depict normal basins of attraction; the dotted oval depicts a basin following semantic damage (Adapted from Plaut, 1999).

to phonology via semantics. Introduction of a single lesion at various locations within this system (including the semantic layer) again produced reading impairments analogous to those observed in deep dyslexia. However, as noted by Buchanan et al. (1999), in a move reflecting tacit acceptance of the prevailing assumption that sublexical phonological processing is impossible for deep dyslexics, Plaut and Shallice (1993) did not implement a direct pathway from the grapheme unit system to the phonological output layer in any of the architectures tested. Consequently, none of the networks developed had the capacity to support nonword reading. Thus, to the extent that nonword reading was impossible, the architectures were already dyslexic prior to lesioning (Buchanan et al., 1999).

The Right Hemisphere Hypothesis

The theoretical models of deep dyslexia reviewed thus far all assume that the residual aloud reading ability of patients with the syndrome reflects the operation of a partially impaired normal left hemisphere reading system following damage or abolition of certain of this system's components. In contrast to these "subtractive" accounts, Coltheart's (1980b; 2000) right hemisphere hypothesis proposes that the characteristic oral reading impairments observed in deep dyslexia result instead from use of a subsidiary language-processing system of limited capacity located in the right hemisphere which is brought online following extensive damage to the primary reading system of the left hemisphere (see also Saffran, Bogyo, Schwartz, & Martin, 1980). According to this account, access from orthography to the left hemisphere lexicon is lost in deep dyslexia. Thus, initial word recognition is processed by an orthographic input lexicon in the intact right hemisphere. The orthographic entry activates a representation in the right

hemisphere semantic system which is subsequently transmitted to the left hemisphere. This information is then used to access an entry in the phonological output lexicon which, in turn, supports the retrieval and production of a pronunciation (Coltheart, 1980b; Coltheart, 2000).

Within this model, the right hemisphere is considered incapable of mapping sublexical orthographic to sublexical phonological units (Coltheart et al., 1987). This assembled phonological recoding process is viewed as the exclusive province of a left hemisphere mechanism abolished in deep dyslexia. Consequently, the deep dyslexic is unable to read aloud nonwords. To account for the concreteness effect in reading accuracy, Coltheart (1980b) proposes an inherent deficiency in the right hemisphere semantic system; it is assumed that the semantic representations of abstract words are selectively impoverished.

Notably, the right hemisphere hypothesis distinguishes two types of semantic paralexic error and posits a distinct explanation for each. *Shared-feature paralexias* are those in which the target word and the response share, as the name suggests, a subset of semantic features. These include synonyms and category super-, sub-, and co-ordinates (e.g., SEPULCHRE → "*tomb*", MUTTON → "*meat*"). *Associative paralexias* are linked to their target stimuli not by feature-overlap but rather by linguistic co-occurrence (e.g. NEXT → "*exit*", MERRY → "*Christmas*")(Coltheart, 1980b, 1980c). Shared-feature semantic errors are assumed to arise in deep dyslexic reading because a small degree of difference between the semantic representation dispatched from the right hemisphere and that selected for response in the left hemisphere lexicon is tolerated. The more specific (i.e., the lower in the semantic hierarchy) the disparate feature is, the more likely the

patient is to accept the mismatch (Coltheart, 1980b). In contrast, associative semantic paralexias are attributed to the incorporation of an associative network within the right hemisphere semantic lexicon such that when the entry of a target word is accessed spread of excitation activates representations of words that are contextually likely. This is followed by an incorrect choice from the set of associatively related candidate entries and subsequent interhemispheric transmission of that incorrect selection (Coltheart, 1980b, 1980c).

Support for the right hemisphere hypothesis comes from parallels drawn between deep dyslexic reading performance and that of the right hemisphere of both split-brain patients (i.e., individuals who have undergone callosal and anterior commissural section in order to confine foci of epilepsy; e.g., Zaidel & Peters, 1981) and neurologically intact readers (e.g., Ellis & Shepherd, 1974) as revealed via tachistoscopic studies. However, several authors (e.g., Patterson & Besner, 1984; Gazzaniga, 1983) have advanced arguments which call into question the validity of such comparisons and their interpretation by proponents of the right hemisphere model. Moreover, direct evidence against the hypothesis of right hemisphere mediated reading in deep dyslexia is provided by Roeltgen (1987) who reported the abolishment of residual reading capacity in a patient with deep dyslexia following a second left hemisphere stroke. More recently, two functional neuroimaging studies of deep dyslexic reading (Laine, Salmelin, Helenius, & Marttila, 2000; Price et al., 1998) have also yielded results incongruent with the right hemisphere hypothesis (but see Coltheart, 2000 for a response to Price et al., 1998).

Implicit Phonological Processing in Deep Dyslexia

As noted above, all the models reviewed are founded upon the assumption that a key impairment in deep dyslexia is an inability to derive phonological information from sublexical orthographic units. The basis for this position is the uniformly poor performance of deep dyslexic patients in aloud reading of nonwords, a task requiring explicit processing. However, dissociations of explicit and implicit processing have been demonstrated in several neurological disorders (e.g., amnesia in Warrington & Weiskrantz, 1970, and blindsight in Weiskrantz, 1989). Thus, Hildebrandt and Sokol (1993) contend "...that failure on explicit tasks does not necessarily imply that the targeted process is not occurring" (p. 47).

Echoing this view, Buchanan and colleagues (Buchanan, McEwen, Westbury, & Libben, 2003) have developed a framework for the study of word production deficits in patients with neurolinguistic compromise which formally incorporates the distinction between explicit and implicit processing; the *PEIR* model. According to the PEIR formulation, lexical *P*roduction depends on *E*xplicit access, which is contingent on *I*mplicit access, which, in turn, depends on intact *R*epresentations. At each level, three separate but interacting components of word representation and processing are delineated: phonology, morphology, and semantics. Within this model, *explicit access* is defined as "overt knowledge regarding relevant semantic, morphological, or phonological characteristics of words" (Colangelo & Buchanan, 2005, p. 39). Production errors during reading reflect impairment at the level of explicit access. *Implicit access* refers to sensitivity to semantic, morphological, and phonological lexical manipulations independent of explicit access and production (Colangelo & Buchanan, 2005). Thus,

implicit tasks are those that do not require a conscious awareness of the representations accessed (or produced) by the process under investigation (Hildebrandt & Sokol, 1993).

In line with the proposed dissociation between implicit and explicit phonological processing capacity embodied within the PEIR framework, several recent research findings indicate that deep dyslexics have normal sensitivity to lexical and sublexical phonological information when only implicit access is required, thereby challenging those conceptualizations of the disorder predicated on a complete inability to process subword phonology.

Hildebrandt and Sokol (1993) reported a normal *spelling regularity effect* for low-frequency words in the lexical decision data from a patient (GR) who fit the general deep dyslexic profile: GR made faster and more accurate lexical decisions to low-frequency words with typical spelling-sound correspondences (e.g., TILE) than to words of low frequency with irregular spelling-sound correspondences (e.g., WAND). In the normal word recognition literature theorists operating within the dual-route model have generally interpreted the regularity effect as evidence for the contribution of sublexical phonological processing to word recognition and production (e.g., Waters & Seidenberg, 1985): In the case of regular words sublexical phonological information from the assembled routine coincides with the pronunciation derived from the lexical pathway leading to facilitation of word recognition. In contrast, in the case of exception words conflict between the pronunciations derived by the two reading routines is believed to impede processing.

Further evidence for intact subword phonological encoding in deep dyslexia comes from Buchanan, Hildebrandt, and MacKinnon (1994, 1996) who demonstrated in a

series of experiments with three deep dyslexic patients a normal *pseudohomophone effect*: Reaction times in lexical decision to reject pseudohomophones (i.e., nonwords that sound like words, e.g., TAYBUL) were slower than those required to reject nonword orthographic controls (e.g., TARBLE). According to dual-route theory, this elevation in response times for the former stimulus type results from the reading routines generating incongruent decisions regarding the lexical status of the pseudohomophone. Orthographic information from the pseudohomophonic nonword travels through the addressed routine (pathway B) which produces the correct "no" response. In contrast, activation based upon sublexical phonological information derived from the pseudohomophone via grapheme-phoneme conversion spreads through the assembled route (pathway A) and travels to the semantic system where it activates the representation corresponding to the phonologically identical word (e.g., TAYBUL activates the semantic node [table]), leading in turn to a "yes" response. Time is required to resolve the resultant conflict within the system and the pseudophomophone effect is produced (Buchanan et al., 1999). Buchanan and colleagues (Buchanan, Hildebrandt, & MacKinnon, 1994, 1996) also investigated whether a *pseudohomophone priming effect* (i.e., lower response latencies in lexical decision in response to a word, e.g., CHAIR, when preceded by a semantically related pseudohomophone prime, e.g., TAYBUL, than when presented after a orthographic control nonword, e.g., TARBLE) is evident in deep dyslexia since reports of this effect in neurologically intact readers have been taken as evidence for feedback from the sublexical phonological pathway to the semantic network (e.g., Lukatela & Turvey, 1991). In contrast to the null effect predicted by models of deep dyslexia that hinge upon the assumption of eradicated subword phonological processing,

all three of Buchanan et al's (1994, 1996) patients showed normal semantic priming with pseudohomophone primes in lexical decision.

It should be noted that the possibility of preserved implicit or automatic phonological knowledge in deep dyslexia was first proposed by Katz and Lanzoni (1992). These researchers reported a deep dyslexic patient (JA) who demonstrated, like normal subjects, a rhyming advantage in a paired-stimuli lexical decision task; JA showed faster reaction times on trials with rhyming, orthographically similar word pairs (e.g., BRIBE-TRIBE) relative to control trials (consisting of non-rhyming, dissimilarly spelled stimuli), but slower response times on trials with non-rhyming, orthographically similar stimuli (e.g., COUCH-TOUCH). Given that this effect was word class specific, being found for open-class but not function words, Katz and Lanzoni (1992) concluded that the underlying implicit phonological processing was taking place within the phonological output lexicon of the addressed routine.

The Selection Impairment Model

To accommodate the mounting evidence for preserved implicit assembled phonology in deep dyslexia, Buchanan, Hildebrandt, and MacKinnon (1994, 1999) developed an alternative left hemisphere model of the syndrome which accounts for the deep dyslexic reading pattern by positing a single locus of damage within the phonological output lexicon. This single lesion results in reduced sensitivity to activation levels of candidate representations and, in turn, a *selection impairment*.

Buchanan and colleagues' (1994, 1999) proposed model is based upon the dual-route framework described previously. However, as shown in Figure 3, a non-semantic lexical reading routine, i.e., a direct pathway from the orthographic input lexicon to the

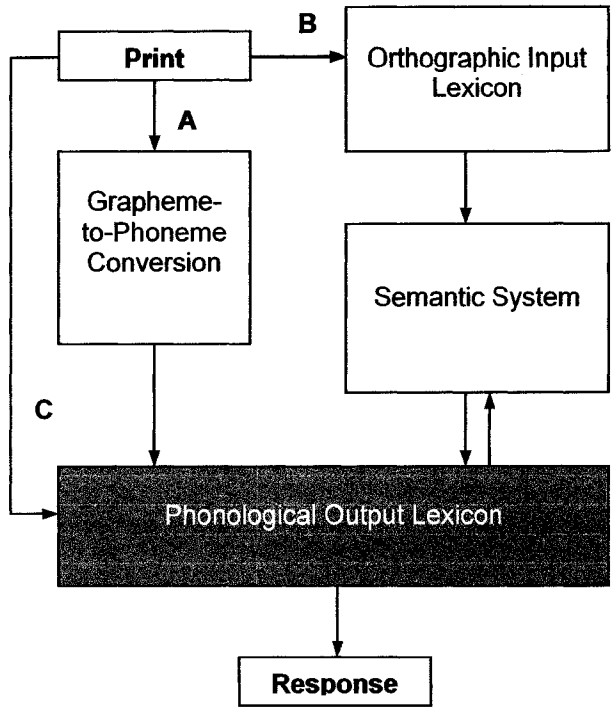

Figure 3. The Buchanan et al. (1994, 1999) dual-route model of reading. The shaded region indicates the locus of damage in deep dyslexia as proposed by the selection impairment account. (Adapted from Buchanan et al., 1999).

phonological output lexicon, is included in the reading system (pathway C).[6] In normal aloud reading of words activation spreads in cascade through all three routines. For example, upon presentation of the word DOG grapheme-to-phoneme conversion and assembly is initiated within pathway A, and the assembled representation activates the phonological entry associated with DOG in the phonological output lexicon. Activation then spreads within the phonological lexicon from the target representation to nodes corresponding to phonological neighbours such as [doll] and [bog]. At the same time, within pathway B activation spreads from the orthographic representation of DOG to the corresponding representation located in the semantic system. Here activation spreads automatically via a network of interconnections from the target concept node ([dog]) to

[6] For discussion of arguments for and against the existence of a non-semantic lexical pathway, see Buchanan and Besner (1993), and Rapp, Folk, and Tainturier (2001).

representations within the target's semantic neighbourhood, i.e., to representations that are semantically or associatively related (e.g., [cat], [bark], etc.). Activation from the target and several of its semantic neighbours is then fed forward from the semantic system to the phonological output lexicon. Finally, operation of pathway C leads to activation of the phonological representation in the phonological output lexicon corresponding to DOG and its orthographic neighbours. Thus, activated nodes within the phonological output system include the target and several phonological, semantic, and visual neighbours (Buchanan et al., 1994, 1999).

In a neurologically intact individual the most highly activated representation (i.e., the entry receiving input from the most sources) in the phonological output lexicon is selected for production. Buchanan et al. (1994, 1999) proposed that damage to this selection mechanism alone results in the aloud reading errors observed in deep dyslexia. For example, incorrect selection of an activated semantic neighbour of the target representation in the phonological output lexicon would result in a semantic paralexia (e.g., DOG → "*cat*"). According to this selection impairment model, the assembled routine is uncompromised in deep dyslexia to the extent that sublexical phonological descriptions can be derived from both words and nonwords, with this information influencing processing at the implicit level. The phonological deficits observed in the syndrome reflect production errors due to compromised explicit access.

Within this formulation, the deep dyslexic's inability to name nonwords is also attributed to the selection impairment in the phonological output system. In normal nonword naming the assembled phonology of the nonword is fed forward to the phonological output lexicon. Here activation spreads to the phonological neighbours of

the nonword. The pronunciation of the nonword specified by the grapheme-phoneme routine is then checked against those of the nonword's phonological neighbours during an analogical mapping procedure. Since the phonological output lexicon does not contain a lexical representation corresponding to the nonword this lexical check produces a "nonmatch" and the assembled phonology, as the "best guess" of the system, is selected for response (Buchanan et al., 1999).

In a patient with deep dyslexia the reduced sensitivity of the selection mechanism in the phonological lexicon decreases the efficiency of the analogical mapping process. Consequently, the time required to make comparisons with activated lexical candidates typically exceeds the time that the assembled pronunciation can be maintained in the phonological buffer and no response is made. Although the system generally "times out" before all activated lexical phonological neighbours can be mapped to the assembled phonology, on occasion the word that has received the most activation may be chosen and offered in response to the nonword (Buchanan et al., 1999; Colangelo, 2003).

A Revision: The Failure of Inhibition Model

Recently, Buchanan, McEwen, Westbury, and Libben (2003) further specified the selection impairment account of deep dyslexia. According to this revised formulation, selection impairment in the phonological output lexicon results from a failure of inhibition; attenuated inhibitory connections within the phonological output lexicon fail to prune activated non-target candidate representations, which, in turn, remain available for selection. Operating within the PEIR framework (see above review), Buchanan et al. (2003) contend that inhibition failure within the phonological output system does not

disrupt implicit access to representations but rather leads to compromised explicit access and production.

Support for the Failure of Inhibition Account

A general prediction that evolves from the failure of inhibition model is that any manipulation that affects the number of activated candidates in the phonological output lexicon should have an impact on reading performance in patients with deep dyslexia. Thus, the provision of phonemic cues during aloud reading should enhance patient performance by raising the activation level of candidates with corresponding phonology and thereby reducing the number of potential candidates for selection. In contrast, supplying miscues based on the initial phonemes of semantically related candidates should function to decrease patient performance. Consistent with these hypotheses, providing the first phoneme of target words has been shown to significantly aid deep dyslexic oral reading performance (Katz & Lanzoni, 1997; Buchanan, Kiss, & Burgess, 2000), whereas miscueing leads to poorer performance (Katz & Lanzoni, 1997).

Further support for inhibition failure as the mechanism of selection impairment in deep dyslexia comes from an oral reading study in which *compound words* (e.g., HONEYBEE) were employed as the target stimuli (Buchanan et al., 2003). If semantic paralexias result from a failure to inhibit activated candidates within a target's semantic neighbourhood in the phonological output lexicon, then increasing the number of semantic neighbourhoods associated with each target word should function to increase reading errors since more candidates are available for potential selection. Buchanan et al. (2003) tested this hypothesis by manipulating the transparency of the constituents of, and thereby the number of semantic neighbourhoods associated with, compound targets in a

reading task with a deep dyslexic patient (JO). The *transparency* of a constituent (e.g., HONEY in the compound HONEYBEE) refers to the extent to which its meaning corresponds to the meaning of the whole compound. A transparent constituent shares an obvious semantic relationship with the compound (e.g., JAIL in JAILBIRD). In contrast, no such relationship exists in the case of an opaque constituent (e.g., STRAW in STRAWBERRY). Buchanan et al. (2003) utilized four types of compound word as defined by constituent transparency: transparent-transparent (TT, e.g., FIREMAN), opaque-transparent (OT, e.g., POTHOLE), transparent-opaque (TO, e.g., SHOEHORN), and opaque-opaque (OO, e.g., DEADLINE).

Importantly, this transparency manipulation varied each compound word's number of independent semantic representations and, in turn, the number of distinct semantic neighbourhoods associated with each target. In the case of TT compound words, such as BATHROOM, there is a single semantic neighbourhood, because both constituents are semantically related to the compound and therefore both possess semantic neighbourhoods that overlap with that of the compound. In contrast, in OO compound words, such as HUMBUG, each constituent's semantic representation is independent of that of the compound. Consequently, fully opaque compound words are associated with three distinct semantic neighbourhoods; one for the compound and one completely separate neighbourhood for each constituent. Thus, the number of semantic errors that arise from the reading of OO compound words should be greater than for fully transparent compound targets. Finally, OT and TO compound words are both associated with two independent semantic neighbourhoods, one for the whole word and its transparent constituent and another for the opaque constituent. Therefore, when

presented as targets they should result in an intermediate level of aloud deep dyslexic reading performance. However, given that English compound words are *right-headed* (i.e., the second constituent, e.g., FLY in BUTTERFLY, carries more semantic load than does the first), OT compounds should be read with greater facility than TO compounds.

With these distinctions in mind, Buchanan and colleagues (Buchanan et al., 2003) hypothesized that a deep dyslexic patient's reading accuracy for the four types of compound target should reflect the transparency manipulation as follows: highest for fully transparent words, then opaque-transparent, then transparent-opaque, and lowest for fully opaque. In line with this prediction, when all words that were eventually read correctly were considered, JO's gradient of oral reading accuracy was: TT compounds, 40 percent correct; OT compounds, 33 percent correct; TO compounds, 23 percent correct; and OO compounds, 13 percent correct.

Performance patterns congruent with failure of inhibition model predictions have also been obtained in deep dyslexia in studies investigating the influence of neighbourhood size. The size of a lexical neighbourhood can be defined in terms of orthography (i.e., the number of real words that can be generated from the target word by replacing one letter at a time), phonology (i.e., the number of real words that can be generated from the target word by replacing one phoneme at a time), or semantics (i.e., the number of words that co-occur with the target in similar contexts in text) (Colangelo, 2003). Words with larger or denser neighbourhoods eventuate a larger number of potential candidates for selection in the phonological output lexicon. Therefore, within the deep dyslexic context of attenuated inhibitory connections, larger target word neighbourhood size is predicted to raise the probability of incorrect candidate selection

and, in turn, the number of paralexic errors. Indeed, in line with this expectation both semantic neighbourhood (Buchanan, Burgess, & Lund, 1996; Buchanan et al., 2000) and phonological neighbourhood (Buchanan et al., 2000) size have been found to be negatively correlated with deep dyslexic reading performance.

The Integrity of Semantics in Deep Dyslexia

The Nature of Semantic Memory: Evidence from Semantic Priming

Semantic memory is generally conceived of as an amodal system responsible for the storage and processing of overlearned world knowledge, as distinguished from autobiographical (or episodic) and procedural knowledge (Tulving, 1985).[7] Several models describe this system as a network of interconnected nodes where each node represents a concept (e.g., Anderson, 1983; Collins & Loftus, 1975). Links between nodes are established on the basis of both semantic relatedness and prior association in experience. Degree of semantic relatedness is determined by the number of semantic features shared by concepts with the greater the number of common features the greater the number of interconnections between the respective nodes (Collins & Loftus, 1975). Linguistic associative links are formed when the lexical referents of concepts either appear in similar contexts in large bodies of language use (global co-occurrence) or are regularly encountered in a temporally or spatially contiguous fashion in language (local co-occurrence; Buchanan, Westbury, & Burgess, 2001). Network models of semantic memory typically characterize the process leading to the retrieval of semantic

[7] Several theorists have argued in favour of multiple modality-specific semantic systems (e.g., Beauvois, 1982; McCarthy & Warrington, 1988). While a full discussion of this literature is beyond the scope of this thesis (for a review and counter-arguments, see Caramazza & Shelton, 1998), it is noted that the primary line of evidence cited in support of this position – different levels of patient semantic task performance across modalities/semantic-categories – can be accounted for within a unitary system model (Coccia, Bartolini, Luzzi, Provinciali, & Lambon Ralph, 2004).

information as one of activation of the relevant node(s) and invoke the concept of *automatic spreading activation* to explain a variety of experimental data (e.g., Anderson, 1983; Collins & Loftus, 1975). Within the spreading activation framework, activation is assumed to spread automatically from one strongly activated concept node to other semantically and associatively linked nodes, rendering the latter more accessible to subsequent processing operations (Dagenbach & Carr, 1994).[8]

Much of the evidence used to support the conceptualization of the semantic system as a multi-nodal network characterized by automatic spread of activation between representations comes from studies that have employed the *semantic priming paradigm* (see McNamara, 1992a, 1992b, 1994, and Neely, 1991, for reviews of the role of semantic priming studies in semantic memory model development). In general, this procedure entails asking participants to either read aloud or make lexical (i.e., "word" or "nonword") decisions to target letter strings. People have typically been found to respond faster and more accurately to a target word (e.g., BUTTER) when it is preceded by a semantically and/or associatively related prime word (e.g., BREAD) than when it is preceded by an unrelated prime (e.g., HOUSE). This consistently observed *semantic priming effect* is widely considered to reflect the operation of automatic spreading excitation within the semantic system: When the node representing a prime related to the target is activated by the presentation of the prime, activation spreads to the related node of the target. The residual activation accumulating at the target node facilitates

[8] With the exception of the connectionist account advanced by Shallice and colleagues (Hinton & Shallice, 1991; Plaut & Shallice, 1993), all of the deep dyslexia models discussed above adopt this automatic spreading activation view of the semantic system with concepts represented as holistic units or nodes. As discussed earlier, in the connectionist model the units of the semantic network do not represent whole concepts but rather simple semantic features with a specific pattern of weighted features representing a particular concept. In this framework retrieval of a concept such as DOG facilitates processing of related concepts, such as CAT, not because of spread of activation, but rather because the distributed set of semantic features (e.g., fur, four legs) that constitute the two concepts largely overlap.

recognition of the target word upon its presentation (Hutchison, 2003; McNamara, 1992b).

A key component of automatic spreading activation models of semantic memory is the assumption that when a concept node (e.g., [lion]) is activated, activation spreads not only to directly related concepts (e.g., [tiger]) but also to more distant concepts (e.g., [stripes]) that are several nodes or "steps" away in the network (e.g., Collins & Loftus, 1975). These models therefore predict *indirect* or *mediated semantic priming*, i.e., a benefit to the speed or accuracy of responding to a target word (e.g., STRIPES) when preceded by an indirectly related prime word (e.g., LION), as compared to responding to the same target preceded by an unrelated prime. Mediated priming is expected to occur because activation is assumed to spread from the prime concept ([lion]) through the directly related mediating node ([tiger]) to the target representation ([stripes]), thereby facilitating the response to the target word.

The extent of spreading activation within the semantic network was first systematically investigated by de Groot (1983). She conducted a series of experiments using the standard lexical decision task to examine priming for both directly and indirectly related word pairs. In contrast to the "multiple-step" spreading activation position, de Groot (1983) consistently found direct but not mediated priming. Balota and Lorch (1986) attributed de Groot's (1983) inability to demonstrate a priming effect for mediated word pairs to a particular strategy adopted by participants to aid performance in standard lexical decision. Specifically, they suggested that participants conduct a postlexical access check for a relation between the prime and the target and use the presence or absence of such a relation to bias a "word" or "nonword" response,

respectively. This *relatedness checking* or *semantic matching strategy* emerges because of an implicit assumption that words appear in meaningful context and because the detection of a relation indicates that the target must be a word. Postretrieval semantic matching should facilitate priming for directly related word pairs but impair responding to unrelated or mediated pairs since the absence of a direct relationship in the latter pairs biases a "nonword" response. Thus, it is possible that any facilitation that may have been present due to multiple-step spreading activation in de Groot's (1983) experiments was obscured by this relatedness checking procedure.

Following Balota and Lorch (1986), McNamara and Altarriba (1988) attempted to eliminate strategic processing in mediated priming experiments by varying either the list composition or the structure of the lexical decision task. These authors argued that mediated priming should occur in lexical decision if stimulus lists do not include directly related word pairs since it is the presentation of these items that sensitizes participants to the presence or absence of relations between primes and targets, which in turn leads to relatedness checking. In line with this prediction, McNamara and Altarriba (1988), along with several other research groups (e.g., McKoon & Ratcliff, 1992; Sayette, Hufford, & Thorson, 1996), have demonstrated significant mediated priming employing stimulus lists containing only mediated and unrelated word pairs.

McNamara and Altarriba (1988) also contended that use of a *continuous lexical decision procedure*, in which participants respond to every letter string presented, should also curtail postlexical relatedness checking and thereby create an experimental environment conducive to the detection of mediated priming effects. They reasoned that this procedure eliminates the effectiveness and, in turn, the use of semantic matching,

regardless of the presence or absence of directly related word pairs, since failure to detect a relation with the previous word provides no reliable information regarding the lexical status of the current letter string. Indeed, using the continuous lexical decision procedure, McNamara and Altarriba (1988) found significant priming effects for both direct and indirect conditions. Subsequently, several researchers who have adopted this procedure have found significant mediated priming (e.g., Bennett & McEvoy, 1999; McKoon & Ratcliff, 1992; McNamara, 1992b). Thus, collectively these results demonstrate that, when measures are taken to eliminate strategic relatedness checking, mediated priming consistently emerges across studies, thereby providing support for the operation of multiple-step spreading activation within the semantic network.[9]

Semantic Processing in Deep Dyslexia: The Failure of Inhibition Model Position and an Alternate Logically Possible Account

Notably, all of the traditional theoretical conceptualizations of deep dyslexia reviewed above posit, in addition to eradication of the assembled reading routine, a form of functional and/or structural impairment within the semantic system. In contrast, the failure of inhibition model assumes a fully intact semantic network, with a single deficit in the phonological output lexicon – a selection impairment secondary to reduced or slowed inhibitory connections – considered sufficient to accommodate the deep dyslexic oral reading pattern.

Evidence for the position of preserved semantic processing in deep dyslexia comes from several lines of investigation. First, Colangelo and colleagues (Colangelo, Stephenson, Westbury, & Buchanan, 2003) contended that if the semantic system is

[9] Given the absence of shared features between mediated word pairs, distributed models of semantic memory such as that proposed in the connectionist account of deep dyslexia are unable to accommodate mediated priming. For further discussion, see Hutchison (2003).

uncompromised in deep dyslexia then patients with the disorder should perform normally in an auditory word association task. Consistent with this hypothesis, when presented with a spoken cue and instructed to respond verbally with the first word that came to mind two deep dyslexic patients produced performances reflecting those observed in normal participants, with the majority of their responses found to be typical as gauged by comparison with entries within the Nelson, McEvoy, and Schreiber (1998) associative norms database. Second, Colangelo and Buchanan (2005) maintained that evidence in favour of intact implicit semantic access and, in turn, intact semantic representations in deep dyslexia is provided by the finding of more accurate lexical decisions for ambiguous words (i.e., words with two independent meanings, e.g., BANK) than for unambiguous words (e.g., FOOD); an effect suggesting normal facilitation for words with multiple representations. Finally, the observation of increased deep dyslexic semantic error production in oral reading for words blocked in semantic categories has been interpreted as indicative of intact ability to activate target neighbours in semantic memory (Colangelo, Buchanan, & Westbury, 2004).

Importantly, however, these findings cited in support of the integrity of the semantic system in deep dyslexia do not exclude an alternate logically possible model of the disorder that incorporates a functional impairment within semantic memory. This alternate left hemisphere account represents an extension of the failure of inhibition model in that it assumes intact implicit sublexical phonological processing capacity in deep dyslexia and identifies attenuation of inhibitory connections as the source of oral reading impairment. However, within the alternate model failure of inhibition is not confined to the phonological output lexicon but extends to the semantic network: The

normal automatic inhibitory mechanism that serves to dampen and constrain spread of activation within the semantic system, limiting it to relatively immediate neighbours of the target representation, is compromised. Following the resultant unfocused exaggerated spread of activation within the semantic network an abnormally large number of candidate representations are fed forward to the phonological output lexicon and are subsequently available for selection. Reduced inhibitory connections in the phonological lexicon in turn result in decreased sensitivity to the activation levels of neighbours and ultimately impaired selection.

For ease of reference, this alternate conceptualization of deep dyslexia is termed the *dual-component attenuated inhibition model*, a label reflecting its key tenet of inhibition failure in both the phonological output lexicon and the semantic system. It is important to note that, in contrast to the inhibitory connections in the phonological lexicon which subserve explicit access (i.e., selection), the inhibitory mechanism within the semantic network which the alternate model assumes compromised subserves implicit access. An inhibitory mechanism brought online automatically to dampen and focus spread of activation within the semantic system, and thereby increase the efficiency of normal implicit semantic access has been proposed by several authors (e.g., Chiarello, 1988; Kiefer, Ahlegian, & Spitzer, 2005). Support for Kiefer et al's (2005) prefrontally-mediated focusing mechanism has been obtained from both behavioural and electrophysiological data (Kiefer et al., 2005; Kiefer, Weisbrod, Kern, Maier, & Spitzer, 1998).

Notably, the dual-component attenuated inhibition model with its semantic impairment can accommodate all of the aforementioned findings presented by Colangelo

and colleagues as evidence in favour of fully preserved semantic processing in deep dyslexia. Both ease of production of semantic associates to verbally presented cues (Colangelo et al., 2003) and higher deep dyslexic accuracy in lexical decision for ambiguous than for unambiguous words (Colangelo & Buchanan, 2005) are also predicted by this alternate model since exaggerated spread of activation within the semantic system should aid both generation of semantic associates and activation of ambiguous word secondary semantics. Colangelo et al's (2004) argument for preserved ability to activate the semantic neighbours of a target representation in deep dyslexia is entirely consistent with the dual-component attenuated inhibition position since the deficit in semantics proposed by the latter results not in absent activation, but rather raised and more diffuse activation secondary to impairment of inhibitory processes.

Rationale and Predictions

Given that the extant data are not sufficient to effectively adjudicate between the failure of inhibition theory and the dual-component attenuated inhibition model with respect to their competing positions regarding the integrity of semantics, the aim of the current study is to empirically assay the tenability of the latter account's additional assumption of reduced inhibitory connections within the semantic system. Since, as discussed in the preceding section, automatic spreading activation to representations of related concepts within the semantic network is the standard explanatory construct employed to account for semantic priming in lexical decision, this paradigm represents a suitable vehicle for this investigation. If, as proposed by the alternate model, the inhibitory focusing mechanism within the semantic system is compromised in deep dyslexia, leading to exaggerated and greater spread of activation within semantic

neighbourhoods, one would anticipate both heightened direct and mediated priming in deep dyslexia vis-à-vis a neurologically intact control population (semantic priming operationalized as the difference score of mean response times between the priming condition and the respective control condition). In contrast, if the failure of inhibition model as delineated by Buchanan and colleagues (Buchanan et al., 2003), with its assumption of a fully intact semantic system, is correct then the finding of deep dyslexic semantic priming effects falling within the normative range should obtain. Thus, an assessment of the magnitude of direct and indirect semantic priming difference scores achieved by a patient with deep dyslexia relative to a distribution of such scores obtained by a neurologically uncompromised control group will serve to adjudicate between these two divergent predictions, and, in turn, provide evidence either for or against the competing theoretical models from which they arise.

CHAPTER III

METHOD

Participants

One individual with deep dyslexia (JO) and 60 control participants were recruited.

Patient Description

JO is a 54-year-old woman with 14 years of formal education, two of which were at the postsecondary level. She was first assessed 20 years after the removal of a tumour in the left temporal-parietal region. The tumour and its treatment (surgery and radiation) left JO with right side paralysis and profound language disturbances. Her speech is halting and characterized by word finding difficulties. During an initial screening phase conducted in 1999, JO was asked to read aloud 300 common monomorphemic English words and 108 orthographically legal nonwords. Only 126 of the 300 words were read correctly. Of 174 errors 46 were frank semantic paralexias (e.g., DEBT → "*money*"). This figure represents an underestimate since JO produced semantic errors as first responses to a further 21 words which she subsequently read correctly and were thus assigned a correct response rating. Responses to word targets also included phonological (e.g., STYLE → "*smile*"), orthographic (e.g., TRIED → "*tired*"), and morphological (e.g., SHOWN → "*showing*") paralexias. With respect to nonword oral reading, JO initially failed to produce even a single correct pronunciation. Given her pattern of aloud reading impairment JO clearly fits the deep dyslexic profile.

Control Group

Control participants were 60 undergraduate students at the University of Windsor who were compensated for their participation with extra course credit. All were native

speakers of English with normal or corrected-to-normal vision and no history of neurological disorder.

An age-matched control group was not necessary as participant age has been found not to affect semantic priming results: Bennett and McEvoy (1999) compared semantic priming in continuous lexical decision across older (mean age = 81.3 years) and younger (mean age = 22.8 years) participants and found that the magnitude of both direct and mediated priming effects did not differ between the two age groups.

Materials

The critical stimuli consisted of four sets of 48 prime-target word pairs with each set corresponding to an experimental condition. Of the 192 target word trials, 48 were preceded by direct associate primes (direct priming condition, e.g., GUIDE-TOUR), 48 by indirect associate primes (mediated priming condition, e.g., LION-STRIPES), and 96 by unrelated words (direct and mediated priming control conditions, e.g., STORM-TOUR and DRUG-STRIPES). The remainder of the experimental stimulus set consisted of 192 non-critical word-nonword pairs and 192 nonword filler items. The 48 indirectly related (i.e., two-step) prime-target word pairs were taken from Balota and Lorch (1986). The procedure for development of the 48 directly related (i.e., one-step) prime-target pairs was as follows. First, target words were selected from the CATSCAN database (Durda & Buchanan, 2006) with each matched to a mediated priming condition target with respect to frequency of occurrence in the language and letter length. Directly related primes were then obtained by selecting the strongest associate of each target from the University of South Florida associative norms database (Nelson, McEvoy, & Schreiber, 1998). The stimuli for the mediated and direct priming control conditions were constructed by

replacing the prime in each of the two- and one-step pairs, respectively, with a word of similar frequency and letter length (Durda & Buchanan, 2006) but judged to have no semantic or associative relationship with the paired target. To avoid potential word-specific confounds targets were presented once in the primed condition and once in the unprimed condition. This repetition of target words would not be expected to affect semantic priming given the additive effects of repetition and relatedness (Chapman, Chapman, Curran, & Miller, 1994; Durgunoglu, 1988).

Half of the 96 target nonwords in the word-nonword pairs were matched to the mediated priming condition targets on length, syllable count, and orthographic neighbourhood, and the other half were similarly matched to the direct priming condition word targets. As with the experimental word-word items, the nonword targets in the word-nonword pairs were presented twice so that repetitition status could not be used as a cue to a target's lexical status. Each presentation of a nonword target was preceded by a different word prime with each of the latter obtained from the CATSCAN database (Durda & Buchanan, 2006) and equated in terms of frequency and length to a prime in either the mediated or direct priming condition. Half of the 192 filler nonword items were matched to indirect primes and the other half to direct primes on length, syllable number, and orthographic neighbourhood.

The experimental stimuli were divided into two blocks of 480 trials each. The target words were counterbalanced across blocks; target words that appeared in a primed pair in the first block served in an unprimed pair in the second block and *vice versa.*

Two 12-item practice lists and twelve 4-item sets of buffer trials preceded each block of experimental stimuli and followed each of twelve self-limited breaks,

respectively. The construction of the practice lists and buffer items mirrored the construction of the experimental items.

Apparatus and Procedure

Semantic priming was measured in a continuous lexical decision task in which a lexical decision was required for all stimuli presented. This procedure was selected over the standard lexical decision task in which only targets require a response since, as discussed above, by eliminating postlexical relatedness checking it allows for the emergence of mediated priming regardless of the presence of directly related word pairs (McNamara & Altarriba, 1988).

Stimulus presentation and data collection were controlled by Direct RT software (Jarvis, 2006) run on a personal computer. Stimuli were displayed in lowercase 28 point Times New Roman font. Latency of response was measured as the time between onset of the stimulus and the response, to the nearest millisecond.

Participants received both written and oral instructions that described the nature of the task and its requirements. Participants were asked to silently read each stimulus and then indicate as quickly and as accurately as possible whether they considered the letter string to be a word or a nonword by pressing one of two designated keys. Prior to each experimental block participants received a block of 12 practice trials followed by a self-limited break. The two experimental blocks were separated by a 20-minute interval in which three nonverbal tasks were administered. In order to avoid fatigue and reduce demands on attentional resources, a self-limited break occurred after every 80 experimental trials. Four buffer trials followed each break.

The sequence of events for all practice and experimental blocks was as follows: The instruction to press the "Enter" key when ready to proceed, a blank screen for 1 s, presentation of the first letter string until the participant responded, an interval of 100 ms, presentation of the next letter string until the participant responded, an interval of 100 ms, etc. All participants received the same stimuli. Order of presentation of experimental items was randomized for each participant with the restriction that prime-target pairs were always on contiguous trials. Order of experimental block presentation was counterbalanced across participants.

CHAPTER IV

RESULTS

Response time (RT) analyses were based on means computed for each participant and each condition. Only correct target responses preceded by correct prime responses were included in these means. For each participant, response latencies three standard deviations from the mean for each condition were classified as outliers and excluded from analyses. Semantic priming effects for each participant were computed by subtracting their mean RTs in the direct and mediated conditions from the same in the respective control conditions.

Control Data

Using the dependent variables RT and percentage of correct responses, initial mixed analyses of variance (ANOVAs) indicated that neither gender nor order of experimental block presentation had an effect on response latencies or accuracy (F values for all main effects and interactions < .95). Thus, these between-subject variables were dropped from consideration and the RT and accuracy data were analyzed by participants using 2 x 2 repeated-measures ANOVAs with priming (primed, unprimed) and relationship type (direct, mediated) as within-subject factors. The subject analyses (F_1) were complemented by item analyses (F_2). In the latter, target items were treated as the random effect in 2 x 2 mixed ANOVAs with priming introduced as a within-item factor and relationship type as a between-item factor. All means presented were obtained from the participant analyses.

Response Time Analyses

The mean correct RTs for each condition are displayed in Table 1. As expected, the overall ANOVA revealed a main effect of priming, $F_1(1, 59) = 36.65, p < .001$; $F_2(1, 94) = 9.38, p = .003$, with faster responses to targets preceded by related primes than to targets preceded by unrelated primes. The main effect of relationship type did not approach significance, $F_1(1, 59) = .84, p = .336$; $F_2(1, 94) = .25, p = .617$. Simple effects analyses by participants with a Bonferroni correction ($\alpha = .05/2 = .025$) showed both a direct priming effect (25.6 ms), $F_1(1, 59) = 28.41, p < .001$, and mediated priming effect (10.5 ms), $F_1(1, 59) = 6.61, p = .013$. By-item tests of simple effects revealed a priming effect in the direct condition (26.0 ms), $F_2(1, 47) = 9.67, p = .003$, but not in the indirect condition (11.23 ms), $F_2(1, 47) = 1.61, p = .210$. Qualification of the main effect of priming by a priming-by-relationship type interaction for participants, $F_1(1, 59) = 5.16, p = .027$, indicated that the semantic priming effect obtained for the direct condition was significantly larger than that observed in the mediated condition.

Table 1
Control Participant Mean Correct Response Times (ms) as a Function of Priming and Relationship Type

	Relationship Type					
	Direct			Mediated		
Priming	RT	SD	Priming effect	RT	SD	Priming effect
Unprimed	655.15	101.09		643.88	101.57	
Primed	629.54	96.15	25.61**	633.34	97.36	10.54*

Note. RT = response time; SD = standard deviation.
* $p < .05$
** $p < .001$

Accuracy Analyses

The mean percent correct values for each condition can be found in Table 2. The omnibus ANOVA revealed a main effect for priming by participants, $F_1(1, 59) = 5.59, p = .021$, but not by items, $F_2(1, 94) = 1.51, p = .223$. Neither the main effect for relationship type, $F_1(1, 59) = .17, p = .680$; $F_2(1, 94) = .08, p = .785$, nor its interaction with priming was significant, $F_1(1, 59) = 3.35, p = .072$; $F_2(1, 94) = 3.39, p = .069$. Simple effects analyses by participants with a Bonferroni correction ($\alpha = .05/2 = .025$) indicated greater accuracy in lexical decision for primed versus unprimed targets in the direct, $F_1(1, 59) = 8.76, p = .004$, but not in the indirect, $F_1(1, 59) = .38, p = .541$, condition. The finding of more accurate performance for targets in the direct condition than for targets in the direct control condition approached significance by items, $F_2(1, 47) = 5.02, p = .030$.

Patient Data

JO's mean correct RTs for each condition are displayed in Table 3. A 2 x 2 mixed ANOVA of RT data (priming within, relationship type between) did not reveal a main effect of priming, $F(1, 94) = .03, p = .862$, or a main effect of relationship type,

Table 2
Control Participant Mean Accuracy Scores (percent correct) as a Function of Priming and Relationship Type

	Relationship Type			
	Direct		Mediated	
Priming	Accuracy	SD	Accuracy	SD
Unprimed	98.23	1.85	98.62	1.43
Primed	99.01	1.43	98.78	1.95

Note. SD = standard deviation.

$F(1, 94) = 3.103, p = .081$. Tests of simple effects with a Bonferroni correction ($\alpha = .05/2 = .025$) indicated that both the direct priming effect (18.0 ms), $F(1, 47) = .06, p = .804$, and the interference effect of the mediated condition (-2.68 ms), $F(1, 47) = .003, p = .958$, were not significant.

Analysis of JO's accuracy data (Table 4) revealed comparible error rates for each condition, $\chi^2 (3, N = 190) = .021, p = .99$.

Comparison of Semantic Priming Effects

As noted, following convention, semantic priming was operationalized as the priming difference score, i.e., the mean RT on unprimed or control word pairs minus the mean RT on primed word pairs. However, an assessment of the magnitude of direct and

Table 3
Patient Mean Correct Response Times (ms) as a Function of Priming and Relationship Type

	Relationship Type					
	Direct			Mediated		
Priming	RT	SD	Priming effect	RT	SD	Priming effect
Unprimed	1153.03	365.05		1065.74	262.74	
Primed	1135.00	365.63	18.03	1068.42	276.92	-2.68

Note. RT = response time; SD = standard deviation.

Table 4
Patient Accuracy Scores (percent correct) as a Function of Priming and Relationship Type

	Relationship Type	
	Direct	Mediated
Priming		
Unprimed	97.50	100.00
Primed	100.00	97.78

indirect semantic priming difference scores achieved by JO relative to the distributions of such scores obtained by the control group would not be appropriate since raw priming difference scores can be spuriously inflated in neurological patients who, like JO, exhibit longer than average response times. Fortunately, Chapman et al. (1994) have developed a post hoc analytic technique that serves to remove the effects of overall performance levels. In accordance with this procedure, the regression equation predicting the priming difference score from a measure of overall slowness (unprimed + primed RTs) was computed for each priming condition, using only the data from the control participants. This equation was then used to compute predicted priming difference scores for all participants. Subsequently, the difference between the observed and predicted priming scores for each participant was calculated. As noted by Chapman et al. (1994), each of these residuals represent a corrected priming difference score that measures the extent to which a participant displays more or less priming than would be expected given her or his overall level of performance/slowness. The magnitude of direct and mediated priming demonstrated by JO vis-à-vis the control group was then assessed by treating JO as a sample of $N = 1$ and comparing her corrected difference score in each priming condition against the normative sample via a modified two-tailed independent samples t test as described by Sokal and Rohlf (1995) and advocated for use in single case studies by Crawford and Howell (1998). Results indicated that both the direct and mediated corrected priming difference scores obtained by JO fell well within the normative range; direct priming condition: $t(59) = -.905$, $p = .369$, mediated priming condition: $t(59) = -1.005$, $p = .319$.

CHAPTER V

DISCUSSION

Consistent with the multiple-step automatic spreading activation models of semantic access (e.g., Anderson, 1983; Collins & Loftus, 1975), semantic priming was obtained in the control group for both directly associated and mediated word pairs, with the size of the facilitation effect smaller in the indirect condition. The presence of mediated priming despite the use of a critical stimulus set containing both directly and indirectly semantically related prime-target pairs was an expected replication of previous studies which also employed the continuous lexical decision task (e.g., Bennett & McEvoy, 1999; McKoon & Ratcliff, 1992; McNamara & Altarriba, 1988), reaffirming the ability of this procedure to eliminate the experimental conditions that foster strategic postlexical relatedness checking.

The dual-component attenuated inhibition account with its definitive postulate of exaggerated unfocused spread of activation within the semantic system secondary to attenuated inhibitory connections results in the prediction of heightened direct and mediated semantic priming in deep dyslexia. This prediction was not supported by the data: JO's difference scores for both priming conditions corrected for overall performance level fell well within the normative range. This finding of deep dyslexic semantic priming effects quantitatively indistinct from those observed in a population without neurolinguistic compromise suggests intact implicit semantic access in deep dyslexia, and, in turn, provides further evidence for the position of a fully preserved deep dyslexic semantic system as advanced by Buchanan et al's (2003) failure of inhibition model.

Accounting for "Semantic" Effects

In order to accommodate the concreteness and syntactic class effects that characterize the oral reading of individuals with deep dyslexia, the traditional left hemisphere multiple-deficit models of the disorder proposed by Morton and Patterson (1980) and by Friedman and colleagues (Friedman, 1996; Glosser & Friedman, 1990) both posit functional lesions within the semantic system. For example, Morton and Patterson (1980) contend that the greater facility demonstrated by deep dyslexic patients in aloud reading of concrete versus abstract words is due to selective impairment of the subsystem within the semantic network in which the nodes for abstract words are located.

To date, Buchanan and colleagues have not offered an account of the deep dyslexic concreteness effect within the framework of the failure of inhibition model. However, it is possible to accommodate the observation of greater aloud reading accuracy for concrete relative to abstract words in deep dyslexia without recourse to additional forms of impairment beyond attenuated inhibitory connections within the phonological output lexicon. This inhibition failure account hinges upon a proposed differential in the representation of abstract and concrete words within the semantic system: Given the high shared semantic feature to unique semantic feature ratio (i.e., low specificity) of abstract word nodes relative to concrete word nodes, the former are proposed to have, on average, a far larger number of immediate semantic neighbours (i.e., more dense immediate semantic neighbourhoods). Thus, even with Kiefer et al's (2005) automatic focusing mechanism subserving implicit semantic access intact it is proposed that a greater number of candidate representations will be activated in the semantic network via spreading activation and subsequently addressed in the phonological output

lexicon following presentation of an abstract word than after presentation of a concrete word. In addition, the less uniquely specified, more closely inter-related semantic representations of abstract words relative to those of concrete words are assumed to result in lower activation level differentials between target and neighbouring semantic representations after focused spread of excitation for the former word category. The magnitude of target-neighbour activation level differentials as determined by target word concreteness is preserved in the transmission of activation to the phonological output lexicon. Figure 4 provides idealised graphical descriptions of the proposed immediate neighbourhood activation patterns within the semantic system for concrete and abstract words with feed-forward transmission to the phonological output lexicon.

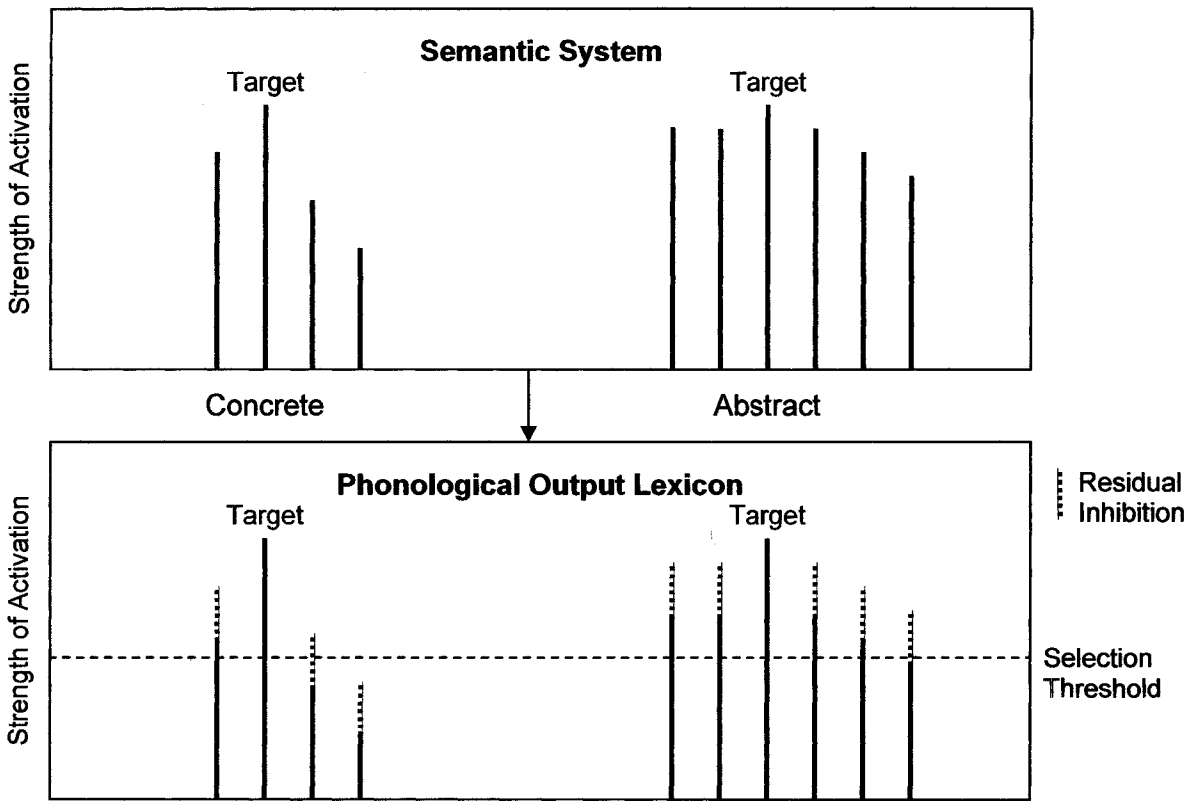

Figure 4. A segment of the deep dyslexic reading system showing the proposed nature of normal immediate neighbourhood activation in the semantic system for concrete and abstract words with feed-forward transmission to the phonological output lexicon.

As Figure 4 indicates, attenuation of the inhibitory connections subserving explicit access in the phonological lexicon has a more deleterious effect upon selection accuracy for words of low imageability: Subsequent to residual (i.e., pathologically reduced) inhibition, the activation levels of a larger number of non-target candidates remain above the selection threshold in the case of abstract words. This results in the common finding of greater semantic paralexic error production to abstract than to concrete words in most cases of deep dyslexia (e.g., Barry & Richardson, 1988).

Support for the hypothesis of lower semantic representation specificity, and, in turn, larger immediate semantic neighbourhoods, for abstract words vis-à-vis concrete words is provided by a study conducted by Newton and Barry (1997): In a task requiring production of words to definitions taken from the Oxford Paperback Dictionary (1988), normal participants found it more difficult to produce target abstract words than to produce frequency-matched target concrete words, with a larger number of incorrect alternative responses being provided for the former word type. For example, for the definition "One who is hostile towards another and seeks to harm the other" only 12 of 30 subjects produced the correct abstract target "enemy," with the wide range of alternate responses including "aggressor," "assailant," "dangerous," "villain," and "vindictive." In contrast, for the definition "A piece of armour carried on the arm to protect the body against missiles or thrusts," 27 participants produced the concrete target "shield," with alternates confined to "guard" and "arm plate."

The syntactic class or part-of-speech effect of the deep dyslexic symptom-complex, in which nouns are read more accurately than modifiers and verbs, can also be explained by the inhibition failure model without positing an additional semantic

impairment since several lines of evidence suggest that this effect is reducible to uncontrolled differences in the concreteness of words in different syntactic categories. Allport and Funnell (1981) presented a set of 30 nouns and 30 verbs matched for imageability and word frequency to five deep dyslexic patients and found no difference in reading accuracy for the two types of words; across all patients 55 percent of the nouns and 61 percent of the verbs were read correctly. Similarly, a multiple regression analysis of the reading performance of the deep dyslexic patient GR conducted by Barry and Richardson (1988) showed that syntactic class had no independent effect when other variables (including concreteness and frequency) were statistically controlled. If, as indicated by available data, the part-of-speech effect is indeed an artefact of concreteness this feature of deep dyslexic reading can also be accounted for by the failure of inhibition formulation on the basis of the divergent concrete and abstract word semantic and phonological representation activation patterns proposed above.

Reading Comprehension in Deep Dyslexia

Consistent with the results of the current investigation, the failure of inhibition model proposes that deep dyslexics exhibit their characteristic errors and effects by processing words through a *normal* semantic system. Thus, by extension one might anticipate such patients to exhibit *normal* reading comprehension.[10] At this juncture, one could simply accept Coltheart's (1980a) broad claim about deep dyslexic comprehension of print: that, where data are available, "when a word cannot be read aloud correctly, it can nevertheless be comprehended" (p. 41), generalized as, "[c]omprehension with inability to pronounce" (p. 29). However, closer analysis of the literature reveals that,

[10] The term *reading comprehension* as employed here refers to comprehension of printed words presented in isolation.

although persons with deep dyslexia have remarkably preserved comprehension of written stimuli relative to their significant oral reading impairments, none show perfect comprehension; some semantic confusion in single word reading, although minor, is apparent (e.g., Newcombe & Marshall, 1980; Patterson, 1978).

One method employed by several researchers to gain insight into reading comprehension in deep dyslexia is that of confidence ratings. Here the patient attempts to read aloud a word and is then required to indicate her or his level of confidence in the veracity of each oral response. Typically, the patient is asked to say "sure" to indicate a high level of confidence, "maybe" if there is doubt regarding response accuracy, and "no" or "wrong" if it is felt that the word uttered was not the presented target. Patterson (1978) used this procedure with two deep dyslexics, DE and PW, who assigned a "sure" rating to 31 and 38 percent of their semantic paralexias, respectively. Similarly, the patients GR (Newcombe & Marshall, 1980) and BL (Nolan & Caramazza, 1982) rated only 61 and 62 percent, respectively, of their semantic errors as false responses. Therefore, it appears that these patients are not aware of the erroneous nature of at least a substantial minority of their semantic paralexic errors.

Evidence of imperfect comprehension of visually presented words in deep dyslexia has also been obtained from word-picture matching and synonym-matching tasks. In the former the patient is presented with a printed target word and is required to select the picture corresponding to the word from an array which includes semantic distractors. Newcombe and Marshall (1980) report that 33 percent of patient GR's responses in a test of word-picture matching were semantic errors, i.e., a semantic distractor (e.g., 'trumpet') was selected over the target (e.g., 'violin'). The deep dyslexic

FD (Friedman & Perlman, 1982) was also asked to complete a word-picture matching task. On six of the 47 trials (13 percent) a distractor semantically related to the target was selected. Patterson (1979) employed synonym-matching to further test the written word comprehension of patients PW and DE. Both were shown 58 items comprising of target words (e.g., COMPETITOR) and an array of five alternatives including a synonym of the target (e.g., RIVAL) and more distant semantically related distractors (e.g., PLAYER); their task in each trial was to select the alternative that was closest in meaning to the target (i.e., the synonym). The performance of both patients (PW, 37/58 correct; DE, 31/58 correct) was clearly poorer than that of control participants (control mean = 52.4 correct, standard deviation = 1.28).

How might the failure of inhibition model with its assumption of a fully intact semantic system explain these findings of subnormal deep dyslexic reading comprehension? In order to address this question, one first needs to delineate the possible mechanisms by which the meaning of a word can be determined from print. In principle, the mapping from print to meaning can occur either directly from knowledge of the target word's visual form (orthography to meaning) or via a phonologically mediated route (orthography to phonology to meaning). Historically, the extent to which each of these mechanisms is used to drive semantic access has proved a highly contentious issue, with some theorists adopting the extreme position that phonological information serves no useful role in word recognition (e.g., Smith, 1973, 1983). However, in the last 20 years, behavioural studies of both children and adults have provided strong evidence for the extensive use of phonology in reading for meaning (e.g., Van Orden, 1987; for fuller discussion, see Frost, 1998; Harm & Seidenberg, 2004). Further support for

phonologically mediated access to word meaning is provided by research employing neuroimaging techniques, with a number of investigations suggesting a confluence of phonological and semantic processing in several regions of the prefrontal cortex (see Westbury & Buchanan, 2006, for a review).

Given the critical role assigned to phonological processing in the computation of meaning, the failure of inhibition model with its core tenet of spurious activation within the phonological output lexicon can readily accommodate the evidence indicating a relatively mild impairment of written word comprehension in deep dyslexia: In the intact reading system phonological information transmitted from the phonological output lexicon to the semantic system subserves explicit semantic access, functioning to aid pruning of activated non-target candidate representations within the semantic system thereby helping to ensure veridical selection, and, in turn, accurate reading comprehension. In deep dyslexia attenuation of inhibitory connections within the phonological lexicon disrupts this phonologically mediated peripheral stabilizing mechanism of the semantic system, which, in turn, can lead to failure to select the appropriate candidate within the activated semantic field. In most instances of visual word recognition, however, orthographic information is sufficient to ensure accurate explicit semantic access in the absence of the normal inhibitory feedback from the phonological output lexicon to the semantic network. Consequently, the magnitude of the deep dyslexic reading comprehension impairment is relatively small in comparison to the difficulty observed in oral production.

Binder and colleagues (Binder, Westbury, McKiernan, Possing, & Medler, 2005) contend that explicit semantic access for abstract words is more reliant on the

informational constraints offered by phonological processing than such access for concrete words. Therefore, if the above failure of inhibition account is correct, the finding of less accurate deep dyslexic reading comprehension for abstract words should obtain. In line with this prediction, Patterson and Besner (1984) report a significant concrete word advantage in reading comprehension in seven deep dyslexic patients as assessed by a word-picture matching task.

Conclusion

The finding of the present study of deep dyslexic direct and mediated semantic priming performance within the normative range provides support for the position of preserved implicit semantic access in deep dyslexia, serving, in turn, to offset a threat posed to the failure of inhibition model by an alternate logically possible account not excluded by the extant empirical data – the dual-component attenuated inhibition model. The current results therefore represent further evidence for a fully intact semantic system in deep dyslexia. Notably, operating upon the parsimonious assumption that the functional aetiology of the disorder is confined to compromised selection in the context of spurious activation within the phonological output lexicon, the inhibition failure model can accommodate not only those aspects of the symptom-complex traditionally attributed to semantic system impairment, namely the concreteness and syntactic class effects, but also the often overlooked single word reading comprehension deficit exhibited by deep dyslexic patients.

Directions for Future Research

If, as proposed above, deep dyslexic impaired reading comprehension results from a disturbance of the phonologically mediated route to word meaning following attenuated

inhibition within the phonological output lexicon then any manipulation that increases reliance upon phonologically mediated semantic access should lead to poorer deep dyslexic performance in reading comprehension tasks. For example, one would anticipate impaired phonological processing to have a more deleterious impact upon comprehension performance for pseudohomophone targets relative to lexical targets in patients with deep dyslexia given that access to the semantic entries associated with the former is determined entirely by phonology.

In closing, it is noted that, in contrast to the failure of inhibition theory position advanced above, Newton and Barry (1997) failed to find a concreteness effect in single word comprehension in deep dyslexia. However, the three tasks employed by these researchers to assess reading comprehension all contained distractor stimuli which were not semantic associates of the target items. For example, in a word-picture matching test the distractor items for the target CAUTION were 'inducement' and 'guilt.' Thus, an individual with deep dyslexia would be able to achieve above-chance performance on these tasks without having a "full" understanding of the target words involved since access to the semantic neighbourhood of the presented item alone without correct selection would be sufficient to obtain the correct response. In other words, the tasks administered by Newton and Barry (1997) do not exclude the possibility of a correct response in patients with deep dyslexia despite incorrect selection of an immediate semantic neighbour of the target representation secondary to impairment of inhibitory feedback from the phonological output lexicon to the semantic network, as predicted by the failure of inhibition model. Therefore, in order to assess the inhibition failure account's prediction of poorer comprehension for abstract versus concrete words in deep

dyslexia given greater reliance of the former word type on compromised phonologically mediated semantic access, use of forced-choice word comprehension tasks in which the distractors are immediate semantic neighbours of the presented items is required.

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VITA AUCTORIS

NAME:	Bruce Andrew King
PLACE OF BIRTH:	Salisbury, Zimbabwe
YEAR OF BIRTH:	1980
EDUCATION:	Sherborne School, Sherborne, England 1997
	St. Charles College, Pietermaritzburg, South Africa 1998
	University of Natal, Pietermaritzburg, South Africa 1999-2001 B.Sc. 2002 B.Sc. Hons.

Consummatum est.