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What we talk about when we talk about access deficits

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Semantic impairments have been divided into storage deficits, in which the semantic representations themselves are damaged, and access deficits, in which the representations are intact but access to them is impaired. The behavioural phenomena that have been associated with access deficits include sensitivity to cueing, sensitivity to presentation rate, performance inconsistency, negative serial position effects, sensitivity to number and strength of competitors, semantic blocking effects, disordered selection between strong and weak competitors, correlation between semantic deficits and executive function deficits and reduced word frequency effects. Four general accounts have been proposed for different subsets of these phenomena: abnormal refractoriness, too much activation, impaired competitive selection and deficits of semantic control. A combination of abnormal refractoriness and impaired competitive selection can account for most of the behavioural phenomena, but there remain several open questions. In particular, it remains unclear whether access deficits represent a single syndrome, a syndrome with multiple subtypes or a variable collection of phenomena, whether the underlying deficit is domain-general or domain-specific, whether it is owing to disorders of inhibition, activation or selection, and the nature of the connection (if any) between access phenomena in aphasia and in neurologically intact controls. Computational models offer a promising approach to answering these questions.

1. Introduction

Neurological damage, such as neurodegenerative disease or stroke, often causes deficits of word-level ('lexical') processing. These deficits can be observed in explicitly verbal tasks, such as picture naming or word-to-picture matching, as well as non-verbal tasks, for example matching two different pictures of the same type of object (for example, a cell phone and a rotary phone). A central distinction among such acquired lexical-semantic[1] deficits is between 'storage' and 'access' deficits. The (often implicit) framework underlying this distinction is cartoonishly schematized in figure 1: there is a container that stores words (or word meanings) and a mechanism for retrieving those words (or meanings) from the container. Within this framework, a storage deficit means that the container is missing some of its contents and an access deficit means that the retrieval mechanism is less effective.

The pattern of semantic deficits associated with semantic dementia (SD) [1]—a variant of frontotemporal dementia that is closely related to the semantic variant of primary progressive aphasia (sPPA)—has played a particularly important role in distinguishing storage and access deficits. Individuals with SD exhibit a progressive semantic deficit that selectively affects specific semantic knowledge before general semantic knowledge (e.g. [2]). For example, as the disease progresses, naming a picture of a swan might progress from the correct response, to a category label ('bird'), to a superordinate category label ('animal'), indicating relative sparing of the more general category information while the more specific item information is lost. Similarly, if participants are shown a line drawing of an object and then asked to reproduce it after a delay, individuals with SD tend to miss atypical features (e.g. a camel might lose its hump) and incorrectly add typical features (e.g. a duck might develop four legs). Although interpretations of SD and sPPA differ to some degree, they all agree that the deficit involves some deterioration in semantic representations, that is, a deficit of the semantic store

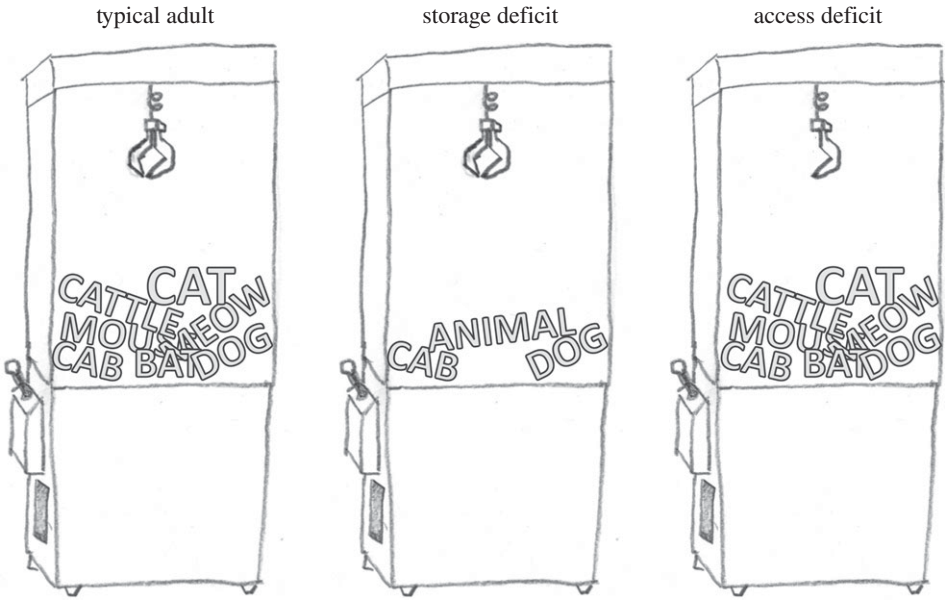

Figure 1. A schematic diagram of the (implicit) framework underlying the storage–access distinction. The typical adult state is shown on the left—there is a collection of words (or concepts) and a mechanism for accessing them. A storage deficit is shown in the middle—some of the words (or concepts) are missing. An access deficit is shown on the right—the words (or concepts) are intact, but the access mechanism is broken.

(e.g. [2–6]). Critically, this syndrome has often been used as the counterpoint for lexical-semantic deficits owing to left hemisphere stroke, with the aetiological difference between dementia and stroke thought to correspond to the distinction between storage and access deficits.

The central premise of access deficits is that the knowledge itself is intact, but access[2] is ineffective, inefficient or inconsistent. The next section will review key phenomena that have been described as reflecting access deficits. In that section, we will avoid discussing theoretical accounts because, in many cases, these accounts address different subsets of phenomena and are differentially compatible, so it will be useful to have all of the phenomena on the table before discussing theoretical perspectives. Once we have reviewed the behavioural data, we will review the theoretical perspectives that have been proposed and assess how well they account for the observed access deficit phenomena. This assessment will lead to a discussion of the remaining open questions and promising directions for future research.

2. The phenomena

In the 30+ years since Warrington & Shallice [7] first described 'semantic access dyslexia', some 40 individual cases of access deficits have been reported and additional group studies have reported access deficit phenomena in aphasia. Although there are clusters of phenomena that have been tested together and many articles (particularly the case studies) report multiple phenomena, overall, the studies use a variety of different experimental approaches and often make only partial contact with one another, so it will be useful to organize this review by phenomenon rather than by report or patient case. Table 1 provides a summary of the key access deficit phenomena, tasks in which they have been shown and patients (or patient groups) that have shown the effect.

(a) Sensitivity to cueing

In phonemic cueing studies, participants are asked to name pictures and, if they cannot name a picture, are provided with a phonemic cue (typically the first two phonemes of the target word). Access deficit (stroke aphasia) patients exhibit strong facilitation from such cues—their performance is much better with a cue than without it. By contrast, storage deficit (SD) patients show virtually no effect of cueing [8–10]. A similar cueing effect has also been shown for miming object use: performance was better when participants were presented with a picture of the object and its name than when presented with only its name [11].

(b) Sensitivity to presentation rate

Once a participant responds to the current trial, the next trial can be presented after a short delay (e.g. 1 s) or a long delay (e.g. 15 s). Participants with access deficits perform much better when this 'response–stimulus interval' (RSI) is long than when it is short, but the performance of storage deficit (SD) patients is not affected by this manipulation of presentation rate. This pattern has been shown in spoken word-to-picture matching [12–15,17–24], spoken-to-written word matching [16,25,26], picture naming [9,23,27,28], reading [29] and matching of non-verbal sounds to pictures or words [30].

(c) Performance inconsistency

When a stimulus is tested multiple times, individuals with an access deficit exhibit inconsistent performance. That is, their pattern of correct and incorrect responses approximates a binomial distribution scaled to their overall accuracy—their performance is like flipping a coin that has probability of coming up 'correct' equal to that participant's overall accuracy. By contrast, storage deficit (SD) patients tend to exhibit highly consistent performance—for a particular stimulus, they tend to either respond correctly on all presentations or incorrectly on all presentations—significantly deviating from a binomial distribution. This pattern has been shown in word-to-picture matching [12,14,15,18,19, 21,22,24], spoken-to-written word matching [16,17], picture-to-picture matching [18], picture naming [27], word reading [31] and matching of non-verbal sounds to pictures or words [30].

Table 1. Summary of key access deficit phenomena, tasks in which they have been shown, and patients (or patient groups) that have shown the effect.

phenomenon	task	patients
sensitivity to cueing	picture naming	10/10 SA patients [8][a]; 4/4 SA patients [9][a]; 6/6 SA patients [10][a]
	object use	8/8 SA patients [11][a]
sensitivity to presentation rate	spoken word-to-picture matching	AA [12]; HEC [13]; VER [14]; YOT [15]; AZ [17,18]; UM-103 [19]; HA, DZ [20]; JM [21]; MH, RS[b] [22]; BM [23]; 7/10 pre- and 6/8 postsurgery glioma patients [24][c]
	spoken-to-written word matching	MED [16]; AZ [25,26]
	picture naming	4/4 SA patients [9][a]; BM [23]; FAS [27]; 18 aphasic patients [28][d,e]
	reading	VYG [29]
	matching non-verbal sounds to pictures or words	AZ, BBB [30]
performance inconsistency	spoken word-to-picture matching	AA [12]; HEC [13]; VER [14]; YOT [15]; MED [16]; AZ [18]; UM-103 [19] JM [21]; MH, RS[b] [22]; 9/9 pre- and 5/6 postsurgery glioma patients [24][c]
	spoken-to-written word matching	AZ [17]; MED [16]
	picture-to-picture matching	AZ [18]
	picture naming	FAS [27]
	word reading	VEM [31]
	matching non-verbal sounds to pictures or words	AZ, BBB [30]
negative serial position effect	spoken word-to-picture matching	AA [12]; HEC [13]; AZ [17,32]; JM [21]; RS[b] [22]; VER [14]; IRQ [33]; 2/9 pre- and 2/7 postsurgery glioma patients [24][c]
	spoken-to-written word matching	AZ [17,25,26,32]; MED [16]; IRQ [33]; JM [34]
	picture naming	FAS [27]
	matching non-verbal sounds to pictures or words	AZ, BBB [30]
sensitivity to number and strength of competitors	spoken word-to-picture matching	AA [12]; UM-103 [19]; HA, DZ [20]; JM [21]; MH, RS[b] [22]; AZ [18]; YOT [15]; IRQ [33]; AZ [32]; 9/10 pre- and 7/8 postsurgery glioma patients [24][c]
	spoken-to-written word matching	AZ [18,25,26,32,35]; MED [16]; IRQ [33]; FBI, NBC [36]
	naming pairs of pictures	2/2 semantic short-term memory deficit patients [37]; JHM [38]
	miscueing effects in picture naming	3/3 aphasic patients [39]; 5/6 SA patients [40][a]; seven SA patients [41,42][a,e]
	matching non-verbal sounds to pictures or words	AZ, BBB [30]
	selecting an object to complete a common task	7/8 SA patients [11][a]
	selecting an object to solve a mechanical puzzle	6/7 SA patients [43][a]
combined negative effects of serial position and competitors	picture naming	3/4 SA patients [9][a]; FAS [27]; 13/18 aphasic patients [28][d]; JHM [38]
	spoken word-to-picture matching	6/8 SA patients [9][a]; 5/9 SA patients [44][a]
disordered activation of strong and weak competitors	spoken word-to-picture matching with eye tracking	five people with Broca's aphasia, three with Wernicke's aphasia [45]

(*Continued*.)

Table 1. (*Continued.*)

phenomenon	task	patients
correlation with executive function deficits	Raven's matrices	10 SA patients [8][a]; seven SA patients [43][a]
	Wisconsin card sorting task	41 stroke patients [46]
	object use tasks	seven SA patients [43][a]; eight SA patients [47][a]

[a]The group of semantic aphasia (SA) patients were participants with chronic impairment from a CVA who failed both picture and word tests of semantic association; all groups of SA patients presented here were subsets of the 10 patients originally tested by Jefferies & Lambon Ralph [8].
[b]RS had multi-focal intrinsic cerebral tumour in the left hemisphere.
[c]A total of 20 patients with temporal lobe glioma tumours was enrolled and tested before and after surgery. Only the high-grade glioma group showed substantive access deficits, so only those patients are considered here.
[d]Left hemisphere CVA patients, six months or more post-onset.
[e]Only the overall effect for the whole group was reported.

(d) Negative serial position effect

In addition to generally inconsistent performance, individuals with access deficits exhibit declining performance over repeated presentations of a stimulus, typically called a *negative serial position effect* (where 'serial position' refers to repetition of the same item, unlike in serial order recall tasks where it refers to position of different items). In the simplest studies, trials were presented in a (pseudo-)random order and repeated multiple times. Individuals with access deficits tended to perform better on the first presentation of a stimulus than on the second. This pattern has been demonstrated in word-to-picture matching [12–14,17,21,22,32,33], spoken-to-written word matching [16,17,25,26,32–34], picture naming [27] and in matching of non-verbal sounds to pictures or words [30].

(e) Sensitivity to number and strength of competitors

In word-to-picture and other matching tasks, participants are presented with an array of response options and must select the one that matches the word. Those options can be semantically related or unrelated to the target (or closely versus distantly related). Access deficit patients perform much better when the distractors are unrelated to the target than when they are related. This has been shown in word-to-picture matching [12,15,18–22,24,32,33] and spoken-to-written word matching [16,18,25,26,32,33,35,36]. In addition, participants with non-fluent aphasia have been reported to exhibit exaggerated slowing when naming pairs of semantically related pictures than unrelated pictures [37,38]. Misleading phonemic cues (e.g. the initial phoneme of a semantically related item, also called a 'miscue'), which presumably strengthen activation of semantic competitors, also lead to significantly poorer picture-naming performance in access deficit patients [39–42].

These effects have also been found in non-verbal matching tasks: non-verbal sound-to-picture (and sound-to-word) matching [30], selecting an object to complete a common task (e.g. 'kill a fly') [11] and selecting an object to solve a mechanical puzzle (remove a wooden block from a clear plastic cylinder) [43]. In the latter study, compared with SD patients, access deficit patients exhibited equal or better knowledge of actions involved in using common objects, such as tools and kitchen implements. However, the access deficit patients were significantly worse at solving mechanical puzzles that required selecting among four implements. That is, they were more impaired when the task required selecting between multiple competitors, but not when it required using only one object.

Access deficit studies typically report that SD patients perform approximately equally well whether the target is presented with related or unrelated distractors; however, studies of SD often show sensitivity to target-distractor similarity. For example, in picture-to-word and written-to-spoken word matching tasks sPPA patients have difficulty rejecting within-category mismatches, for example *zebra–giraffe* [6]. Neurologically intact control participants are also slower and less accurate in speeded word-to-picture matching when the distractors are semantically related to the target [48] and slower even when participants are not speeded and the four-picture display contains only one semantically related distractor [49].[3] As a result, sensitivity to semantic relatedness of competitors may not be specific to access deficits, but access deficits may exaggerate this sensitivity.

(f) Combined negative effects of serial position and competitors

Several studies have examined the combined effects of serial position and semantically related competitors in 'blocked cyclic' paradigms. In these experiments, participants are asked to perform a task (typically picture naming) on small sets of stimuli that are either related or unrelated and these sets are repeated multiple times. For example, a related ('homogeneous') set might consist of six pictures of animals and participants would be presented with these pictures in a random order and asked to name each one, then they would be presented again in a new random order, then again, and so on, for multiple 'cycles' through the entire set. Then participants would go through another set of six pictures from a different category. In a different session, participants would name the same pictures, but the sets would be mixed so each cycle would consist of a mixture of pictures from different categories.

Patients with access deficits exhibit worse performance when the pictures are presented in related (homogeneous) sets than when they are presented in unrelated (mixed) sets and this difference progressively increases as the sets are repeated [9,27]. That is, these patients show an effect of semantic relatedness (sensitivity to competitors) in the first block of trials and this effect exhibits a negative serial position pattern of worsening performance over repetitions.

Table 2. Summary of access deficit case reports of frequency effects by task and type of frequency effect observed.

task	facilitative	none	inhibitory
spoken–written word matching	DZ[a] [20]	HA [20], IRQ [33], MED [16], UM-103 [19]	AZ [26]
word–picture matching	AA[b] [12], VER [14]	AZ [32], HEC [13], MH [22], RS [22]	
reading	VYG [29], JP[c] [63]	AR [7], UM-103 [19]	
picture naming	BM [23], FAS [27]		JP[c] [63]
repetition		AZ [18]	LS[d] [64], PG[d] [64]

[a]Performance was poorer for low-frequency words than medium-frequency words, but high-frequency words were intermediate.
[b]Frequency effect was in response time, not in accuracy.
[c]Not identified as an access deficit patient.
[d]Delayed repetition task.

This pattern has also been demonstrated in a word-to-picture matching version of the task [9,44].

The broader population of individuals with stroke aphasia also appears to exhibit progressively worse performance for related sets relative to unrelated sets and the effect is particularly pronounced in individuals with Broca's aphasia [28,38].

(g) Disordered activation of strong and weak competitors

Eye-tracking studies using a spoken word-to-picture matching task (known as the 'visual world paradigm' [50,51]) have revealed that typical college students exhibit strong partial activation of words that share onsets ('cohorts', e.g. *beaker–beetle*) and weaker activation of words that share offsets ('rhymes', e.g. *parrot–carrot*) [52]. A study of five participants with Broca's aphasia and three participants with Wernicke's aphasia found that, compared with age-matched controls, the Wernicke's aphasic group exhibited increased activation of the cohorts and the Broca's aphasic group exhibited increased activation of the rhymes [45].

In addition, individual-level analysis of the eye-tracking data revealed a negative correlation between rhyme and cohort activation across the eight participants with aphasia but not in age-matched neurologically intact controls [45], suggesting that the participants with aphasia had different trade-offs between activation of strong competitors (cohorts) and weak competitors (rhymes).

(h) Correlation with executive function deficits

Unlike SD patients, individuals with multi-modal semantic deficits following stroke exhibit concomitant deficits on tests of executive function, such as Raven's matrices [53] and the Wisconsin card sorting task [54,55]. In particular, performance on these executive function tests has been found to be correlated with lexical-semantic tasks, such as picture naming, word-to-picture matching, and picture–picture matching [8,43,46] and action semantic tasks, such as object-directed action knowledge and naturalistic sequential action [43,47]. Importantly, SD patients do not exhibit such correlations between performance on semantic tasks and executive function tests.

(i) Reduced word frequency effects

A word's frequency or familiarity is one of the strongest (and longest known) predictors of how quickly and accurately it will be produced and recognized (e.g. [56–59]). This is generally true in aphasia (e.g. [60,61], but see [62]) but insensitivity to frequency has been identified as a diagnostic symptom of access deficits in aphasia. However, the evidence is somewhat mixed.

Table 2 provides a summary of frequency effects reported in case studies of access deficit patients. Although the most commonly reported pattern is the absence of frequency effects, for each of the four most commonly tested tasks (word-to-picture matching, spoken–written word matching, picture naming and word reading) there is at least one case of an access deficit patient showing the standard facilitative effect of frequency. Group and case series studies of patients with semantic access deficits provide further evidence that such patients tend not to exhibit word frequency effects in a wide variety of tasks [8,9,24]. However, closer examination of patients tested by Jefferies *et al*. [8,9] showed that they exhibit the same word frequency effects as SD patients, though they are less sensitive to typicality/regularity [65].

Two studies have also reported reversals of the standard word frequency effect for three access deficit patients. In one study, patient A.Z. was more accurate at spoken-to-written word matching for lower frequency words than higher frequency words [26], although the same patient had shown an absence of frequency effects in repetition [18] and word-to-picture matching [32]. In the second study, patients L.S. and P.G. exhibited reverse frequency effects in a delayed repetition task [64], although both had exhibited no frequency effects in synonym judgement [9] and object use [43]. In addition, these patients were part of the access deficit group[4] that showed no frequency effects in picture naming, word–picture matching, and picture and word versions of the camel and cactus test [8], but did show standard frequency effects in lexical decision, object decision and word reading [65]. Although not specifically identified as an access deficit patient, jargon aphasic patient J.P. [63] exhibited a reverse frequency effect in picture naming, sentence completion, listing members of a semantic category, and answering feature verification questions (e.g. 'Does a tree have foliage?'), and a normal frequency effect for word reading and lexical decision (at least for low imageability words). Given that each of these patients exhibited reversed frequency effects in some tasks and absent or normal frequency effects in other tasks, it would not be accurate to list reversed frequency effects among the access deficit phenomena. Rather, these reports appear to support the more general notion that frequency effects are weak or inconsistent in access deficits and that they may interact with task demands.

In summary, although there are many reports of access deficit patients exhibiting insensitivity to frequency, there are also reports of such patients (in some cases the exact same patients) showing standard facilitative effects of word frequency. As a result, it is unclear whether insensitivity to frequency should be regarded as a core phenomenon of access deficits. As will be discussed in more detail in §3*c*, it is possible that these inconsistent findings are a result of frequency effects being masked by other factors [66] that more strongly influence performance in access deficits, whereas the canonical storage deficit (SD) is associated with exaggerated frequency and typicality effects [2,65,67].

(j) Summary

This section reviewed the behavioural phenomena associated with access deficits, which share the general theme that lexical-semantic representations are intact but access to these representations is impaired. For example, performance inconsistency suggests that representations are sometimes, but not always, available for access and sensitivity to cueing suggests that the representations are intact but difficult to access (otherwise a cue would do no good). Similarly, sensitivity to contextual factors, such as presentation rate, number and relatedness of distractors, and whether this (or a related) concept, has been processed recently (i.e. negative serial position effects) suggest that the problem is with the access mechanism rather than the representations themselves. The general notion of 'access deficits' has been more formally elaborated in several theoretical and computational accounts, which are described in §3.

3. Theoretical perspectives

(a) Abnormal refractoriness

Warrington and colleagues proposed the earliest account of access deficit phenomena, which was based on abnormal refractoriness: a delay in the lexical-semantic system's return to a 'ready' state [7,14–16,18,21,22,27]. The core principle of this account is schematically shown in figure 2*a*: following activation, there is an abnormally long 'refractory period' during which the representation's activation is suppressed. This account intuitively captures sensitivity to presentation rate (longer RSI gives the system more time to return to the ready state). Building on this intuition and incorporating properties of neural signals and the neuromodulatory systems that affect their dynamics, Gotts & Plaut [68] implemented the notion of abnormal refractoriness in a computational model and showed how this approach could account for other key phenomena. Their model used a standard three-layer feed-forward architecture and was trained to map phonological 'spoken word' inputs to semantic representations (i.e. spoken word-to-picture matching). The SD storage deficit was implemented by damaging the connections between units that encode semantic information, as in other models of SD (e.g. [5,69]) and the access deficit was implemented with damage to neuromodulatory systems. The neuromodulatory damage had two effects: (i) Reduced sensitivity to input; that is, a shallower slope for the nonlinear relationship between a unit's net input and its activation (this relationship is also called 'input gain'). (ii) Increased synaptic depression—the diminished effect on a postsynaptic unit when an individual presynaptic unit fires repeatedly. Together, these two effects

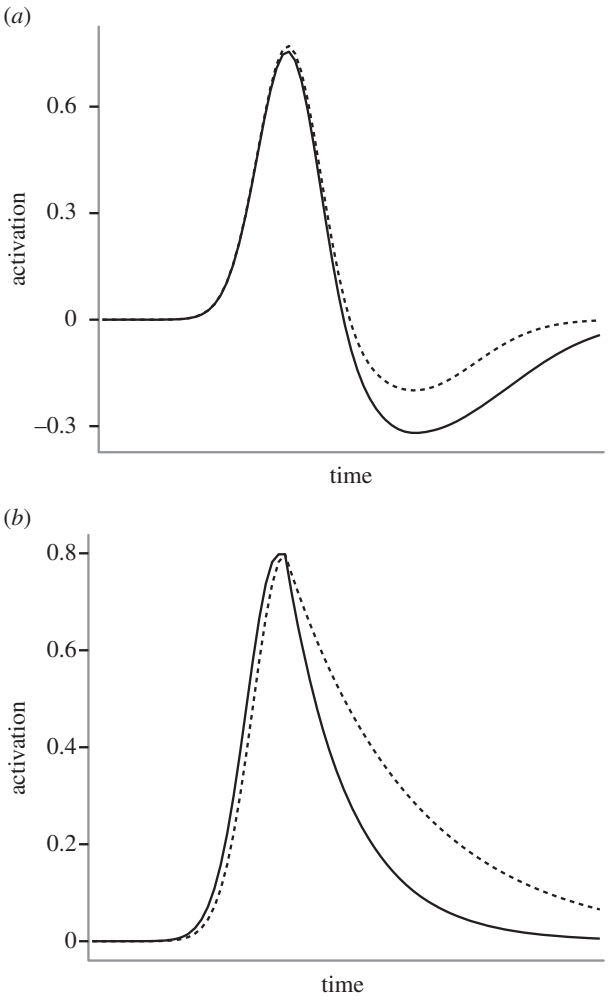

Figure 2. (*a*) A schematic of abnormal refractoriness. Following activation, there is an abnormally large and/or longer lasting refractory suppression. Solid line denotes excessive, whereas dashed line denotes normal. (*b*) A schematic of excessive residual activation. Following activation, there is an abnormally slow decay of activation. Solid line denotes normal, whereas dashed line denotes slow.

produced a transient reduction to the degree to which inputs were able to activate semantic representations.

Simulations demonstrated that this model accounted for presentation rate effects because the synaptic depression effects fade over time; semantic relatedness effects (poorer performance in semantically related versus mixed blocks of trials) because processing semantically related stimuli (by definition) involves some of the same units and connections, which had been affected by synaptic depression; negative serial position effects because synaptic depression builds up over repeated presentations; reduced sensitivity to word frequency because higher frequency words have higher activations and synaptic depression builds up more quickly for units that are more active; and lack of performance consistency owing to peculiarities of stimulus order (i.e. on one repetition the word was identified incorrectly because it happened to follow a highly semantically related word and on another trial it was identified correctly because it happened to follow an unrelated word). Damage to connections produced the complementary storage deficit pattern for each of these phenomena.

(b) Too much activation

The neuromodulatory damage model [68] stands out as perhaps the most comprehensive and neurobiologically motivated

attempt to account for access deficit phenomena and the distinction between access and storage deficits. However, there is one phenomenon—negative serial position effects—for which this model's account contrasts sharply with alternative accounts. The build-up of synaptic depression over repeated presentations of a stimulus makes that stimulus representation less likely to be reactivated, similar to other accounts that proposed excessive inhibition or refractoriness as the basis of access deficits [14,21,27,70]. In contrast to this view, other researchers have argued that negative serial position effects are owing to an excessive build-up of activation, as schematically shown in figure 2*b*. That is, rather than being inhibited (or synaptically depressed), representations remain active too long and this residual activation competes with new inputs, preventing them from becoming active. If the stimuli are semantically related (as in blocked cyclic picture naming), then the new input would also provide partial activation to the residually active representations from previous trials, thus exacerbating the excessive residual activation.

Key evidence for this view comes from analyses of errors in blocked cyclic picture naming and related paradigms. If semantic representations are being excessively inhibited, then the progressive decrease in accuracy over cycles should correspond to omission errors (i.e. all representations are inhibited and cannot be activated enough to produce a response) with perseveration errors—unintended and erroneous repetition of a previous target—uncommon. By contrast, the observed error pattern included semantic errors (in semantically homogeneous blocks of trials) and greater-than-chance probability of perseveration errors [28,71]. This pattern is consistent with representations remaining excessively active and interfering with new inputs.

A third view also holds that the interference effects are owing to excessive activation, but argues that this activation is owing to incremental learning rather than residual activation [72,73]. On this view, each presentation of a stimulus strengthens its connections, so that when a semantically related stimulus (which shares some of the units and connections) is presented, the representation of the previously presented stimulus is more strongly re-activated, thus increasing competition with the new target. The most important evidence in support of this view is that the decay of interference appears to be driven by the number of intervening trials rather than elapsed time, suggesting that the excessive activation does not decay passively but requires input to overcome it. Note that this pattern is in direct contradiction to the presentation rate effects—better performance when there is a longer delay between trials—which do suggest a passive decay. There is also growing evidence that both failure to activate representations of current inputs and failure to inhibit representations of previous inputs may contribute to perseveration errors [74].

(c) Competitive selection

Another strain of accounts focuses on the competitive selection demands of lexical-semantic tasks. Lexical-semantic processing involves 'parallel activation'—multiple, related candidates are activated and some cognitive control is required to select one to become the response. Functional neuroimaging studies of neurologically intact participants have identified the left inferior frontal gyrus (LIFG) as particularly important for this competitive selection function [75,76]. In blocked cyclic naming, LIFG activation is correlated with number of errors produced by neurologically intact controls and degree of damage in LIFG is correlated with the degree of build-up of the blocking (interference) effect in participants with aphasia [77]. Similarly, patients with lesions that include LIFG exhibit stronger blocked cyclic effects in word-to-picture and picture-to-picture matching tasks than patients with strictly temporal–parietal lesions [44]. Even in simple picture naming, targets that have many close semantic neighbours (such as 'duck', which is closely related to goose, seagull, dove, chicken and many other concepts, when compared with 'frog', which has fewer closely related concepts) are more prone to naming errors [78] and this negative effect of semantic neighbours is particularly strong in patients with LIFG damage [79].

The reduced or reversed effects of word frequency may also be related to deficits of competitive selection. For example, the reverse frequency effects exhibited by patient J.P. were proposed to be owing to high-frequency words having more or stronger associations (neighbours) that produced too much competition [63] (and see [80] for a similar account). Word frequency is also strongly correlated with 'semantic diversity' [81]: words that are used more frequently tend to be used in a wider variety of different contexts so they also tend to be more semantically variable or ambiguous. This ambiguity may have a detrimental effect on processing for individuals with a deficit selecting between alternative competitors, that is, between different meanings or senses of the word. Indeed, semantic diversity had a strong detrimental effect on synonym judgements for a group of stroke aphasic patients but not for matched SD patients [66]. Furthermore, the aphasic group did not exhibit an effect of frequency unless semantic diversity was taken into account, at which point the standard facilitative effect of word frequency emerged.

Because these effects involve difficulty in selecting among semantically related concepts, it may be tempting to propose that they are due to a blurring of distinctions among semantic representations, that is, a semantic storage deficit. However, individuals with the quintessential semantic storage deficits—SD and the semantic variant of primary progressive aphasia—show blurring of within-category distinctions [5,6] but not the build-up of interference in blocked cycling tasks [9]. This sort of dissociation is the motivation for distinguishing between representational deficits (i.e. the semantic representation no longer has enough detail to distinguish between category members) and selection deficits (i.e. all category members are activated with sufficient detail but the mechanism for selecting between them is impaired).

The eye-tracking data discussed in §2*g* [45] are also relevant because they suggest impairments of selection between phonological competitors rather than semantic competitors. The key finding in that study was a negative correlation between activation of words with similar onsets (e.g. *penny–pencil*) and activation of words with similar offsets (e.g. *carrot–parrot*). That study also examined computational mechanisms that might account for their results and found that the best account was differences in the nonlinear relationship between activation of a representation and the probability of a response (similar to the input gain effect of neuromodulatory damage in Gotts & Plaut's model [68]). These differences are schematically shown in figure 3. On this view, 'competitive selection' is the probability that a representation will be selected to become the behavioural

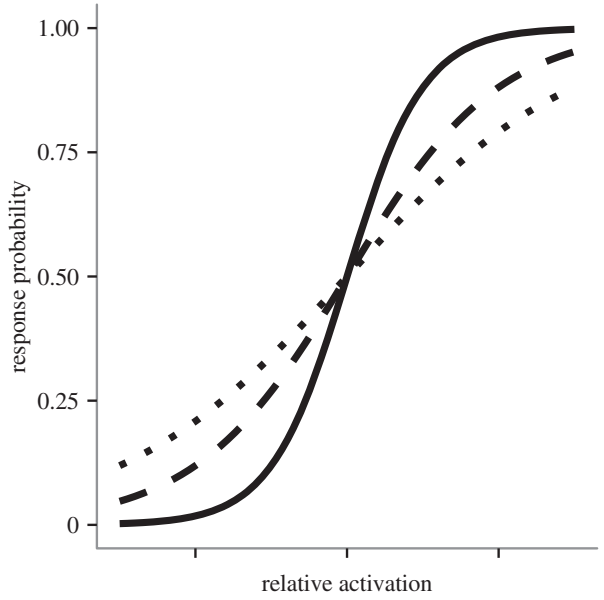

Figure 3. Effect of selectivity (solid line denotes high, dashed line denotes medium and dotted line denotes low) on the mapping between relative activation and response probability. Lower selectivity approaches a linear relationship such that any incremental increase in relative activation produces an (almost) equivalent increase in response probability. Higher selectivity approaches a threshold relationship such that suprathreshold relative activation will (almost) always produce that response and subthreshold relative activation will (almost) never produce a response.

response based on that representation's degree of activation relative to other representations. This is simply a mathematical formulation of the intuitive notion typically implied by the term 'selection': multiple representations are activated and if one rises above the others by a sufficient amount, then it is selected for response. High selectivity approximates a threshold function such that no response can be made unless a representation becomes sufficiently active relative to other representations and as soon as one does, it will be the only response that will be produced. Low selectivity corresponds to an approximately linear relationship such that incremental increases in relative activation correspond to (approximately) equal increases in response probability. In the extreme, this implementation of reduced selectivity eliminates the selection process entirely: response probability is (approximately) equivalent to relative activation.

Assuming that lexical-semantic representations and activation processes are intact in access deficit patients and that the task is structured such that control performance corresponds to near-ceiling selection of the target (as is typical in picture naming, word-to-picture matching, and other commonly used tasks), then target representations should be the most active by a fairly large margin; that is, the target should have fairly high relative activation. In the high-selectivity case, the sharp selection function means that response probability will be relatively insensitive to small differences in relative activation produced by having more or stronger competitors. By contrast, low selectivity means that even if the target is much more active than all of the competitors, response probability will nevertheless show sensitivity to increases in the activation of those competitors. Thus, reduced selectivity can account for the effects of number and strength of competitors.

The phonological competition eye-tracking data provide important insights into selection (for fixation) of items with low relative activation—the rhyme competitors. For these items, the effect of selectivity should be reversed: high selectivity means a very strong preference for high relative activation items, so these low relative activation items should (almost) never be fixated; low selectivity means that even low relative activation items can be selected (albeit not as much as high relative activation items). If Broca's aphasia involves this formulation of reduced selectivity, then it would account for the disordered selection among strong and weak competitors.

These simulations used the TRACE model of speech perception and spoken word recognition [82] and the selection mechanism was based on the Luce choice rule [83], so the model does not make a theoretical commitment regarding whether the selection deficits should be domain-specific (like the TRACE model) or domain-general (like the Luce choice rule). It also does not require a claim regarding the neural basis of such deficits, though the empirical evidence suggests that LIFG damage should correspond to reduced selectivity and that this should be a relatively domain-general effect.

(d) Semantic control

Jefferies and Lambon Ralph have proposed that access deficits reflect impairments of 'semantic control processes that allow task- and context-relevant aspects of knowledge to be brought to the fore, while irrelevant information is suppressed' [8,84, p. 618]. This view is related to the competitive selection account described in the previous section, though semantic control mechanisms are proposed to be somewhat more general. In particular, semantic control is proposed to involve executive function mechanisms beyond just competitive selection, thus explaining the observed correlations between semantic deficits and performance on executive function tests, for example Raven's matrices.

As mentioned earlier, LIFG is thought to play an important role in competitive selection but this and neighbouring brain regions (which are likely to be damaged even in focal lesion cases) are also important for non-selection control functions, including semantic retrieval [85], inhibitory control [86] and complex sequential behaviour [87,88]. Thus, it is quite possible that access deficits are caused by impairments of cognitive control that extend beyond response selection. However, the semantic control account remains mechanistically vague because it has not been implemented computationally. In the absence of a concrete implementation, it is difficult to assess to what degree this view can account for the access deficit phenomena and how it differs from the other theoretical perspectives.

(e) Doubts of storage–access distinction and alternative perspectives

A much more radical alternative theoretical position is to question the storage–access distinction itself. Rapp and Caramazza [89] questioned the distinction (1) because purported access deficit patients did not consistently exhibit all of the defining characteristics of access deficits or exhibited them in one modality but not in others and (2) because the distinction requires certain theoretical commitments regarding the nature of storage and access (for example, that

Table 3. Access deficit phenomena and potential accounts. Checkmark indicates that the proposed mechanism has been demonstrated to account for the phenomenon; question mark indicates potential for the proposed mechanism to account for the phenomenon.

phenomenon	abnormal refractoriness	too much activation: slow decay	too much activation: learning	competitive selection	semantic control
sensitivity to presentation rate	✓	✓	✗		
negative serial position effect	✓	✓	✓		
combined negative effects of serial position and competitors	✓	✓	✓		
error types in blocked cyclic naming	✗	✓	✓		
time course of perseveration errors	✗	✗	✓		
performance inconsistency	✓	?	?	?	?
sensitivity to number and strength of competitors				✓	✓
disordered activation of strong and weak competitors				✓	?
correlation with executive function deficits				?	✓
sensitivity to cueing				?	?

access deficits cause inconsistent performance but storage deficits cause consistent performance) that are not otherwise motivated and, given that not all access deficit patients conformed to all of the criteria, are not substantiated by the data.

In addition, at least some of the access deficit phenomena can arise without appealing to access mechanisms. For example, on the incremental learning account of cumulative semantic interference [72], merely increasing the noise in the system is enough for the incremental learning to produce the observed patterns of aphasic picture-naming errors in blocked cyclic naming and related paradigms. Another perspective draws on the classic neuropsychological notion of 'disconnection' syndromes, for example optic aphasia [90], in which patients have difficulty in naming visually presented objects that is thought to be owing to disconnection of visual-semantic input from lexical output. This perspective argues that perseveration errors arise when new input is unable to overcome normal residual activation, giving rise to domain-specific perseveration errors [91,92]. It is also supported by the finding that high-grade glioma patients who exhibited access deficits had maximum lesion overlap in subcortical white matter in left posterior superior temporal lobe, suggesting that their deficits may have been owing to disconnection of the posterior temporal lexical naming system from the anterior temporal semantic system [24]. Although narrow in terms of the phenomena it currently captures, this view is quite radical (and radically parsimonious) in avoiding reference to special access mechanisms.

(f) A path forward

To some degree, the empirical criticism raised by Rapp & Caramazza [89] has been addressed by the subsequent work of Warrington & Cipolotti [22] and Jefferies and Lambon Ralph [8,84], in which access deficit and storage deficit patients have been directly compared and double dissociations demonstrated across several criteria. The modelling work of Gotts & Plaut [68] showed how the different patterns of performance associated with access versus storage deficits could arise from different kinds of neurological damage, thus making a major step towards addressing the theoretical criticism raised by Rapp & Caramazza [89]. Nevertheless, this review of access deficit phenomena and theoretical perspectives makes plain that no single existing theory can account for all of the phenomena. In part, this is because the theories address different subsets of the phenomena. As a result, although the accounts may be insufficient on their own, there are only a few points of direct conflict, so it may be possible to integrate them to form a complete account.

Table 3 summarizes which theories can account for which access deficit phenomena. The checkmarks indicate that simulations have already shown that a particular mechanism can account for a particular phenomenon or can be straightforwardly expected to account for the phenomenon; the question marks identify cases where the mechanism has potential to account for the phenomenon, but requires implementation and demonstration to be certain. The phenomena in table 3 have been re-ordered to emphasize that the theoretical perspectives appear to account for two essentially non-overlapping

clusters of phenomena: one having to do with build-up of inhibition (refractoriness) or activation across multiple trials and one having to do with selecting the correct response on a single trial.

The Gotts and Plaut implementation of abnormal refractoriness as neuromodulatory damage accounted for sensitivity to presentation rate, negative serial position effects and build-up of semantic interference over repeated presentations, though it makes the wrong predictions regarding the kinds of errors that will be produced in blocked cyclic naming, including the time course of perseveration errors. The observation of semantic and perseveration errors in blocked naming suggests that there is too much activation in the system and only the incremental learning version of this account can explain the time course of perseveration errors. However, this account cannot explain sensitivity to presentation rate.

When implemented as the mapping between relative activation and response probability, competitive selection deficits can account for disordered selection between strong and weak competitors and sensitivity to number and strength of competitors. Gotts and Plaut offered an account of performance inconsistency based on particulars of (random) trial ordering, though it is also possible that selection and/or control deficits may also cause heightened sensitivity to extraneous contextual factors (for example, trial order) and produce inconsistent performance.

Insofar as semantic control and competitive selection are closely related, they should account for the same phenomena in largely the same way. For example, simulations showed that competitive selection deficits can account for disordered selection between strong and weak competitors and perhaps the semantic control account would work the same way. The semantic control account explicitly asserts a relationship between semantic control deficits and executive function deficits; if competitive selection is a domain-general process related to executive functions, then competitive selection deficits may also explain the correlations between semantic deficits and executive function deficits. Sensitivity to cueing may be owing to targets failing to reach sufficient relative activation to be selected for response, which could reflect a selection deficit but could also be owing to other deficits affecting the activation processes. Competitive selection (and perhaps semantic control) may also be able to account for reduced word frequency effects if that reduction is owing to difficulty in resolving competition from semantic diversity [66] or because low selectivity tends to reduce the effect of word frequency [93].

The clustering of phenomena to theoretical perspective may simply reflect the research interests of the theorists, but it may also reflect that there are multiple distinct mechanisms involved in access deficits. These mechanisms may have a common neural substrate: for example, frontal regions involved in competitive selection may also be involved in neuromodulatory circuits that modulate the dynamics of refractoriness. On the other hand, the mechanisms might be dissociable such that access deficits could be divided into a selection deficit subtype and a refractoriness/overactivation deficit subtype. These and other open questions are discussed in more detail in §4.

4. Open questions and future directions

(a) Defining access and access deficits

Perhaps the most obvious conclusion from our review of access deficit phenomena is that there is little agreement about what mechanistic elements constitute semantic access and how to define 'access deficit'. The notion of semantic access is often mentioned in accounts of behavioural data but it is woefully underspecified, being alternately identical to, related to or completely distinct from activation, inhibition, selection and other aspects of processing. Without an explicit theory of what it means to 'access' a concept's representation, the notion of an 'access deficit' is also underspecified. If 'access deficit' is considered to be a syndrome, then patients should be expected to exhibit all (or nearly all) of the access deficit phenomena. Few, if any, of the reported cases would meet such a strict definition. A somewhat less restrictive definition would require only a subset of the phenomena as diagnostic criteria; however, given that each of the phenomena has been the focus of detailed study and each has been called an access deficit, there is currently no principled way to define which phenomena should be considered diagnostic. Another alternative is that there are functional subtypes of access deficit such that each patient should exhibit all of the phenomena that are relevant for that subtype. The clustering of phenomena to theoretical perspective suggests selection and refractoriness/overactivation as two candidate subtypes but, so far, there is limited behavioural evidence to support this distinction. The least restrictive definition would be to consider 'access' not as a single process but as a general component or aspect of processing, similar to 'semantic' or 'phonological', so the term 'access deficits' would refer generally to a set of phenomena that may have common or independent causes—similar to the way 'semantic deficits' refers to a general set of phenomena without a necessary commitment to a single common cause.

(b) Domain-generality and domain-specificity

Many of the case reports of access deficits are task-specific [18,23,27,29]: for example, a patient may exhibit access deficits in picture naming but not in word-to-picture matching or word reading. This task-specificity suggests that access deficits may be domain-specific (possibly owing to partial disconnection of specific elements of processing). On the other hand, semantic control deficit patients exhibit deficits across different tasks [8,84] and correlations with executive function deficits [8,43,46,47], suggesting a domain-general control deficit. However, these patients were specifically selected for multi-modal semantic deficits, so perhaps this is merely a difference in patient selection. In other words, access deficits may be domain-specific and the 'semantic control' patients were those who happened to have domain-specific access deficits in multiple domains. Domain-general access deficits may also interact with modality- or task-specific demands to produce the appearance of task-specific deficits. Distinguishing these alternatives requires careful experimental work (e.g. [94]) and concrete models of domain-general and domain-specific processes, but answering this question critically informs the possible mechanisms underlying lexical and semantic access: is there a general access function that operates over all domains or is access a separate process in each domain?

It is also important to recognize an implicit assumption that, given a link between semantic and executive function deficits, it is the executive function deficits that are causing the semantic deficits. Although this is certainly possible, it is also possible that the causal relationship can run in the opposite direction. That is, lexical and/or semantic deficits

may impair performance on tests of executive function. This kind of relationship has been shown in a variety of ostensibly non-verbal perceptual and cognitive tasks [46,95–97], particularly ones that involve attending to a single feature (e.g. colour), as is the case in many tests of executive function. However, SD patients do not show correlations between severity of semantic and executive function deficits [8,43], indeed, they often perform near ceiling on tests such as Raven's matrices and the Wisconsin card sorting task. So even if lexical and/or semantic deficits are (at least partly) responsible for the executive function deficits in aphasia, there must still be some important difference between access and storage semantic deficits such that they cause executive function deficits in the former but not in the latter.

(c) Inhibition, activation and learning accounts

As reviewed above, there are three different accounts of the same negative serial position and semantic blocking effects: excessive build-up of inhibition, excessive build-up of activation and incremental learning. Insofar as each account can explain the same data, there is no need for all three. On the other hand, each account is also supported by behavioural evidence that appears incompatible with the other accounts. For example, the presentation rate effects suggest a passive decay process, but the sensitivity to number of intervening items rather than elapsed time suggests an incremental learning mechanism, and domain-specific perseveration data [91,92] suggest deficits at particular levels of processing. A full model of lexical-semantic processing would involve activation, inhibition and learning processes, so it is possible that each of these aspects can be impaired separately. However, because these proposed impairment mechanisms account for some of the same data and conflict with other data, a compelling account must balance parsimony and breadth of coverage.

(d) Computational models, not verbal models

As reviewed above, the set of access deficit phenomena is relatively large and complex. In this situation, it is difficult to evaluate whether a proposed verbal model would truly account for the observed data and it is virtually impossible to evaluate whether it would account for new data. In other words, intuitions and verbal theories are not enough because they can be claimed to predict (or not predict) just about anything. Computational models provide a concrete implementation of a proposed theory that can then be tested empirically to evaluate whether it truly accounts for the observed data and to make novel predictions. At their best, computational models do more than just simulate the observed data; they concretely show what mechanisms are needed for a system to exhibit certain phenomena (e.g. [72,92]; and others have discussed the role of computational modelling in more detail [98,99]).

Although computational models have been used in studies of access deficits, they have generally targeted specific non-overlapping subsets of phenomena. Several of the models only capture data from blocked cyclic naming and related picture-naming paradigms [72,73]; the competitive selection model [45] was only evaluated in the context of the phonological competition data. The neuromodulatory damage model [68] was particularly informative because it accounted for multiple access deficit phenomena as well as the dissociation of access and storage deficits. Similarly, the Lichtheim 2 model [100] captured a variety of semantic deficit patterns, though it has not been used to address access deficits. Future models should build on these successes in order to develop a comprehensive account of access deficits. Critically, new models need to connect with existing models and show that they account for the data that old models captured, otherwise we might find ourselves with as many models as phenomena.

Models should also examine access and storage deficits together because, as the scope of access deficit phenomena expands, the boundary between access and storage deficits will become more complex. For example, specific failure to name tools is typically considered a (category-specific) storage deficit, but should slow or delayed activation of tool-use knowledge in limb apraxia [101,102] be considered a storage or access deficit? Does sensitivity to cueing represent difficulty in activating the relevant representations or difficulty in selecting the correct response? Models that capture both access and storage deficits (e.g. [68]) within the same framework provide a way to answer such questions and will do the most to advance our understanding of lexical-semantic processing and deficits.

(e) Access deficit phenomena in controls

Several of the access deficit phenomena have been documented in neurologically intact controls, including sensitivity to presentation rate (better performance in a speeded spoken word-to-picture matching task when there is 1 s between trials than when this RSI is eliminated [48] and in picture naming when there is 5 s between trials than when this RSI is eliminated [28]), negative serial position effects (lower accuracy on the second and third repetition of a stimulus than on the first presentation in a speeded spoken word-to-picture matching task [48]), and sensitivity to semantic relatedness of competitors (discussed above) [48,49]. Neurologically intact control participants also exhibit semantic blocking effects, albeit in response times rather than error rates [28,103,104], and studies with controls have shown that picture-naming times increase linearly as a function of the number of preceding pictures from the same category [73]. Furthermore, the relatedness need not be semantic: the same pattern has been found for orthographically related words in a reading aloud task [105].

The fact that neurologically intact controls exhibit at least some access deficit phenomena indicates that theories of access deficits ought to make contact with normal performance. In other contexts, this has been called the *continuity thesis* [106]: normal performance constitutes one endpoint of a continuum of deficit severity. Computational models can (and often do) naturally capture the continuity thesis if they are constructed to model normal performance and then 'lesioned' in a graded way to produce a continuum of deficit severity.

The induction of access deficit phenomena in neurologically intact controls can also inform theories of access deficits. For example, adding time pressure has been shown to induce sensitivity to presentation rate and negative serial position effects, but not to abolish word frequency effects [48] (indeed, time pressure seems to increase frequency effects in spoken word recognition [93]). There is a substantial amount of behavioural, theoretical and computational work on how time pressure affects lexical processing, including changes in gain [93,107,108], which is closely related to the models of competitive selection [45] and neuromodulatory

damage [68] discussed above. This suggests that it may be possible to develop a single account that captures these phenomena and that reduced frequency effects may have a different computational basis.

5. Concluding remarks

There is a fascinating cluster of phenomena that have been described as acquired lexical-semantic access deficits and that provide important constraints on theories of language processing. However, the very notion of semantic 'access' is underspecified, which has hampered efforts to move beyond a purely empirical cataloguing of phenomena. Computational models offer a critical tool because they require researchers to concretely specify the operation of the system, including what is meant by semantic 'access' and the nature of a proposed 'access deficit'. Computational models are particularly well suited to investigating to what extent access deficit phenomena result from too much inhibition, too much activation or impaired selection, and the relationship between access deficit phenomena in aphasia and in neurologically intact controls. Further behavioural (and perhaps computational) work is also required to establish whether access deficits are best construed as domain-general or domain-specific.

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Endnotes

[1]We will use the term 'lexical-semantic' to refer to semantic processing at the word or object level, as opposed to smaller (morphological) or larger (sentence or discourse) scales. Some of the tasks we will discuss explicitly involve word processing (e.g. picture naming, word-to-picture matching, etc.) and some do not (e.g. picture-to-picture matching), but all of the tasks examine semantic processing on the scale of words.
[2]What is meant by 'access' differs across accounts and several other terms ('activation', 'retrieval', 'selection', etc.) have also been used to describe the locus of the impairment. We will use 'access' as a convenient, relatively theory-neutral umbrella term for the set of phenomena that are distinguished from storage deficits.
[3]This study focused on eye-tracking data and did not report the reaction time effect: participants were, on average, 116 ms (95% CI: 55–177 ms) slower when the four-picture display contained one distractor that was semantically close to the target than when it contained only unrelated distractors ($t_{37} = 3.8$, $p < 0.001$).
[4]Individual patient data for these studies were not available.

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