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NEURAL NETWORK MECHANISMS OF ADULT LANGUAGE

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Despite enormous progress made by linguists, psycholinguists, cognitive neuropsychologists, and behavioral neurologists over the past 75 years in understanding the cerebral basis for language function, a fundamental question has remained unanswered: how biological tissue made up of tens of billions of neurons densely interconnected with each other in neural networks (1,000–10,000 connections per cortical neuron), and operating on the basis of electrochemical principles, could possibly support the substantially symbolic processes involved in language function. In 1986, in an epochal work, McClelland et al. (1986) elaborated an essential insight: representations in the brain are population encoded (distributed), that is, instantiated as patterns of activity involving very large numbers of relatively simple units (plausibly corresponding to neurons or cortical micro-columns). Computer simulations have demonstrated that such networks have very characteristic behavioral properties; these properties are highly germane to understanding how the brain supports language function; and networks incorporating population-encoded representations have a remarkable capacity to emulate the behavior of normal and brain-injured human subjects.

Most of this chapter will involve an overview of language systems that takes into account the fundamental neurobiological principle of population encoding of representations (for further detail, see Nadeau, 2001, 2012; Roth et al., 2006). This conceptualization will define features of aphasia and applications to aphasia therapy, the topics of the final section of the chapter. Although the population encoding of representations has long been well established (Rolls and Deco, 2002; Rolls and Treves, 1998), any particular model, including that discussed here, represents but a hypothesis, to be tested and refined through further empirical work.

Language function

Core functions: Phonology, semantics, and lexical-semantics

The topography of the Wernicke–Lichtheim model (Roth et al., 2006) is presented in Figure 2.1. Each oval signifies a very large number of individual units. Every unit in a given oval is connected to every unit in the adjacent ovals. All connections are two-way, thereby emulating what we see in the brain. Information is represented in the model as the strength of connections between units (in analogy to synapses in the brain). The activity of units is defined as a nonlinear
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function of their input (in many simulations as a sigmoid (∫) function over the range of 0–1). The output of units is another nonlinear function. The units in hidden unit domains support representations that cannot be defined in behavioral terms. These domains, coupled with nonlinear unit functions, are essential to the computational capabilities of the network and endow the network with the capacity for supporting discrete entities (e.g., symbols); translating representations in one domain into substantially unrelated (orthogonal) representations, e.g., concept representations (semantics/word meanings) into phonological representations (word sounds); and representing sequence knowledge. The small circles appended to the three major linguistic domains signify that every unit within a domain is connected to every other unit in that domain. This endows these domains with an auto-associator property: the propensity for the activity pattern within the domain to settle into a state—an attractor basin—that represents an optimal response to the pattern of input. Input to the network leads to spreading activation throughout the network, such that, in the course of repeated bottom-up/top-down interactions, the entire network eventually settles into an optimal state, which defines output. Figure 2.1 also shows how this network might be plausibly mapped onto the brain surface and it captures the fact that, although language is predominantly supported by the left hemisphere in right handers and most left handers, there is some redundant representation of language knowledge in the right hemisphere (see Nadeau, 2012 for detail).

Figure 2.1 Parallel distributed processing model of language (upper left) with plausible mapping to the brain (upper right). There is abundant evidence that there is substantial redundant language knowledge in the right hemisphere (lower). Domains are shaded similarly in each figure. HU = hidden units. (From Roth et al., 2006).
Concept representations: Semantics

I will begin with the domain of concept representations because its function provides the most insight into the meaning of a population encoded representation. The operation of this domain can be best illustrated by a model developed by Rumelhart et al. (1986). This “rooms in a house” model was comprised of 40 “feature” units, each corresponding to an article typically found in particular rooms or an aspect of particular rooms. Connection strengths were defined by the likelihood that any two features might appear in conjunction in a typical house. When one or more units was clamped into the “on” state, activation spread throughout the model and the model eventually settled into a steady state that implicitly defined a particular room in a house. Thus, clamping “oven” ultimately resulted in activation of all the items one would expect to find in a kitchen and thereby implicitly defined, via a distributed or population encoded representation, the concept of a kitchen. No kitchen unit per se was turned on. Rather, kitchen was defined by the pattern of feature units that were activated. The network contained the knowledge, in the totality of its connections, that enabled this representation to be generated. The model could also generate distributed representations of other rooms in a house (e.g., bathroom, bedroom, living room, study) and blends of rooms that were not anticipated in the programming of the model (e.g., clamping both bed and sofa led to a distributed representation of a large, fancy bedroom replete with a fireplace, television, and sofa). This auto-associator model, simple though it is, has the essential attributes of a network that might instantiate semantic knowledge and be capable of generating distributed representations corresponding to concepts.

![Figure 2.2 The topography of the semantic network activity function in the vicinity of the mammal attractor basin.](image)

*Figure 2.2* The topography of the semantic network activity function in the vicinity of the mammal attractor basin. Each point corresponds to an activity level of all features in a N-dimensional feature hyperspace. The point of maximal typicality is represented by the centroid of a basin/sub-basin. Distance from the centroid reflects degree of atypicality. The value of $\theta$ defines the manner in which atypicality is defined. For example, whales and platypuses are both atypical but in very different ways. (From Nadeau, 2012).
Let us now scale this up to a concepts network that could plausibly support the knowledge in the human brain. The activity pattern of this much larger concepts domain would be defined by a function in a N-dimensional feature hyperspace. By taking a three-dimensional “slab” of this network activity function, for example, a slab in the vicinity of mammal knowledge (Figure 2.2), we can achieve some insight into the order in the patterns of activity. The central, lowest energy point—the “centroid” of mammal knowledge—corresponds to the representation of a creature that best defines our sense of “mammalness”. Within the mammal basin, there are innumerable attractor sub-basins corresponding to specific mammals. Very close to the centroid are sub-basins corresponding to mammals likely to be very close to the centroid representation, e.g., dogs, cats, cows, and horses. Distance from the centroid is defined by the degree of atypicality, which is defined by feature and feature combination frequency within the mammal domain. Highly atypical animals, such as whales and platypuses, are represented near the periphery. Within any given sub-basin, there may be sub-sub-basins, for example, corresponding to types of dogs. The depth of the mammal basin and its sub-basins (the z-axis in Figure 2.2) is determined by the strength of encoding of knowledge in neural connectivity. This in turn is determined by the degree to which a given exemplar shares features with other exemplars in the domain, the frequency of the exemplar, and age of acquisition. The depth of a basin relative to that of its sub-basins reflects the degree to which features are shared by the sub-basins within that basin. The network’s settled activity state is most strongly influenced by the specific input features, which in most circumstances will absolutely define the sub-basin or sub-sub-basin into which the network settles (its position in state space), all the other factors exerting their major influence either on response latencies or the occasional errors. Errors will consist of slips into nearby sub-basins. This settling in response to input features instantiates content addressable memory. This capacity for content addressable memory, which is intrinsic to auto-associator networks supporting distributed representations, automatically accounts for the fact that with exposure to but scant features of a memory, we can instantly realize the full memory.

The effect of lesions (focal or diffuse) will be to produce graceful degradation of network performance. Network function does not simply cease. Instead, it becomes less reliable and more errorful, even as it continues to reflect the statistical regularities of remaining knowledge encoded in the network. Graceful degradation is another intrinsic property of population encoded representations. With network damage, deep basins will become shallower and sub-basins, particularly those that are shallower and more distant from the centroid—corresponding to more atypical exemplars—will disappear. As sub-basins become shallow or disappear, responses will reflect the settling of the network into surviving neighbors located nearer the centroid—neighbors of higher typicality (yielding coordinate errors, e.g., horse in lieu of donkey), the parent basin (yielding superordinate errors, e.g., animal in lieu of donkey), or failure to settle at all, yielding omission errors. This is precisely what has been observed in semantic dementia.

There is good evidence that in the brain, the meaning of a given word is distributed not over one network, as we have been discussing, but over a number of networks. This idea, which owes to Lissauer (1988) and Wernicke (as cited in Eggert, 1977), has recently been resurrected as the Embodied Cognition Framework (Buxbaum and Kalénine, 2010). In this conceptualization, the distributed representation of the concept “dog” has major components in the visual association cortices (incorporating knowledge of the visual appearance of dogs in general, as well as particular dogs); auditory association cortices (sounds that dogs characteristically make); the limbic system (one’s feelings about dogs); somatosensory cortex; olfactory cortex; frontal cortex (supporting a predicative component corresponding to our knowledge of what dogs do—a component of the semantic representation of a verb [see below]); and perisylvian...
language cortex, which enables us to translate the semantic representation of dog into an articulatory motor representation. The multiple component representation of concepts provides the basis for category specific naming and recognition deficits (Forde and Humphreys, 1999).

**Phonology**

We return now to Figure 2.1 to consider the acoustic representations–articulatory motor representations pathway. This is referred to as a pattern associator network because it translates representations in one form into corresponding representations in a different form. Acoustic and articulatory motor representations correspond to the acoustic and articulatory forms of phonemes, respectively. As conceptualized in the Wernicke–Lichtheim model, this is the pathway that supports repetition. In addition, because this network has acquired, through language experience, knowledge of the systematic relationships between acoustic and articulatory sequences, it has learned the statistical sound sequence regularities of the language: the phonemic sequences of joint phonemes (e.g., st, str), rhymes, syllables, affixes, morphemes and words characteristic of the language (Nadeau, 2001). I will use a reading model to illustrate the process (Plaut et al., 1996; Seidenberg and McClelland, 1989). It fundamentally recapitulates the acoustic–articulatory motor pathway of Figure 2.1, the major difference (inconsequential to this discussion) being that in place of acoustic representations, it employed orthographic representations. The three-layer pattern associator network was equipped with a learning algorithm and it was trained by presenting the orthographic representations of 3000 English single syllable words and their corresponding phonologic forms.

One of the most striking things about the trained model was that it also was able to produce correct pronunciations of plausible English nonwords (i.e., orthographic sequences it had never encountered before). This was possible because the model had learned the statistical relationships between sequences of graphemes and sequences of phonemes that are characteristic of the English language. To the extent that there is a limited repertoire of sequence types, the model was able to learn it and apply it. Certain sequences, those most commonly found in English single syllable words, were more thoroughly etched in network connectivity. Thus, it was very fast with high frequency words. It was also very fast with words with an absolutely consistent orthographic–phonologic sequence relationship, for example, words ending in “ust,” which are always pronounced /*)ust/* (must, bust, trust, lust, crust, etc.). The model encountered difficulty (reflected in prolonged reading latency) only with low frequency words, and only to the extent that it had learned different, competing pronunciations of the same orthographic sequence. Thus, it was slow to read pint because in every case but pint, the sequence “int” is pronounced /Int/ (e.g., mint, tint, flint, lint). It also was very slow with words that are unique in their orthographic–phonologic sequence relationship (e.g., aisle, guide and fugue). These behaviors precisely recapitulate the behavior of normal human subjects given reading tasks. The model was equipped to acquire the very limited set of sequences involving syllabic onset, nucleus, and coda. However, it provided proof of the principle that sequence knowledge in general can be acquired by a simply pattern associator neural network employing a hidden unit domain and units with nonlinear functions.

**Lexicons: Lexical-semantic knowledge**

The phonologic input lexicon is represented as the connections between the substrate for acoustic representations and the substrate for concept representations (Figure 2.1: pathway 6–5). The phonologic output lexicon is represented as the two pathways between the substrate for concept representations and articulatory motor representations (pathways 1–2 and 4–3). Evidence of the existence pathway 1–2 derives from patients with repetition conduction aphasia.
who demonstrate the ability to repeat by the semantic route but have no capacity for repeating nonwords (Nadeau, 2001). Evidence of the existence of pathway 4–3 comes from normal subjects, who make phonologic slips of the tongue, and from patients with reproduction conduction aphasia, who make phonemic paraphasic errors in both repetition and naming.

**Attractor basins, attractor trenches, and quasi-regular domains**

Implicit in the discussion thus far is an explanation of the mechanism by which regularities encoded in neural networks support language (Figure 2.3). In the final section of this chapter, it will become clear how this mechanism also constrains the generalizability of language therapies. In our discussion of concept representations, we considered a mathematical activity function in N-dimensional hyperspace that, depending on the particular pattern of input, would yield a distributed representation of any entity we know about. The mammal attractor basin (Figure 2.2) is the product of a thought experiment in which we took a three-dimensional slice through the N-dimensional activity function in the vicinity of mammal knowledge. The shape of this complex surface reflects the strength of regularities wired into neural connectivity and shared, to varying degree, by all mammals. The mammal attractor basin corresponds in psychological terms to a quasi-regular domain. It is regular to the extent that all mammals share a set of common features. However, it is quasi-regular because no two mammals are exactly alike.

The same concept can be applied to sequence knowledge in pattern associator networks, e.g., acoustic–articulatory or orthographic–articulatory. Because pattern associator networks support a pathway from one representation to another, the regularities encoded in their neural connectivity support activity patterns that I have termed attractor trenches. These attractor trenches also correspond to psychological quasi-regular domains. The neural activity patterns generated by phonologic sequence knowledge supporting words with the rhyme “int” correspond to one such domain, this one containing two sub-trenches, one corresponding to

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**Figure 2.3** The relationship between neural network connectivity, attractor basins and trenches, and patterns of behaviour. (From Nadeau, 2012.)
lint, mint, stint, and tint, and one corresponding to pint. Figure 2.3 illustrates the fundamental principle that knowledge is represented in patterns of neural connectivity, these patterns define the shapes of attractor basins and attractor trenches, and these basins and trenches correspond to the psychological form of quasi-regular domains.

**Grammatic sequence knowledge**

Grammatic sequence knowledge is evident at two levels: 1) grammatic morphology, which subsumes roots, grammatic inflections (e.g., affixes), and phrase structure rules; and 2) sentence level sequence, that is, syntax. I have discussed the potential for a simple pattern associator network (e.g., Plaut et al., 1996; Seidenberg and McClelland, 1989), to acquire sequence knowledge. Models with intrinsically greater power to capture sequence knowledge have been developed, e.g., the simple recurrent network. In training such a network, a series of consecutive forms (e.g., morphemes comprising a word, words comprising a sentence) is presented, one at a time, to the input layer. Each form generates a pattern of activity in the hidden units and the output layer. However, an additional domain, termed context units, represents the pattern of activity of the hidden units generated in response to one or more prior forms. Equipped with a standard training algorithm, the network has the capability for learning the sequential relationships between the forms. The work of Joanisse and Seidenberg (2003) is exemplary. They reported a simulation involving a phonology-hidden units-context units semantics network in which, during training, connection strengths were adjusted on the basis of present input, target output, and data reflecting up to ten previous inputs, thereby potentially instantiating implicit knowledge of sequence regularities involving much longer sentences. The network, through its experience with the statistical regularities of the dataset, was able to acquire implicit knowledge of rules governing verb argument structure, subcategorization rules, preposition choice, and pronoun anaphora.

For over half a century, linguists have sought a deterministic account for language function and for nearly as long, psycholinguists and cognitive neuropsychologists have sought to explain grammatic breakdown in aphasia in terms of a defect in one or more components of such a mechanism. Here we offer a far simpler explanation: neural networks supporting population encoded representations and endowed with learning mechanisms, while capable of learning unique knowledge (e.g., aisle, guide, and fugue, as discussed above), have a particular facility for acquiring knowledge of statistical regularities, including sequence regularities, from experience. Furthermore, by virtue of the intrinsic properties of these networks, performance breakdown is characterized by graceful degradation.

The work of Thompson and colleagues (2003) is illustrative. They reported a syntactic treatment experiment that, although motivated by linguistic theory, enables us to relate the results of the Joanisse and Seidenberg (2003) simulation in a rather direct way to human acquisition of syntactic skills and to put this within the more general context of neural network support of language function. They trained four subjects with Broca’s aphasia to produce three sentence types, starting from active NP–V–NP (NP = noun phrase; V = verb) sentences (e.g., The thief chased the artist). The sentence types were: WH questions (Who has the thief chased?), object-clefts (It was the artist who the thief chased), and object relatives (The man saw the artist who the thief chased). The two participants trained to produce object relative sentences showed generalization to the production of object-cleft sentences and WH questions. The two participants trained to produce the WH questions showed no generalization to the other forms. These results supported the “complexity account,” in which treatment focused on more complex forms results in generalization to less complex forms requiring the same type of
movement (Thompson et al., 2003). However, an alternative explanation is that Thompson et al. were retraining an attractor trench supported by a recurrent pattern associator network with a sequence knowledge acquisition capacity similar to that of the network employed by Joanisse and Seidenberg and representing the general class of sequences expressed by the sentence types they studied. This network learned variations on a particular theme of sequential concept manipulation (a quasi-regular sequence domain) that could enable production of the correct sequence of words. Training the pattern associator with atypical (“complex”) exemplars retrained network connections that could support both the atypical sequence exemplars (e.g., object relatives) and exemplars close to the centroid of the attractor trench (e.g., WH questions). Retraining WH questions succeeded in retraining connections that could support sequences near the centroid but did little to tune connections that would support atypical exemplars more distant from the centroid.

**Verbs**

Although the function of verbs is traditionally conceptualized as predominantly grammatical, the population encoded neural network model conceptualizes their function largely in semantic terms. Consider this sentence:

“The old man shot the burglar.”

It is a simple matter to conceptualize “old man” and “burglar,” but what about “shot”? Shot can be represented as a modification of the “old man” and “burglar” distributed concept representations but not as its own discrete concept representation. “Old man” becomes “shooter old man,” and thus acquires the property of agency (a specific thematic role) and a “flavor,” that of a shooter rather than a sleeper or a raconteur. Correspondingly, burglar also acquires the property (thematic role) of patient and a flavor (e.g., lying prostrate bleeding). The verb here emerges as a peculiar sort of adjective. Although verbs, in and of themselves, do not correspond to distributed concept representations, they nonetheless have a distributed representation comprised of nominative, thematic, flavor, and implementational components.

The nominative component is defined by the connectivity between substrates for verb and noun representations. There is good evidence that these connections are extensive, as a given verb primes the nouns it is commonly associated with (Ferretti et al., 2001) and vice versa (McRae et al., 2005).

In theory, the thematic component should be represented in prefrontal cortex because prefrontal cortex supports the volitional formulation of plans precisely tailored to the demands of the situation (Nadeau and Heilman, 2007) and intention requires a role to be associated with the action taken. If you are planning, or are engaged in the plans of the actors you are thinking about, then you are imputing agency and consequence. Prefrontal cortex also provides the substrate for dynamic time sense. Extensive empirical studies demonstrating differential impairment in verb production with frontal lesions/Broca’s aphasia provide strong validation of this hypothesis.

The flavor component is supplied variously by manner, path, and limbic representations. The manner/path distinction is illustrated by the following:

The bird flew into the wind. (flew confers manner)
The bird flew over the house. (flew confers path)
Manner is instantiated in nuances of verb meaning, e.g., *walk, amble, stroll, saunter, strut, sashay, march*, and *hasten*. In manner incorporating languages (e.g., English, German, Russian, Swedish, Chinese), it is incorporated directly in the verb, whereas in path incorporating languages (e.g., Greek, Spanish, Japanese, Turkish, Hindi), it is incorporated in a prepositional phrase. The manner component of action verbs would logically be supported by cerebral cortex supporting movement perception, particularly visual—that is, in the vicinity of the posterior inferior temporal gyrus. Deficits in movement perception, action recognition, and action naming have been observed with lesions in this region. Functional imaging studies have also confirmed this hypothesis (Kemmerer *et al.*, 2008). The path component would logically be represented in parietal cortex because it involves movement across space. Functional imaging studies provide some support for this hypothesis as well (Wu *et al.*, 2008). Any verb might have a limbic component to its representation but this is most obviously the case for emotion verbs.

There is abundant evidence that the implementational component of action verb meaning is represented in motor cortex. Reading sentence segments involving movement is slowed when the motion conveyed conflicts with concurrently performed movement. The implementational component is markedly attenuated when the meaning of a verb is changed from ongoing to complete action (i.e., from progressive to perfect aspect). Patients with Parkinson’s disease exhibit robust masked priming of both nouns and verbs in the on-condition (when motor cortex function is optimized, e.g., by the administration of dopaminergic drugs), but prime only nouns in the off-condition.

**Application of the model to aphasia**

The following is a brief sketch of the mechanisms of aphasia viewed through the lens of this model. The signature linguistic manifestation of Broca’s aphasia, simplification of syntax to the point of simple declarative sentences or single words, can be substantially related to attenuation of sentence level sequence knowledge attractor trenches, such that neural connectivity is no longer sufficient to support atypical sequences. Impairments in grammatic morphology differ from language to language. In heavily inflected languages (i.e., substantially, all the major languages of the world except English and Chinese), there is evidence of sufficient redundant grammatic morphologic sequence knowledge in the non-dominant hemisphere that grammatic morphemes are infrequently omitted but are susceptible to substitution (grammatic morphologic paraphasic errors) (reviewed in Nadeau, 2012). It is the poverty and inconsistency of English grammatic morphology, reflective of poor encoding of regularities in neural connectivity, that sets the stage for widespread omission of grammatic morphemes. We have already noted the consequences of the loss of the thematic component of verb representations. Many features of Broca’s aphasia, most notably the limited ability to modify distributed concept representations with serial adjectives and the particular difficulties with two- and three-argument verbs, can be attributed to impairment in working memory.

All perisylvian aphasias (Broca’s, conduction, Wernicke’s) are characterized by disorders of phonologic sequence. With anterior lesions, these reflect attenuation of phonemic sequence attractor trenches, which predisposes to near-miss phonemic slips that tend to be less marked (represented nearer the centroid). With more posterior lesions, paraphasic errors are substantially influenced by disruption of phonemic neighborhoods by damage to connections between semantic and phonologic substrates (i.e., loss of lexical–semantic knowledge), leading to larger substitutions that may share both semantic and phonologic relationships to the target, or no apparent relationship at all, as is often the case in neologistic jargon aphasia.
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Posterior perisylvian aphasias (Wernicke’s and, to some degree, conduction) are characterized by anomia, reflecting in part disconnection of semantic and phonologic substrates by the ischemic lesion, but also degradation of phonologic and/or semantic substrates (see Treatment, below). Wernicke’s aphasia is additionally associated with damage to the substrate for the phonologic input lexicon, hence problems with comprehension.

Transcortical sensory aphasia likely reflects the consequences of semantic–phonologic disconnection in the context of preserved phonologic, morphologic, and sentence level sequence knowledge. Language may be fluent and characterized by complex syntax but it is substantially devoid of major lexical items.

Treatment

This model has strong implications for aphasia treatment, and most particularly, for generalization of treatment effects to untrained items and everyday verbal communication. Consider first the treatment of anomia, the single most common and disabling feature of aphasia. From the foregoing, it is apparent that because the relationship between word meaning (semantics) and word form (phonology) is largely arbitrary (except for onomatopoeic and derivational forms), there are very few regularities in the pattern associator networks linking the substrates for semantics and phonology—regularities that might be further developed through therapy, thereby deepening attractor trenches and re-establishing sub-trenches. If you have trained a patient to name 30 words, the knowledge gained is of no value in naming the thirty-first. This theoretically motivated prediction finds strong support in the results of a recent meta-analysis, which indeed demonstrated no generalization of commonly used treatments for anomia (Wisenburn and Mahoney, 2009). However, to the extent that damage to substrates for phonologic sequence knowledge or semantics contribute to anomia, training of these networks, which do contain extensive regularities, could potentially improve lexical access to untrained exemplars.

Kendall and colleagues (2008, 2015) have just reported the results of a trial of phonologic sequence therapy, a derivative of the Lindamood Phonemic Sequencing Program (LiPS) (Lindamood and Lindamood, 1998), in 26 subjects. The first phase of treatment consisted of developing the neural substrate for linked distributed representations of individual phonemes (see Figure 2.1). For example, at the end of completely successful training, insertion of the acoustic form of /b/ into the acoustic domain (by saying /b/ to the subject) would instantly lead to generation of distributed representations of the articulatory form of /b/, a concept of /b/, the sound of /b/, and an orthographic representation corresponding to the letter b. The concept of /b/ is formed in a variety of ways: labeling it as a “lip-popper,” the tactile feel of the patient’s larynx during phonation, a picture of a sagittal section through the head with mouth and oropharynx positioned for production of /b/, and the patient’s recalled image of her own mouth, reflected in a mirror, as she produces the phoneme. The second phase of treatment trains the regularities of English phonological sequences by inserting 1–3 syllable nonwords and some real words. Three months after completing training, participants demonstrated an absolute gain in untrained real word naming of 5.28 percent (p = 0.002, d = 0.70) and an even greater gain in untrained nonword repetition.

Other treatment studies have provided evidence of broad generalization of treatment effects for semantic (Edmonds et al., 2009) and syntactic therapies (Thompson et al., 2003). In addition, syntactic therapies are intrinsically generalizing to the extent that successfully acquired sequence knowledge (e.g., how to produce an object relative sentence) applies to all concepts and words in the language.
Generalization is traditionally conceptualized as the process of translating treatment effects to daily verbal communication. However, it can also provide a useful therapeutic tool. Plaut (1996) demonstrated, in a computer simulation of “rehabilitation” of a damaged lexical-semantic network, that training with atypical exemplars was preferable to training with typical exemplars. Training atypical exemplars instantiates both knowledge of features unique to atypicals and knowledge shared with typicals, but not the other way around. Subsequently, Kiran and Thompson (2003) confirmed these results in a study of semantic treatment of human subjects with aphasia, and in their syntactic treatment (Thompson et al., 2003). In the phonologic sequence therapy tested by Kendall et al. (2015), only atypical phonological sequences were trained with a view to reducing therapy time and there was robust generalization to untrained sequences.

Conclusion

In this brief review, we have seen how a multi-component neural network supporting population encoded semantic, acoustic and articulatory motor representations, and pattern associator networks supporting the lexical–semantic knowledge instantiating the phonologic lexicons and phonologic, morphologic, and sentence level sequence knowledge, can provide a plausible account for language function. The operation of this network is constrained by regularities encoded in neural network connectivity on the basis of the statistics of language experience. These regularities define a neural activation landscape consisting of attractor basins and attractor trenches, which in turn support quasi-regular knowledge domains. Aphasia, whether due to focal brain injury (e.g., stroke) or degenerative disease (e.g., semantic dementia) reflects graceful degradation of knowledge that corresponds to loss of depth and breadth of basins and trenches. Rehabilitation of aphasia that generalizes to untrained exemplars and daily verbal communication corresponds to further development of residual regularities in neural connectivity such that basins and trenches are deepened and sub-basins and sub-trenches redeveloped. Generalization cannot be achieved by therapies directed at networks (e.g., lexical–semantic) that do not support substantial regularities. The neural network model discussed here provides the potential for rekindling the dialectic between neuroanatomy and neurophysiology on the one hand and language function and rehabilitation on the other.

References

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