

Ageing, Plasticity, and Cognitive Reserve in Connectionist Networks

Michael S. C. Thomas (m.thomas@bbk.ac.uk)

School of Psychology, Birkbeck College, University of London
Malet Street, London WC1E 7HX, UK

Abstract

Neurocomputational modeling has suggested that a range of mechanisms can lead to reductions in functional plasticity across development (Thomas & Johnson, 2006). In this paper, we consider whether ageing might also produce a reduction in plasticity. Marchman's (1993) model of damage and recovery in past tense formation was extended to incorporate the two main proposals for implementing effects of ageing: altered neuromodulation and connection loss. Simulations showed that ageing did reduce plasticity (as assessed by the system's ability to recover from damage) but that effects were modulated by (a) the mechanism used to implement ageing, (b) problem type, and (c) pre-existing levels of cognitive reserve.

Keywords: Ageing; plasticity; compensation; critical and sensitive periods; brain damage; recovery; cognitive reserve.

Introduction

Research on plasticity is typically informed by three sources of empirical evidence: (i) the rate and upper limit of behavioral change at a given age; (ii) the effects of early deprivation on subsequent development; and (iii) recovery from damage at different ages. Several findings have emerged from work on cognitive development (Thomas & Johnson, 2008). First, because plasticity is rarely ever eliminated in older individuals, the term *critical period* has been replaced by *sensitive period* for age-related changes in development. Second, there are multiple varieties of sensitive period ranging from sensory processing to high-level cognitive abilities, each with a different profile. Third, multiple neurocomputational mechanisms may underlie these changes, including *entrenchment*, *pruning*, *competition*, and *assimilation* (Thomas & Johnson, 2006). Finally, many sensitive periods may be a consequence of the basic processes underlying postnatal functional brain development.

In this paper, a neurocomputational perspective is used to consider potential changes in plasticity associated with *ageing*. Recovery from damage is used as the metric of functional plasticity, including a distinction between acute (short-term) and chronic (long-term) phases of recovery. Additionally, there is consideration of the implications of *cognitive reserve* (pre-existing computational capacity) for modulating both the expression of ageing pathology in overt behavior and recovery patterns following damage (Stern, 2002).

Ageing and plasticity

A complex relation exists between ageing and cognition. Cognitive decline, sufficient for a clinical diagnosis of mild cognitive impairment, may not be a necessary outcome of

ageing, and decline itself may be a heterogeneous category, including potentially unrecognized pathologies such as Alzheimer's disease, vascular dementia, and cerebrovascular disease, as well as normal ageing processes (Lindeboom & Weinstein, 2004). Nevertheless, in later adulthood, there are consistently observed reductions in the speed and accuracy of elementary cognitive operations such as working memory (Lindenberger & Baltes, 1997). While older adults are still able to profit from learning, they require more practice and time to achieve performance improvements equivalent to younger adults and older adults may exhibit a reduced ceiling of performance (Baltes & Kliegl, 1992; Baltes & Singer, 2001). However, older adults may continue to display high performance levels in particular skills via a combination of selection, optimization, and compensation (see Baltes & Singer, 2001). For example, individuals may retain ability by increased practice for a narrow set of skills, or they may rely on crystallized knowledge (experience) rather than on-line fluid processing capacity. Compensation in this sense implies using a different method (and potentially, different cognitive systems) to achieve the same task.

When plasticity is assessed via the ability to recover from damage, it is frequently argued that young children show better recovery from focal brain damage than adults (see, e.g., Anderson, Northam, Hendy & Wrennall, 2001; Bates & Roe, 2001). Fewer distinctions are drawn between different phases of adulthood. However, since the majority of strokes occur to individuals already in their sixties, it is important to understand the relationship between plasticity and ageing to assess the potential for recovery in older adults.

Cognitive reserve

The notion of cognitive reserve stems from the observation that there is no direct relationship between the severity of brain damage and the behavioral impairment exhibited by an individual. It is hypothesized that in part, variability in this relationship may be explained by pre-existing differences in people's level of *cognitive reserve* (Stern, 2002). This might take the form of some reserve capacity of the brain (such as brain size or synapse count); or a cognitive level variable, such as a disposition to use functional architectures that are more efficient or more flexible and so more resilient to damage (Stern, 2003), e.g., bilateral rather than unilateral functional networks. The concept has been applied both to an individual's susceptibility to cognitive decline in ageing and to children's potential to recover from early acquired brain damage (Dennis, 2000). Reserve is thought to be affected both by genetic and environmental factors. Proxy measures for cognitive reserve include pre-morbid IQ, edu-

cational level, occupational attainment in adults, physiological measures such as head size, and neural measures such as activation in fMRI paradigms (Staff et al., 2004). A recent longitudinal study indicated that initially normal adults who later exhibited mild cognitive impairment had decreased gray matter volumes in their temporal lobes *prior* to exhibiting clinical symptoms (Smith et al., 2007). Reduced gray matter could be viewed as an indicator of lower cognitive reserve, increasing susceptibility to damage during ageing. The key idea is not that reserve protects against the development of pathology per se but that it delays its behavioral manifestation. Compensation in this sense occurs within the same system as it adapts to preserve behavior.

Neurocomputational models of ageing

Artificial neural network models have been used to simulate cognitive decline through ageing, on the grounds that these models incorporate some of the constraints likely to influence the efficiency of neural processing. Two main parameter manipulations have been explored. The first proposes that ageing operates through a change in neuromodulation (see Li, Lindenbergh, & Sikstrom, 2001, for a summary of evidence and findings). This produces a reduction in the *gain* of simple processing units. Specifically, units use a sigmoid activation function to translate their summed input into an output value. The slope of the function becomes shallower so that units are less able to make large changes in output for small changes in input; hence they lose sensitivity (see Raudys, 2007, for related work). The second approach proposes that ageing involves a reduction in synaptic density, equivalent to eliminating connections and perhaps internal processing units from a network (see e.g., Alvager et al., 2003). Other manipulations have also been used, e.g., simulating Alzheimer's disease in terms of runaway synaptic modification in hippocampal structures (Hassmelmo & Wyble, 1996). Still other manipulations are conceivable, such as increasing weight decay or transmission noise.

The following simulations focused on the two main manipulations: neuromodulation and connection loss. The aim was to assess how these ageing-related parameter changes would affect the plasticity of a learning system with all other factors held constant (i.e., assuming no other parameters change across lifetime). Functional plasticity was measured in terms of the network's ability to recover from late acquired damage.

Simulations

For the base learning system, Marchman's (1993) model of acquisition, loss, and recovery in associative networks was extended to consider ageing. This is an influential model much cited for its demonstration of sensitive periods in recovery (e.g., Elman, 2005). Acute recovery from damage caused by a lesion to network connections became increasingly slow with greater training (where level of training was taken as a proxy for the age of the system). The simulations employed the well-understood domain of English past tense formation, which has often served as a test bed to

illustrate the importance of the frequency and consistency of associative mappings in shaping performance. These features are argued to influence many aspects of cognitive development (e.g., Bates & MacWhinney, 1987, for wider arguments in language development). The English past tense is of note because it is characterized by a predominant rule (e.g., *talk-talked*, *drop-dropped*, etc.) that extends to novel stems (e.g., *wug-wugged*), but also contains irregular verbs of different types (*go-went*, *hit-hit*, *sing-sang*). When Cortese et al. (2006) explored past tense production in a group of healthy ageing adults (n=67, mean age 77 years), they found greater performance decrements for inconsistent and irregular past tense forms. The performance pattern was additionally modulated by diagnosis of semantic or Alzheimer's type dementia.

For our purposes, the past tense serves as a useful test domain because it allows us to examine the effects of problem type on recovery profile, and in particular the effects of consistency, type frequency, and token frequency on functional plasticity. The regular past tense has the highest type frequency and forms a consistent set of mappings (reproduce the stem, add an inflection). The different classes of exception verb fall on a continuum of inconsistency, with no-change past tenses (*hit-hit*) least inconsistent (reproduce the stem but don't add an inflection), vowel change past tenses (*sing-sang*) at an intermediate level (partly reproduce the stem, no inflection), and arbitrary past tenses (*go-went*) most inconsistent (no relation between stem and past tense). Arbitrary past tenses have the lowest type frequency but require the highest token frequency in order to be acquired.

The simulations had a modest objective. Marchman's model was relatively simple, both in its topology (a 3-layered feed forward network) and in its learning (the back propagation rule). It was a cognitive model rather than a model of a language-related brain area, and its assumptions have restricted biological plausibility. Nevertheless, it provides a transparent framework in which to consider the direct consequences of three factors on functional plasticity: ageing mechanism, pattern consistency, and cognitive reserve.

Design

We assessed the functional plasticity of this system by measuring its recovery from damage at different points in training. In the normal condition, a network was trained for 500 epochs, where an epoch is a presentation of the whole past tense training set. Five hundred epochs was defined as the network's *lifetime*. The network was lesioned between 400 and 500 epochs, that is, in the last 20% of its lifetime. Lesions were applied to the network at 400, 420, 440, 460, 485, 490, or 495 epochs by probabilistically removing 50% of the weights. Its recovery profile was then assessed in three ways: (i) the improvement in performance after the first epoch of retraining post-lesion was recorded as a measure of *acute* recovery. This recovery was benchmarked against the speed of learning on the first epoch of the network's lifetime; to assess *chronic* recovery (ii) the net-

work's level of performance at the end of its lifetime was recorded; this means that the later a lesion occurs, the shorter time the network has to recover. Nevertheless, this confound reflects reality; and (iii) the network's performance given a fixed period of recovery irrespective of time of lesion (500 epochs) was recorded in order to assess the maximum recovery possible in the system.

Ageing was implemented in two ways. Under the *neuromodulation* hypothesis, the gain parameter of the network was reduced in steps from a normal value of 1 to a value of 0.5 at epoch 430, to a value of 0.25 at epoch 450, and a value of 0.125 at epoch 475. Under the *connection loss* hypothesis, network connections were probabilistically lesioned ($p=0.2$) at 430 epochs, again at 450 epochs, and a final time at 475 epochs. Plasticity was always assessed at least 10 epochs following a parameter manipulation to give the network an opportunity to adapt to the alteration in conditions. The schedule is illustrated in Figure 1.

Figure 1: Schedule of parameter manipulations to implement network ageing. Points at which functional plasticity was assessed via damage and recovery are shown with vertical dotted lines. Network lifetime was 500 epochs.

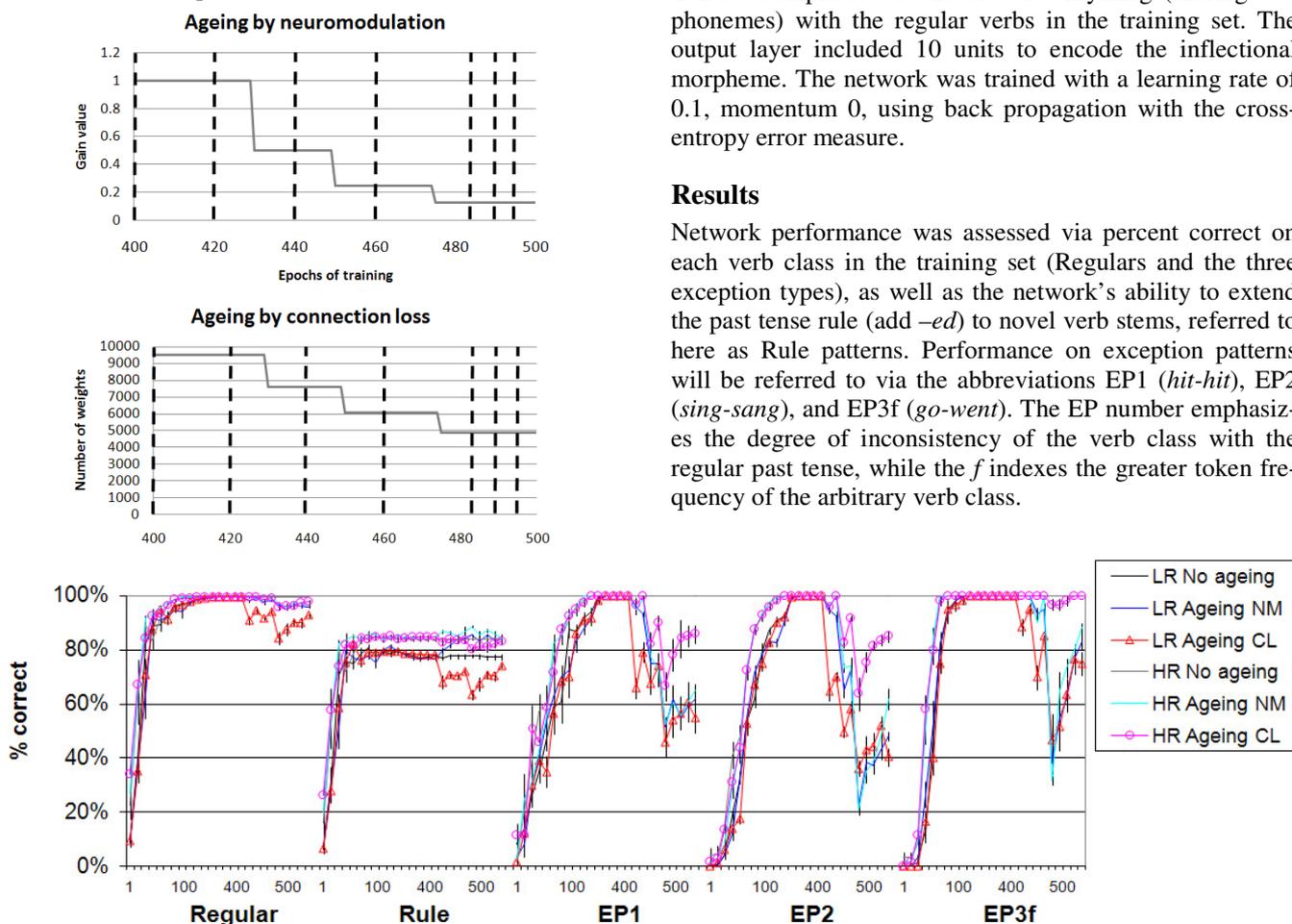


Figure 2: Developmental profiles of low reserve (LR) and high reserve networks (HR) for conditions of No ageing, Ageing implemented via neuromodulation (NM), and Ageing implemented by connection loss (CL), for each verb type.

The basic architecture of the system was a 3-layer feedforward network (90 input units, 100 output units). *Cognitive reserve* was implemented by calibrating the number of internal (hidden) units to a level just sufficient to allow successful learning, in this case 50 internal units. This formed the low reserve (LR) condition. This value was then doubled to 100 for the high reserve (HR) condition.

Finally, several control conditions were implemented. These included running the model in the absence of ageing; applying the most severe level of ageing manipulation to the startstate of the network (a kind of 'early onset ageing') to explore whether the system could overcome this sub-optimal parameter setting given sufficient time; and applying a lesion to the startstate of networks that would later experience ageing, to investigate whether early damage would predispose the network to suffer more severe effects from the subsequent ageing process.

The training set was an artificial language comprising tri-phonemic verbs stems represented using an articulatory feature-based phonological code (30 bits per phoneme). There were 410 regular verbs (adding *-ed* to form past tense); irregular verbs were of three types: 68 vowel change verbs, 20 no-change verbs, and 10 arbitrary verbs. The generalization set comprised 410 novel verbs rhyming (sharing two phonemes) with the regular verbs in the training set. The output layer included 10 units to encode the inflectional morpheme. The network was trained with a learning rate of 0.1, momentum 0, using back propagation with the cross-entropy error measure.

Results

Network performance was assessed via percent correct on each verb class in the training set (Regulars and the three exception types), as well as the network's ability to extend the past tense rule (add *-ed*) to novel verb stems, referred to here as Rule patterns. Performance on exception patterns will be referred to via the abbreviations EP1 (*hit-hit*), EP2 (*sing-sang*), and EP3f (*go-went*). The EP number emphasizes the degree of inconsistency of the verb class with the regular past tense, while the *f* indexes the greater token frequency of the arbitrary verb class.

Six replications of each condition were run with different random seeds determining initial connection weights, pattern presentation order, and probabilistic lesioning. Mean results are reported averaged over these replications, along with the standard error of the mean.

Developmental trajectories with and without ageing

Both low reserve and high reserve networks reached ceiling performance by 200 epochs of training. By 400 epochs, performance was therefore well entrenched. Figure 2 depicts these developmental trajectories, focusing on initial and latter parts of training. The following points are of note. After 400 epochs, without ageing, performance remained at ceiling. With ageing, performance declined most markedly on exception verbs. Decline was exaggerated by inconsistency of mappings (EP2 worse than EP1, EP1 worse than Regular) but attenuated by high token frequency (EP3f better than EP3). High reserve protected the system most when ageing was implemented via connection loss. For ageing via neuromodulation, high reserve only protected the system against the decline of the consistent, high type frequency regulars. The effects of reserve therefore interacted with the mechanism by which ageing operated.

Acute recovery from damage Two points of damage, at 400 and 420 epochs, preceded the onset of ageing, while the remaining points of damage occurred following different levels of parameter change, 440 (mild ageing), 460 (moderate ageing), 480, 490, and 495 (severe ageing). Following damage at each of these 7 points, we assessed recover after 1 epoch of training, expressed as a difference score compared to how much was learned in the network’s first epoch of life. For example, if a network achieved 30% accuracy on regular verbs by the end of the first epoch of its lifetime, it might reasonably be expected to reach 30% after 1 epoch of recovery following damage – akin to starting from scratch. If it acquires less, say 20%, its functional plasticity must be reduced. Plasticity is represented via the difference between these two numbers (20%-30% = -10%). Figure 3 demonstrates the respective plasticity in the initial recovery phase, for the systems with No Ageing, Ageing via neuromodulation, and Ageing via connection loss, for low reserve and high reserve conditions, respectively.

For the low reserve condition, the acute recovery phase

showed little difference in starting from scratch. For No Ageing and Ageing via connection loss, there was no modulation by pattern type. By contrast, Ageing via neuromodulation demonstrated *increased* plasticity for more consistent patterns (Regular, Rule, and EP1). This occurred because changes in gain served to reduce the entrenchment of consistent mappings, so that any preserved knowledge of these mappings after damage could be utilized.

For the high reserve conditions, both the No ageing and Ageing via neuromodulation conditions produced heightened plasticity for more consistent mapping, reflecting preserved knowledge useful during retraining. As with the LR condition, a change in gain exaggerated effects of consistency. However, for the Ageing via connection loss condition, the greater the severity ageing, the more the reserve was reduced and the more it resembled the LR condition, i.e., with no retraining advantage. Note that functional plasticity was never negative in Figure 3 – that is, retraining was not generally slower than starting from scratch. However, the peak functional plasticity of this system occurs around epoch 50. Compared to that reference point, functional plasticity was greatly reduced by entrenchment. These results are therefore consistent with those of Marchman (1993).

Chronic recovery from damage Figure 4 displays the level of longer-term recovery that could be expected for the networks in the different ageing conditions. In all cases where age of lesion was confounded with recovery time (i.e., the older the lesion, the shorter the recovery time [white bars]), prospects for recovery declined with increasing age. When this confound was removed [black bars], the No-ageing condition indicated that networks could recover whatever their age of damage. However, for the low reserve network, ceiling performance was no longer attainable, because the reserve had fallen below a level that could support normal function. Functional plasticity was therefore limited by capacity in this case.

For the low reserve condition, Figure 4 demonstrates that ageing impacted more severely on recovery of the inconsistent mappings, with loss of connections having the greater effect. However, prognosis for recovery was greater if ageing was caused by neuromodulation. In particular, high token frequency (that is, intense practice of a narrow range of

Figure 3. Acute (short-term) recovery rates from damage occurring at increasingly later ages

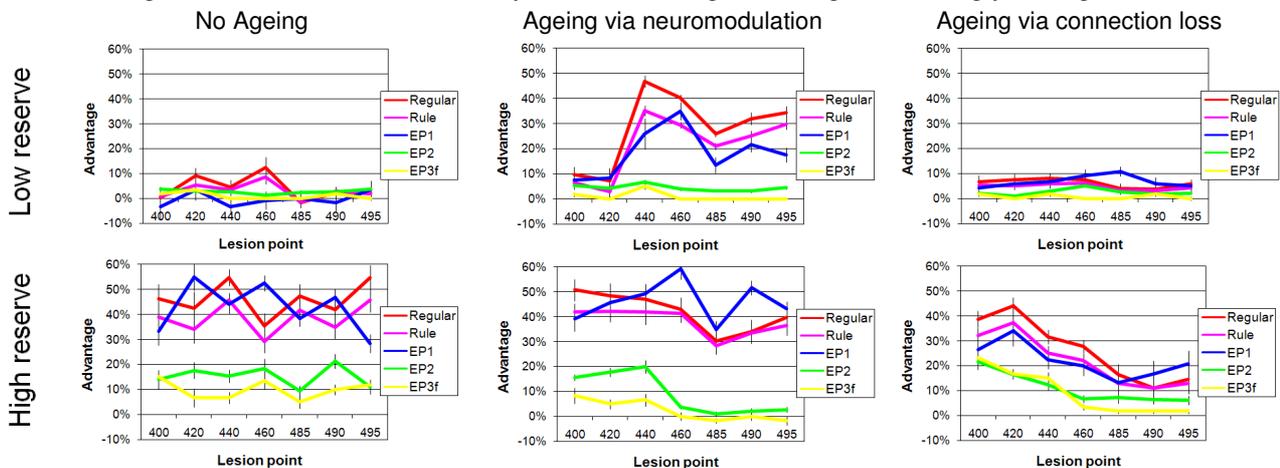
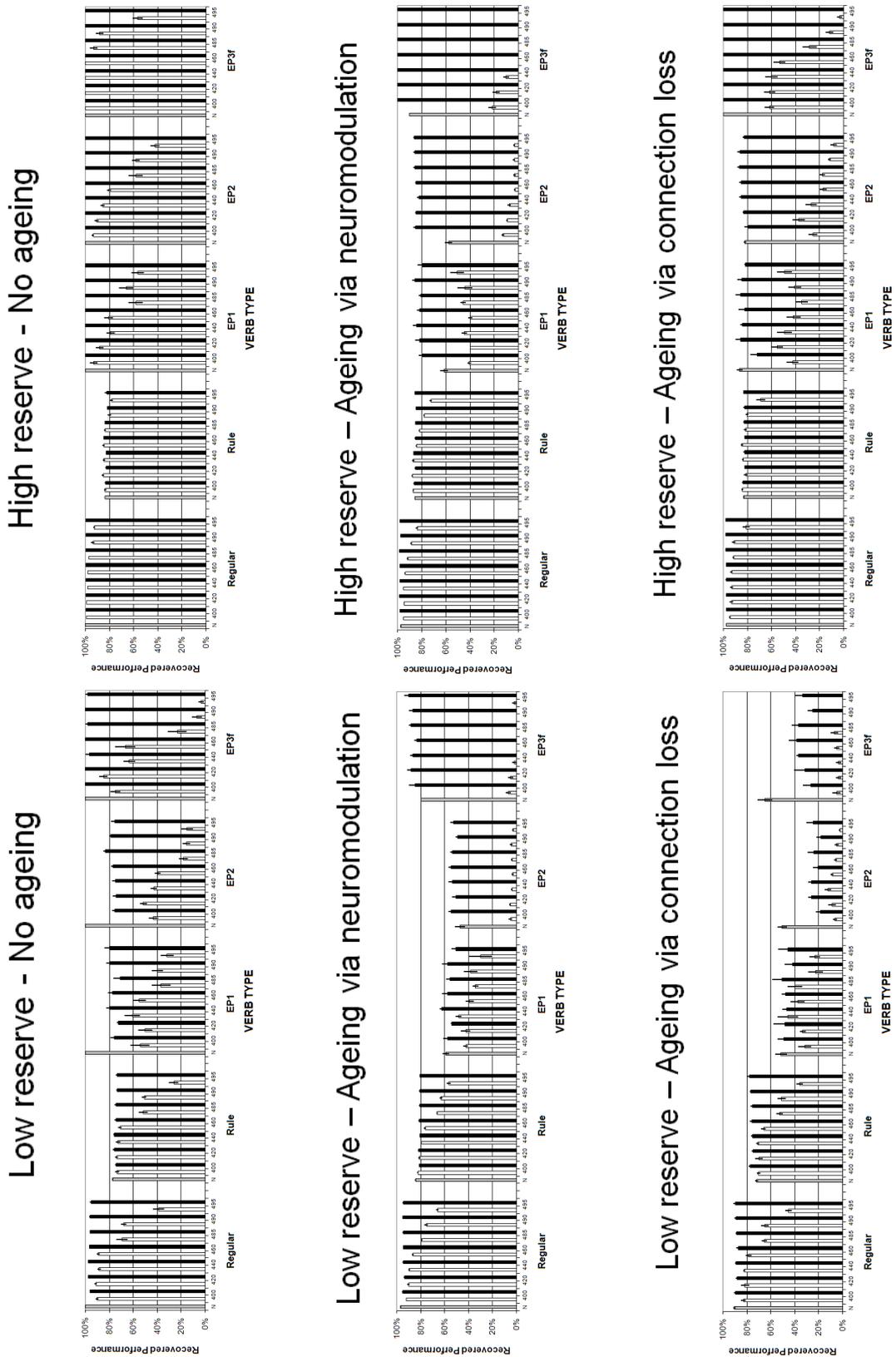


Figure 4. Chronic (long-term) recovery outcome after a lesion. The first bar in each verb class series (gray bar) depicts end-state performance without a lesion. The white bars then depict recovered performance at end-of-life for lesions occurring at increasingly late stages. The black bars indicate recovered performance for lesions occurring at these points but for networks given a fixed recovery period (500 epochs); old networks therefore had an extended life to allow for recovery time.



items) produced good recovery. For connection loss, low consistency patterns were never recovered. For both ageing conditions, the consistency and high type frequency of the regulars signaled good recovery. Plasticity during ageing, then, was highly dependent on problem type. For the high reserve condition, when a fixed recovery period was used, both ageing conditions showed good recovery, with high type and token frequency again aiding performance. Unlike the low reserve condition, recovery now reached ceiling performance. Over the longer term, plasticity in ageing was protected by high cognitive reserve.

Control conditions Two control conditions were run to place the ageing conditions in context. First, networks were run with the most severe ageing manipulation applied to the startstate – could the network compensate for this deficit over a lifetime? The answer was yes, but only completely in the case of systems with high cognitive reserve. Second, would networks that had experienced damage early in their lives (from which they had subsequently shown good recovery) exhibit increased sensitivity to ageing? The answer was yes, even in the case of high reserve.

Conclusion

First, as with the empirical data of Cortese et al. (2006), the simulations showed that inconsistent information is more vulnerable to loss in ageing. Second, both ageing manipulations (neuromodulation, connection loss) reduced functional plasticity in terms of the network's ability to recovery from damage. Additionally: (1) High cognitive reserve protected the system from behavioral impairment and loss of plasticity, so that there was no direct relationship between damage and behavior. However, its action depended on manipulation type, implying that cognitive reserve may be pathology specific. (2) Properties of the knowledge stored (the consistency and frequency of verb classes) modulated recovery pattern. (3) The model generated a novel prediction that ageing by neuromodulation might attenuate entrenchment effects, as observed in acute recovery. Further work is necessary to validate the generality of the current results by extending the simulations to alternative architectures and cognitive domains.

Acknowledgments

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Appendix

Table 1: Sample Endstate performance (lifetime). LR = low cognitive reserve; HR = high cognitive reserve, se = standard error of the mean over six replications.

Condition			Regular	Rule	EP1	EP2	EP3f
No Ageing	LR	Mean	100%	77%	100%	100%	100%
		se	0%	1%	0%	0%	0%
	HR	Mean	100%	84%	100%	100%	100%
		se	0%	0%	0%	0%	0%
Ageing Neuromodulation	LR	Mean	97%	85%	59%	47%	80%
		se	0%	1%	3%	4%	0%
	HR	Mean	98%	86%	62%	58%	90%
		se	0%	0%	3%	3%	0%
Ageing Connection loss	LR	Mean	90%	72%	52%	50%	65%
		se	1%	1%	5%	4%	6%
	HR	Mean	98%	83%	87%	82%	100%
		se	0%	1%	3%	1%	0%
Early onset ageing Neuromodulation	LR	Mean	99%	81%	87%	90%	100%
		se	0%	1%	3%	1%	0%
	HR	Mean	99%	84%	96%	99%	100%
		se	0%	0%	2%	0%	0%
Early onset ageing Connection loss	LR	Mean	97%	75%	81%	83%	100%
		se	1%	1%	2%	2%	0%
	HR	Mean	100%	84%	100%	100%	100%
		se	0%	1%	0%	0%	0%
Ageing Neuromodulation + early damage	LR	Mean	94%	85%	48%	10%	23%
		se	1%	1%	2%	1%	6%
	HR	Mean	96%	87%	52%	22%	42%
		se	0%	1%	3%	2%	2%
Ageing Connection loss + early damage	LR	Mean	78%	66%	44%	13%	10%
		se	2%	3%	9%	2%	5%
	HR	Mean	94%	83%	64%	41%	72%
		se	1%	2%	5%	3%	4%