Cognitive reserve and intelligence: Modulating the effects of damage in ageing dynamical systems

Frank D. Baughman[1] (frank.baughman@curtin.edu.au)
Natalie Baughman[2] (natalie.baughman@uwa.edu.au)
Simon A. Mills[1] (simon.mills@curtin.edu.au)
[1]Curtin University, School of Psychology, Bentley, Perth 6102
[2]University of Western Australia, School of Psychology, Crawley, Perth 6009

Abstract

The term cognitive reserve (CR) is used to describe the lack of direct relationship between the severity of brain damage, or pathology and subsequent levels of observed impairments. It has been suggested by Stern (2009), that CR may reflect differences in (a) pre-existing levels of some reserve capacity of the brain (the *passive* form); or, (b) differences in the underlying functional architectures supporting cognitive processes (the *active* form). In this paper, we explore two implementations of cognitive reserve that seek to target both these forms, extending recent work using dynamical systems framework (Baughman & Thomas, 2008; van der Maas et al., 2006). We examine how variability in cognitive reserve may modulate the effects of damage, at different levels of intelligence. The resulting simulations showed that level of intelligence does not differentially modulate the pattern of cognitive change following complete destruction of a single cognitive process, but that the effects of damage are proportionate across each level of intelligence. Following the two implementations of cognitive reserve that we tested, we found: (1) higher levels of connectivity within a given architecture resulted in greater spread of damage and lower endstate performance; and, (2) functional architectures that are characterized by greater specialization of function, rather than distributed function, differentially protected against the effects of damage, with these models also exhibiting better recovery.

Keywords: Cognitive reserve; intelligence; ageing; damage; recovery; dynamical systems; functional architecture.

Introduction

The term *cognitive reserve* (CR) is often used in relation to the pattern of general cognitive decline found in normally ageing adults, and to the more extreme forms of cognitive breakdown seen following brain damage (e.g., stroke), or disease (e.g., dementia and Alzheimer's). In healthy ageing adults, the term is used to refer to the variability observed between individuals of the same age. In clinical samples, the term refers to the observation that levels of brain damage, or pathology have no clear relationship to the severity of subsequent impairments. This is to say, two individuals with similar levels of brain damage may exhibit different cognitive profiles (e.g., the impairments for one individual may be subtle, while for the other they may be much more pronounced). The lack of direct relationship between the degree of pathology, or brain damage and clinical manifestation, has led to the suggestion that individuals differ with respect to their pre-existing levels of cognitive reserve (Stern, 2002; Stern, 2009).

A number of studies have reported mixed findings concerning the extent to which factors such as ones levels of intelligence, educational attainment, occupation and activity are associated with reduced risk of dementia, stroke, and lower levels of general decline (Kaplan et al., 2009; Koenen et al., 2009; Nithianantharajah & Hannan, 2009; Tucker-Drob, Johnson, & Jones, 2009; Whalley, Deary, Appleton, & Starr, 2004; Zahodne et al., 2011). However, these studies have not yielded causal accounts detailing how variability in CR may directly influence cognitive performance. Theoretical accounts of CR have however distinguished two broad forms (Stern, 2009). The *passive model* posits that CR may be delivered through differences in pre-existing reserve levels of some capacity of the brain (e.g., this might be number of neurons, or number of connections). Under this view, damage to a cognitive system with lower pre-existing levels of capacity, will lead to poorer outcomes, compared to cognitive systems where these levels are higher. The *active model* describes that differences in CR may be explained by differences in functional architectures underlying cognition. Under this view, it is hypothesized that some functional architectures are more efficient, and thus more resilient to the effects of damage, than others (Stern, 2009). Computational approaches provide an ideal platform from which to examine these issues because they provide an explicit framework for testing how various neurocomputational properties may directly lead to changes in a cognitive system. Here, we describe one approach using dynamical systems theory which aims to capture a broad pattern of development across a range of cognitive profiles and which allows for the consequences of damage to be assessed at the level of the whole cognitive system and across time.

Computational approaches to the study of ageing and damage

Computational studies to ageing, and to damage in ageing systems, have mostly focused on the effects of variation to three main parameter manipulations: (1) reducing the slope of gradients in activation functions (Li, Von Oertzen, & Lindenberger, 2006); (2) reducing the connectivity between processes (Alstott, Breakspear, Hagmann, Cammoun, & Sporns, 2009); and, (3) removal, or deletion of processing units to simulate neuronal death (Rubinov, McIntosh, Valenzuela, & Breakspear, 2009). The effects of these parameter manipulations can be subtle and varied. However,

their effects are generally that they show reduced levels of performance, require that networks need more time to learn (akin to older adults needing more time to learn, compared to younger adults) and result in a more protracted process of recovery, following damage. Individual differences in ageing and damage within a cognitive system with more CR might thus be explained by: (a) steeper gradients in the activation functions; (b) a greater number of pre-existing levels of connections (or, weights) between processing units; or, (c) lower rates of cell death.

Thomas (2008) recently examined issues concerning ageing and cognitive reserve within a connectionist model of English past tense learning. In these simulations, aging was implemented separately via: (1) a reduction of gradient in processing units; and, (2) a reduction (loss) of connections. CR was implemented via manipulating the number of hidden units within the model. Specifically, low cognitive reserve models were assigned 50 hidden units (a level just sufficient to allow the model to learn) and high cognitive reserve models were assigned 100 hidden units. Damage, applied at various different timepoints, was implemented by probabilistically removing 50% of the connection weights in the network. This work is notable in that it provides an explicit test of one role of CR in modulating the effects of damage within a cognitive domain. There are relatively few studies that have sought to develop on this approach. Furthermore, most computational approaches to date appear to have targeted the capacity reserve (passive) form of CR proposed by Stern. We argue that a better understanding is needed for how the use of different functional architectures may modulate the effects of damage.

In this paper we examine the effects of damage within ageing dynamical systems models. Our central goal is to test two implementations of CR. We implement passive and active forms of CR proposed by Stern (2009). In the first instance, we assess the effects of varying the degree of connectivity between processes in a given architecture. In the second instance, we examine how the use of different functional architectures may modulate the effects of damage. Our target architectures are the Fully distributed, Hemispheric, Central processor, Bi-directional and Uni-directional architectures, represented in Figure 1. We further aim to examine how intelligence levels may modulate the patterns of damage, given the different implementations of CR.

Dynamical systems theory

Dynamical systems theory (DST) provides one way of addressing these questions as it offers a framework for exploring the interaction between multiple component processes in a cognitive system. This then allows the possibility of tracing the consequence of changes to a given system over time. By specifying the relationship between component processes, we may stipulate exactly what the functional architecture is, and then test how the effects of ageing and damage unfold in a particular architecture. We base our approach, on the 'mutualism model' of intelligence first proposed by van der Maas and colleagues (2006) and which was subsequently extended by Baughman and Thomas (2008) to explore the effects of early focal impairments to a process within a range of different functional architectures.

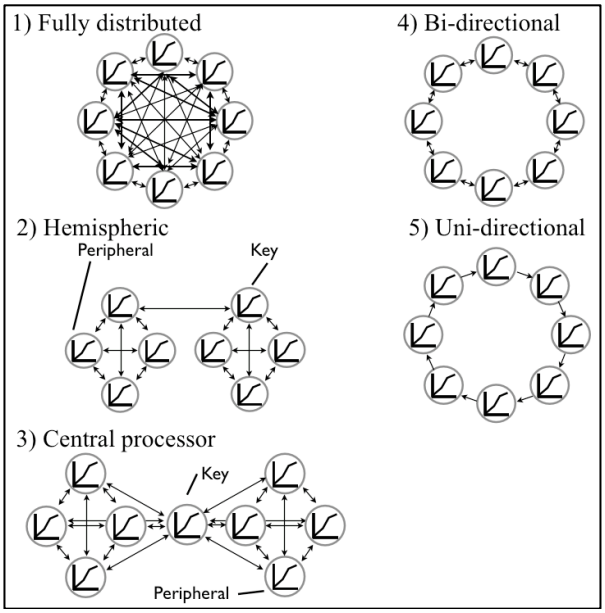

Figure 1: Five alternative model architectures. Note, this figure is illustrative of the architectural design. The actual models contained 16 processes each, and 17 in the case of the Central processor model.

The mutualism model

van der Maas et al. (2006) offered a fully connected dynamical systems model of the development of intelligence that simulates cognitive development for a number of components (depicted by Model 1, of Figure 1). The model provides a number of parameters that influence development for each individual processes, but where development of the model, as a whole, is influenced dynamically by all processes within the model. A key feature of their model, is that the processes which are connected to each other within a system interact with one another and influence each other, in a mutually beneficial way throughout development. Hence, the model is called the 'mutualism' model. Equation 1 gives the dynamics of the mutualism model.

$$\frac{dx_i}{dt} = a_i x_i \left(\frac{1 - x_i}{K_i}\right) + a_i \sum_{\substack{j=1 \\ j \neq i}}^{w} M_{ij}\, x_j x_i \,/K_i$$

Equation 1. The mutualism model (van der Maas (2006)

The mutualism equation is derived from population dynamics and the Lotka-Volterra equation. Briefly, the equation states that at each point in time (t) the change in the performance level x of a given process i (dx_i) is a

product of the sum of the interaction weights of each process j to which it is functionally connected ($M_{ij}x_jx_i$), multiplied by the rate of growth of process i (a_i), multiplied by the current level of performance of process x_i, divided by the asymptote level for that process (K_i). Changes in x_i at each time step are thereby constrained by the performance (and thus the individual properties) of all other processes to which it is connected.

Extending the mutualism model, Baughman and Thomas (2008) showed that following impairments to a single process, early on in development, architectures characterized by greater connectivity between processes offered greater *compensation* and showed reduced levels of *spread* of damage. Additionally, they showed that compensation and spread were further modulated by where in the cognitive system impairments were applied. Baughman and Thomas distinguished *peripheral* processes from those that occupied *key* positions within a given architecture. For example, while in the Fully distributed model all processes are equal (and so impairment to one process is equivalent to damage to any other process), this is not the same for the Hemispheric and Central processor architectures. Both these models contain peripheral processes (e.g., processes in one hemisphere which do not directly influence processes in the other hemisphere) and key processes (e.g., processes within one hemisphere share a direct connection to processes in the other hemisphere). Figure 1 illustrates the distinction between peripheral and key processes. The effects of damage to peripheral versus key processes within different functional architectures remains largely untested. As such, it is not obvious whether the same architectures that offer advantages following damage to processes early in development, will also offer advantages to damage later in development.

Simulations

In both the Normal and Damaged models, Ageing and IQ were implemented by manipulating values of the capacity for each process (K).

Ageing: General cognitive decline was simulated by applying a fixed level of decay (0.075%) to the capacity (K) of each process from 400 timesteps onwards. For the present simulations, we did not examine the consequences of variability in the rate, or the onset of decay.

IQ: To create Low IQ, Average IQ and High IQ models, models were calibrated to begin with different starting values of K (Low IQ=2, Average IQ=3, and High IQ=4).

Cognitive Reserve: For the passive form of CR, we tested three levels of Connection strength between processes (M). However, because the boundaries of values that this parameter accepts without exhibiting catastrophic effects are limited, the range we implemented was small. We used M=0.049, M=0.050, and M=0.051, to simulate Low, Average and High Connectivity, respectively. For the active form of CR, we compared the effects of damage in Fully distributed, Hemispheric, Central processor, Bi-directional, and Uni-directional architectures (see Figure 1).

Damage: In the damaged models, a single process was removed from the cognitive system to simulate total destruction of that process. Damage was applied separately to a peripheral process in each architecture, then to the key processes in the Hemispheric and Central Processor architectures. We held constant the level of damage (one process was damaged in under all architectures) and the onset of damage. Damage was applied to either a peripheral or a key process at timepoint 550, just over half-way through the models 'lifetime'. The damaged process was thus removed from the network and the relevant connections to and from it, also removed. All other parameters specified in the mutualism model, namely those relating to the growth rates of processes (a), and the initial starting states of each process (x) were also held constant and did not vary in these simulations (x=0.05, a=6.0). Finally, because one of our primary concerns was examining specific levels of IQ, we were not concerned with population variability. Thus, we did not require the models to be run for many pseudosubjects and only a single model was run for each architecture in Figure 1 for 1000 time steps. The full set of models that we tested totaled 108. These were comprised of: (i) Normal ageing models at 3 levels of IQ (Low, Average and High) within 3 levels of Connectivity (Low, Average and High) and 5 Architectures (Fully distributed, Hemispheric, Central processor, Bi-directional, and Uni-directional); (ii) Peripherally-damaged ageing models (as Normal, but with one process damaged); and, (iii) Key-damage ageing models (IQ: Low, Average and High x Connectivity (Low, Average and High) x 2 Architectures (Hemispheric, Central processor). Figure 2 shows the trajectories for Normal and Damaged models for the Fully distributed, Central processor and Uni-directional architectures, at Average IQ, Average Connectivity levels.

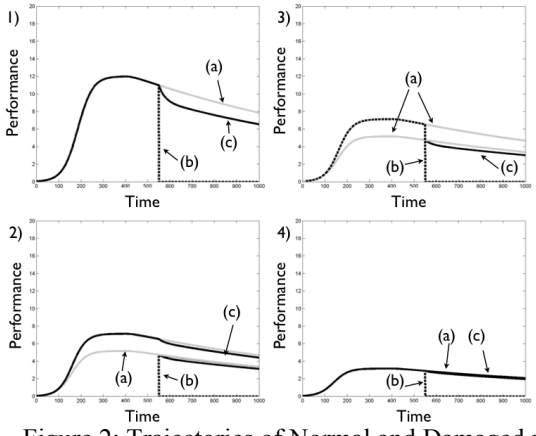

Figure 2: Trajectories of Normal and Damaged models for the Fully distributed (Tile 1), Central Processor (Tile 2: damage to peripheral process; Tile 3: damage to key process), and Uni-directional (Tile 4) architectures at Average Connectivity (M=0.05) and Average IQ (K=3) levels. Tiles depict processes in the Normal models (a) with a grey line, and the damaged (b) and affected processes (c) in the Damaged models, with dashed and solid black lines, respectively.

Measures

Asymptote levels in the architectures differ as a consequence of the number of processes that are connected within it. As such, comparisons between the absolute levels reached by two architectures would be misleading. Instead, we use each Normal model as the benchmark for which to compare the performance of its damaged counterpart. This allows for relative comparisons across the different architectures. The two key metrics we use to assess the effects of damage are: (1) Area - the extent to which the trajectories of processes in the Damaged model resembles those in the Normal model (we compute the area under the curve, for each Damaged processes, and this is turned into a proportion of the area of the processes in the Normal model); and, (2) Endstate level – the extent to which the endstate levels of the Damage model reaches the functional endstate of the Normal model. Thus, area gives a measure of models attempt to compensate for damage, and endstate provides a measure of the models ability to recover.

Results

Table 1 provides the Area data for Normal and Damaged models, at each level of intelligence and each level of Connectivity. The table shows effects of manipulations to IQ and Connectivity, across each of the architectures tested. The uppermost part of the table provides the data for comparisons for Normal versus Peripherally-Damaged models, the lowermost part of the table shows these comparisons for Normal versus Key-Damaged processes, in the Hemispheric and Central Processor models.

Intelligence

As expected, varying the level of intelligence (IQ) in a model had direct effects on the overall level of performances reached. Table 1 shows that for each architecture higher IQ models performed better compared to lower IQ models (e.g., the level of performances reached in the Uni-dimensional architecture at each level of IQ, under Low Connectivity, are 11769.8, 17654.6 and 23539.5, respectively). However, the results of the simulations showed that IQ level did not modulate the effects of damage within architecture, at the various levels of CR. That is, within a given level of Connectivity, the effect of damage was proportionate at each level of IQ. For example, in the Low Connectivity Fully distributed model, the proportion of area reached by the Damaged models in Low IQ, Average IQ and High IQ models were all 80.2% of Normal levels.

Cognitive reserve as differences in connectivity

Varying CR, when implemented as level of Connectivity, showed small, but consistent effects on level of performance reached (e.g., the levels reached in the Normal Hemispheric model at each level of Connectivity, under High IQ, are 35425.8, 35852.7 and 362900.0. However, greater levels of CR did not protect models from the effects of damage. In fact, the reverse was found to be the case. Increased connectivity between processes resulted in higher proportion of spread of damage and poorer endstate recovery. This outcome was true for all architectures, but most apparent in the Fully distributed model. Figure 3 shows the proportion of area and endstate levels reached for each architecture, at each level of Connectivity, following peripheral damage.

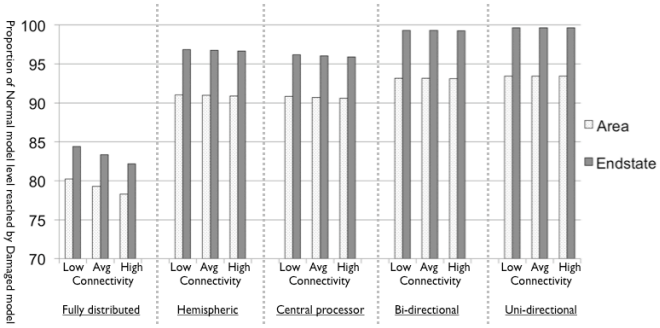

Figure 3: Proportion of Area and Endstate obtained in peripherally-damaged models by level of Connectivity.

Figure 4 shows that these effects are further exaggerated by damage to the key processes, in the Hemispheric and Central processor models. This figure shows that following damage to the key process, the Hemispheric model reached levels of recovery that were similar to the peripherally-damaged model (the greatest difference between key and peripherally-damaged process endstate was 1%). In the Central processor model, endstates differed by approximately 9%. The figure also shows that in the Central Processor model, key damage resulted in both lower recovery (Endstate) and more protracted form of recovery (Area).

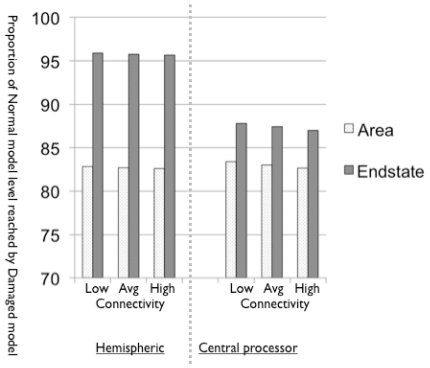

Figure 4: Proportion of Area and Endstate obtained in key-damaged models by level of Connectivity.

Table 1. Calculations of area under the curve for trajectories from the Normal and Damaged models

		Fully Distributed Connectivity			Hemispheric Connectivity			Central Processor Connectivity			Bi-Directional Connectivity			UniDirectional Connectivity		
Normal models		Low	Average	High	Low	Average	High	Low	Average	High	Low	Average	High	Low	Average	High
IQ	Low	42237.8	44772.1	47629.9	17712.9	17926.3	18145.0	20610.9	20929.6	21258.5	12409.1	12436.7	12464.4	11769.8	11782.1	11794.6
	Average	63356.7	67158.1	71444.8	26569.3	26889.5	27217.5	30916.3	31394.4	31887.8	18613.7	18655.1	18696.6	17654.6	17673.2	17691.8
	High	84475.6	89544.1	95259.7	35425.8	35852.7	36290.0	41221.8	41859.2	42517.0	24818.2	24873.4	24928.8	23539.5	23564.3	23589.1
	mean	63356.7	67158.1	71444.8	26569.3	26889.5	27217.5	30916.3	31394.4	31887.8	18613.7	18655.1	18696.6	17654.6	17673.2	17691.8
Damaged Peripheral		Low	Average	High	Low	Average	High	Low	Average	High	Low	Average	High	Low	Average	High
IQ	Low	33881.7	35499.0	37278.8	16127.0	16306.4	16489.9	18718.7	18984.4	19258.0	11559.8	11583.9	11608.1	10999.4	11010.2	11021.1
	Average	50822.6	53248.4	55918.1	24190.5	24459.6	24734.8	28078.1	28476.6	28887.0	17339.7	17375.9	17412.2	16499.1	16515.4	16531.6
	High	67763.4	70997.9	74557.5	32253.9	32612.8	32979.8	37437.4	37968.8	38516.0	23119.7	23167.8	23216.2	21998.8	22020.5	22042.2
	mean	50822.6	53248.4	55918.1	24190.5	24459.6	24734.8	28078.1	28476.6	28887.0	17339.7	17375.9	17412.2	16499.1	16515.4	16531.6

			Connectivity			Connectivity		
Damaged Key			Low	Average	High	Low	Average	High
	IQ	Low	14669.3	14827.4	14988.8	17187.1	17378.1	17573.5
		Average	22070.7	22308.5	22551.6	25780.6	26067.1	26360.2
		High	29490.6	29808.5	30133.5	34374.1	34756.2	35146.9
		mean	22076.9	22314.8	22558.0	25780.6	26067.1	26360.2

Cognitive reserve as differences in functional architecture

Implementing CR, as different functional architectures, did modulate the effects of damage. However, it was not those architectures characterized by more connectivity between processes that proved most resilient to damage. Indeed, it was those architectures comprised of more limited connectivity where the effects of damage were minimized and the endstate levels of recovery most complete. In the architectures tested here, this was the Uni-directional architecture. Damage to any process in this architecture had effects on processes downstream of the damaged process.

But these effects became increasingly small, over the remainder of the models lifetime. Figure 5 shows Area (left) and Endstate levels (right), respectively, for each of the architectures tested.

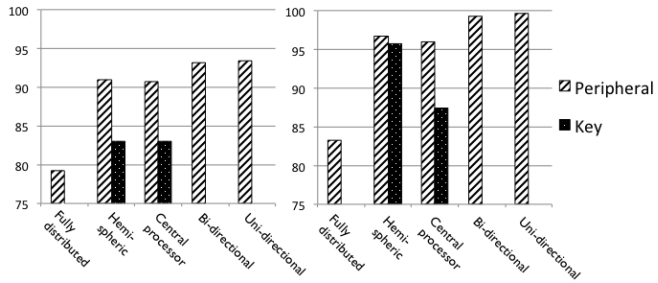

Figure 5: Comparisons of functional architectures by proportion of Area (left) and Endstate (right) obtained in peripherally-damaged and key-damaged models.

Conclusions

Previous simulation studies have showed that following early forms of focal impairment, architectures characterized by greater levels of connectivity offer superior levels of protection compared to those with more limited connectivity (see Baughman & Thomas, 2008). However, in the simulations reported here, where permanent damage occurred to a system late on in its development, it was those models characterized by less connectivity (i.e., more specialized in function) that offered greatest resilience to damage. Examples of those offering the greatest protection are the Bi-directional and Uni-directional models, with the Fully distributed architecture offering the least protection following damage. These results indicate that throughout the process of development, similar events that impair just a limited number of processes to a system may have very different consequences for its outcome. These results are consistent with the notion that different functional architectures may underlie different stages of development (Fransson, Aden, Blennow, & Lagercrantz, 2011), possibly through a process of emergent specialization (Karmiloff-Smith, 2009). Future work is needed to investigate how the parameters we held constant (such as rate of decline, cognitive growth, and the severity and onset of damage) might provide a more complete account of the factors that contribute to real-world variability in ageing.

References

Alstott, J., Breakspear, M., Hagmann, P., Cammoun, L., & Sporns, O. (2009). Modeling the impact of lesions in the human brain. *Plos Computational Biology*, *5*(6).

Baughman, F. D., & Thomas, M. S. C. (2008). Specific impairments in cognitive development: A dynamical systems approach. In *Cognitive science.* In B. C. Love, K. McRae, & V. M. Sloutsky (Eds.), Proceedings of the 30th Annual Conference of the Cognitive Science Society (pp. 1819-1824). Austin, TX: Cognitive Science Society.

Fransson, P., Aden, U., Blennow, M., & Lagercrantz, H. (2011). The functional architecture of the infant brain as revealed by resting-state fmri. *Cerebral Cortex (New York, N.Y. : 1991)*, *21*(1), 145-154.

Kaplan, R. F., Cohen, R. A., Moscufo, N., Guttmann, C., Chasman, J., Buttaro, M., . . . Wolfson, L. (2009). Demographic and biological influences on cognitive reserve. *Journal of Clinical and Experimental Neuropsychology*, *31*(7), 868-876.

Karmilof-Smith, A. (2009). Nativism versus neuroconstructivism: Rethinking the study of developmental disorders. *Developmental Psychology, 45(1)*, 56-63.

Koenen, K. C., Moffitt, T. E., Roberts, A. L., Martin, L. T., Kubzansky, L., Harrington, H. L., . . . Caspi, A. (2009). Childhood IQ and adult mental disorders: A test of the cognitive reserve hypothesis. *The American Journal of Psychiatry*, *166*(1), 50.

Li, S. C., Von Oertzen, T., & Lindenberger, U. (2006). A neurocomputational model of stochastic resonance and aging. *Neurocomputing*, *69*(13-15), 1553-1560.

Nithianantharajah, J., & Hannan, A. J. (2009). The neurobiology of brain and cognitive reserve: Mental and physical activity as modulators of brain disorders. *Progress in Neurobiology*, *89*(4), 369-382.

Rubinov, M., McIntosh, A. R., Valenzuela, M. J., & Breakspear, M. (2009). Simulation of neuronal death and network recovery in a computational model of distributed cortical activity. *American Journal of Geriatric Psych*, *17*(3), 210.

Stern, Y. (2002). What is cognitive reserve? Theory and research application of the reserve concept. *Journal of the International Neuropsychological Society*, *8*(3), 448-460.

Stern, Y. (2009). Cognitive reserve. *Neuropsychologia*, *47*(10), 2015-28.

Thomas, M. S. C. (2008). Ageing, plasticity, and cognitive reserve in connectionist networks. In B. C. Love, K. McRae, & V. M. Sloutsky (Eds.), *Proceedings of the 30th Annual Conference of the Cognitive Science Society* (pp. 2089-2094). Austin, TX: Cognitive Science Society.

Tucker-Drob, E. M., Johnson, K. E., & Jones, R. N. (2009). The cognitive reserve hypothesis: A longitudinal examination of age-associated declines in reasoning and processing speed. *Developmental Psychology*, *45*(2), 431-46.

van der Maas, H. L. J., Dolan, C. V., Grasman, R. P. P. P., Wicherts, J. M., Huizenga, H. M., & Raijmakers, M. E. J. (2006). A dynamical model of general intelligence: The positive manifold of intelligence by mutualism. *Psychological Review*, *113*(4), 842-61.

Whalley, L. J., Deary, I. J., Appleton, C. L., & Starr, J. M. (2004). Cognitive reserve and the neurobiology of cognitive aging. *Ageing Research Reviews*, *3*(4), 369-82.

Zahodne, L. B., Glymour, M. M., Sparks, C., Bontempo, D., Dixon, R. A., MacDonald, S. W., & Manly, J. J. (2011). Education does not slow cognitive decline with aging: 12-Year evidence from the victoria longitudinal study. *Journal of the International Neuropsychological Society*, *17*(6), 1039-46.