Understanding the Neurocognitive Organization as Strategies Rather than Functions: Implications for Neurological Research

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Abstract

In neurological research, cognitive/behavioural evaluations typically has the traditional cognitive functions (e.g. working memory or spatial orientation) as the conceptual and methodological point of departure. However, such "functions" appear to be neither unitary nor homogenous (across individuals) - e.g. numerous cognitive tests can be demonstrated to be procedure-dependent. And additionally, there is growing evidence of unexpected individual differences and experience-dependency regarding traditional "functions". Thus, the traditional system of cognitive functions needs to be modified. Presently, it is argued that the adequate focus is the applied strategies rather than traditionally defined functions. This is argued with the Reorganization of Elementary Functions (REF) framework as the theoretical foundation. Furthermore, it is being discussed how best to approach the neurocognitive research process with such a focus on strategies rather than functions. The use of already established "challenge" procedures is being referred to. Also, it is presented that novel computational methods are presently being developed towards even more detailed and unbiased analyses of behavioural/cognitive data as well as analyses of neural processes revealed by EEG/MEG.

Keywords: *Neural Connections; Cognition; Mental States; Neural States; Computational States; Neurology; Neurological Testing; Cognitive Testing; Brain Injury; Integrative Models; REF Framework; Reorganization of Elementary Functions (REF)*

Abbreviations

AM: Algorithmic Module; AS: Algorithmic Strategy; EEG: Electroencephalography; EF: Elementary Function; MEG: Magnetoencephalography; PAT: Prism Adaptation Therapy; REF: Reorganization of Elementary Functions; tDCS: Transcranial direct current stimulation; TMS: Transcranial Magnetic Stimulation

Addressing functions in neurological research

In neurological research - be it basic studies of neurocognitive organization or research addressing one or another therapeutic method - an important element is to evaluate the cognitive abilities and performance of the individual. Both in human studies and in animal based research this opens the question of how to evaluate such cognitive abilities. The point of departure will typically be a desire to evaluate how "intact" or impaired a given function is. For instance, one may want to evaluate whether a given training paradigm can improve the function working memory in stroke patients [1] or one may, in an animal model, want to evaluate whether prefrontal cortical lesions and hippocampal lesions are associated with equal or dissimilar levels of impairment of the function allocentric spatial orientation [2]. In case of working memory the choice of test would typically be one of the standard working memory tests - such as a digit span or n-back test [3]. Addressing allocentric spatial orientation in for instance a rodent one might focus on a water maze based test such as an allocentric place learning task [4].

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Applying such cognitive tasks in humans or animals may seem relatively straightforward: One is to follow the established procedures - especially regarding the "cognitively essential" aspects of those procedures. Unfortunately, the existing knowledge regarding such essential factors may be too limited. For instance, it has been well established that in a Prism Adaptation Therapy (PAT) procedure, a crucial aspect is that the subject receives correct information/feedback regarding the actual position to which she/he was pointing - allowing that position to be compared to the goal position to which she/he was attempting to be pointing [5]. In a computer-based version of the procedure, however, it turned out that two different versions of such feedback gave radically different outcomes - in spite of both giving the position [6]. Returning to the initially mentioned tests of working memory, the n-back test has, for instance, been demonstrated to exhibit method dependence - procedural differences are associated with different patterns of neural activation [7]. Various "span" tasks (e.g. digit or word span) also exhibit different outcomes depending on procedural factors [8]. In general, the straightforward idea of a "working memory" with a limited capacity seems to be questioned by both such procedural effects and by experiments e.g. demonstrating that an increased working memory load (as opposed to a decrease in load of such a system) may be associated with a facilitated language production [9]. The pattern of method-dependency also holds for animal model based studies. For instance, in case of water maze based allocentric place learning tasks, both the neural and cognitive processes depend on the experimental setup [2,10-12]. The fact that apparently similar versions of cognitive tasks may be associated with dissimilar outcomes at the neural and cognitive level is far from limited to these few examples [4,6].

The results described above (and others), point to the need for neurological testing to be supplemented with methods better able to capture the unexpected complexity of the neurocognitive situations. But those results also point to the need to replace or supplement the traditional theories of neurocognitive organization with models better able to capture those complexities.

The REF (Reorganization of Elementary Functions) framework

The above-described results as well as many others tend to disagree with the traditional "modular" models of organization [13-16]. But they also contradict the traditional versions of distributed connectionist network models [17-20] - for discussions of these contradictions: see e.g. Mogensen and Overgaard [21,22]. They are, however, better understood within the REF (Reorganization of Elementary Functions) framework [21-29]. Not the least because the REF model and the REF framework in general emphasize strategies rather than traditional "functions" as an essential mechanism in neurocognitive organization [21,28]. For a recent review of the REF framework: see [21].

The REF model is based on three levels of analysis. Both the top and bottom layers represent types of "functions". However, the way functions are conceptualized differ significantly between the two layers. The upper layer represents what is termed "surface phenomena". Such surface phenomena are all behavioural and/or mental manifestations (including conscious awareness). All normal as well as pathological behavioural/cognitive expressions are surface phenomena. If, for instance, a patient after suffering acquired brain injury demonstrates expressive aphasia (being unable to express herself/himself linguistically or having a pathologically changed language performance), those linguistic changes are all changed (or absent) surface phenomena. And in case of a subsequent functional recovery, the recovered expressive language is also a surface phenomenon. This means that it is also at the level of surface phenomena that we face the apparent contradiction between functional localization and posttraumatic functional recovery. If the linguistic surface phenomena are mediated by specific brain structures (as it is evident from both neuroimaging studies and the results of focal brain injury [30-41]), one would logically not expect such a brain injured patient to be able to regain linguistic abilities via posttraumatic rehabilitative training. Nevertheless, such patients will typically exhibit a more or less complete linguistic recovery - manifest at the level of surface phenomena [30-34,36,38,40-42]. One of the motivations for developing the original version of the REF model [23-27] was to be able - within the same neurocognitive model - to account for both functional localization and posttraumatic functional recovery.

Within the REF framework, it is at the lowermost level of analysis that one finds a true functional localization. What is represented within the lowermost level of analysis are the Elementary Functions (EFs). The neural substrate of a given EF is relatively small (compared

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to the presumed neural substrates of traditionally defined functions) and is strictly localized within a subdivision of a neural structure (be it cortical or subcortical). Every EF performs a ("pre-wired") relatively low-level information processing - receiving an input, performing the fixed processing and subsequently providing the output. The (unique) information processing of a given EF may best be described in mathematical terms - rather than in the more "traditional functional" vocabulary of for instance cognitive psychology. No EF is specifically associated with any of the traditionally defined cognitive functions (such as attention, spatial orientation, and visual perception). Rather, practically every EF is simultaneously associated with information processing mediating surface phenomena that can be characterized as various "cognitive functions" and/or behavioural manifestations. EFs can be seen as the "building blocks" of the mechanisms mediating the surface phenomena.

The mid-level of analysis constitutes the "bridge" between the lowermost and uppermost levels of analysis. Within this intermediate level one finds the Algorithmic Strategies (ASs) which are the mechanisms mediating the surface phenomena. A given AS mediates a specific surface phenomenon - in the form of a task solution, specific cognitive process etc. An AS consists of numerous interconnected EFs. The neural substrate of an AS consists of the neural substrates of all constituent EFs (typically distributed over many regions of the brain) as well as the connections between the neural substrates of these EFs. Consequently, the neural substrate of an AS is highly distributed.

As ASs develop (see below), common areas between individual ASs may gain a functional autonomy - making them Algorithmic Modules (AMs). Like an AS, an AM is a connectionist network within which the "units" are EFs. Contrary to ASs, however, AMs are not able in themselves to mediate surface phenomena. An AM contributes its information processing by becoming integrated into an AS. Thus, AMs are to be considered "higher level building blocks" to ASs. This can be illustrated by an example from language production and language interpretation. Both are mediated by ASs - being the strategies mediating the surface phenomena of production or interpretation of linguistic expressions. But in contrast to these ASs, grammar may be mediated by an AM [43]. While grammar is obviously an important component within both production and interpretation of language, grammar cannot in itself be a surface phenomenon.

When an individual faces a situation calling for a behavioural manifestation and/or a mental process, mechanisms described elsewhere [21] activate a pre-existing AS to become the neural mechanism mediating that behavioural and/or mental process. If an appropriate AS is activated, the process will be successful. But if a less successful AS has been activated, the complete or partial failure will lead to a two-part process. One part is the continued search for a (more) appropriate AS. The other part is a backpropagation process [44-50] restructuring the AS which has been attempted as a mechanism as well as a number of related ASs. Additionally, the mechanisms selecting ASs will (also via backpropagation mechanisms) be modified in such ways that the likelihood of a more successful AS selection will be higher in the future.

If part of the brain tissue is lost, the EFs mediated by that brain region are lost permanently. Consequently, ASs including those EFs are also lost. And the surface phenomena associated with those ASs can no longer be mediated by the ASs in question. EFs are functionally unique and without any "backup" structures. Therefore, the only way to accomplish a functional recovery is for a reorganization of the connectivity between EFs to occur - leading to new ASs being available. If eventually a restructured AS is able to produce a surface phenomenon of equal proficiency to what was seen pretraumatically, the situation is described as a "full recovery". It should, however, be noticed that such a recovery does not replace neither the neural substrate nor the cognitive mechanisms behind the originally lost surface phenomenon [26]. While traditional connectionist networks (which are networks of functionally neutral "neurons") cannot account for a functional localization, the REF model can do so. A given brain region is (primarily genetically) pre-determined to mediate specific EFs. And when developing the functional organization of the normal brain, mechanisms such as those described above (selection and potential modification of ASs) will typically lead to rather similar organizations across individuals - simply because certain EFs are more obvious candidates for inclusion in a given AS than others. But as mentioned: lacking those originally developed ASs, alternative ASs can develop and potentially support the same proficiency of task performance (surface phenomenon) in spite of not doing so via the same neurocognitive processes as in the "normal" brains.

From functions to strategies - methodological and computational challenges

Returning to the issue of "functional evaluations" of neurological patients, intact human beings and/or experimental animals, the "functions" such as working memory or allocentric spatial orientation are all surface phenomena - and mediated by ASs. Consequently,

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the mechanisms that might be most relevant to address when addressing cognition in neurological research are the ASs (and potentially AMs) - the strategies. The fact that strategies rather than functions may be the adequate focus of study is also supported by research demonstrating that neurocognitive mechanisms can be radically dependent on experience. One such example is that when performing a visual spatial task (mental rotation), orchestral musicians (contrary to musically naïve control subjects) include the Broca area in the neural substrate of task performance - and additionally exhibit a superior to normal quality of task performance [51].

Then how can experimental neurology step beyond the traditional concepts of "function" and better approach a strategy-based understanding of neurocognitive processes?

One approach - that has already been utilized and described for years - is to apply organic and/or cognitive/behavioural "challenges" [4,27]. In organic challenges the functional efficacy of a given brain structure or brain system (structurally and/or neurochemically defined) is altered (either increased or decreased) and the associated behavioural/cognitive performance is investigated (often across experimental groups - e.g. originally lesioned and subsequently recovered individuals versus intact controls). Obviously, such methods are most easily applied in animal models [2,11-12,52,53]. In humans, such organic challenges have to remain noninvasive - but can be obtained using for instance TMS (transcranial magnetic stimulation) or tDCS (transcranial direct current stimulation) [54-63]. Functional challenges will typically involve modifications of the environment of the test situation (e.g. modified "cues") and/or modifications of the test procedures (changed duration of pauses, modified order of stimulus presentations etc.) [2,10-12,52,53,64-66].

The combined results of organic and cognitive/behavioural challenges have repeatedly demonstrated that the successful performance of a cognitive test when compared across experimental groups (e.g. differently lesioned but fully recovered individuals) is accomplished utilizing different cognitive strategies [2]. Such results are in themselves informative regarding the fact that strategies may be a more adequate focus than traditionally defined "functions" (see references above). It does, however, not take into account the potential individual strategy differences within a given group. Such individual variations in the utilization of strategies was already demonstrated by Krechevsky [67,68]. Novel methods do, however, hold significant promise regarding a more detailed account of individual strategies in both animal model based and human research. Such methods include the use of computational methods that can - in an experimenter-unbiased way - extract "patterns of performance" from the behavioural/cognitive performance of individuals. A more detailed account of the use of such methods in the context of neurocognitive organization will be published separately. But presently we would like to refer to such methods as those published by Schulz., *et al* [69]. Such an analysis is based on the Damerau-Levenshtein distance analysis. The method has been successfully applied to humanly generated random number sequences. Analysing such sequences, it was possible to identify a "cognitive fingerprint" in each individual subject's sequence generation. In the present context we consider such "fingerprints" to be reflections of the individually developed ASs and AMs. In a related study Rosenberg., *et al.* [70] compared such humanly generated random number sequences in three experimental groups: normal controls, alcoholic individuals, and patients suffering schizophrenia. Focusing on the individual performance of subjects and the potential intergroup variation, the authors demonstrated that while the internal variation matrices were rather similar between the normal group and the group of alcoholics, the intergroup variation (and consequently uniqueness of individual strategies/ASs/AMs) was significantly higher in the schizophrenic group.

The use of further developed computational methods like those presently referred to hold significant promise for a deeper understanding of the ASs and AMs of both individuals and experimental groups.

Computational methods are, however, also developed towards a deeper understanding of the neural substrate of ASs, AMs and potentially EFs. Using EEG (electroencephalography) and/or MEG (magnetoencephalography) while subjects perform various types of cognitive tasks - for instance variants of the Raven test [71] - one can extract activity patterns relevant to various phases of the cognitive performance utilizing a non-linear analysis of the EEG/MEG signal [71-74]. Such an analysis can provide regionally highly specific information regarding local shifts in the level of entropy. Utilization of such computational methods are likely to provide novel and dynamic pictures of the neural activity accompanying activation of individual ASs/AMs.

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The mentioned computational methods are still being further developed in order to provide the optimal analysis of cognition as well as neural processes. It is, however, already obvious that the inclusion of these computational methods holds significant promises regarding an even more advanced understanding of the neurocognitive processes associated with both the individually applied strategies and the overall pattern of strategies across groups.

We do not argue that the described strategy-based analyses (including computational methods) should replace the more traditional testing methods in neurological research. But we do argue that they already constitute an important supplement and are likely to become even more useful and informative within the next few years.

Bibliography

1. van de Ven RM., *et al.* "Computer-based cognitive training for executive functions after stroke: a systematic review". *Frontiers in Human Neuroscience* 10 (2016): 150.

2. Mogensen J., *et al.* "Place learning and object recognition by rats subjected to transection of the fimbria-fornix and/or ablation of the prefrontal cortex". *Brain Research Bulletin* 63.3 (2004): 217-236.

3. Conway ARA., *et al.* "Working memory span tasks: a methodological review and user's guide". *Psychonomic Bulletin and Review* 12.5 (2005): 769-786.

4. Mogensen J. "Animal models in neuroscience". In: J Hau and SJ Schapiro (Eds.), *Handbook of Laboratory Animal Science, Third Edition, Volume II. Animal Models*. Boca Raton, FL: CRC Press LLC (2011): 47-73.

5. Serino A., *et al.* "Mechanisms underlying neglect recovery after prism adaptation". *Neuropsychologia* 44.7 (2006): 1068-1078.

6. Wilms I and Mogensen J. "Dissimilar outcomes of apparently similar procedures as a challenge to clinical neurorehabilitation and basic research - when the same is not the same". *NeuroRehabilitation* 29.3 (2011): 221-227.

7. Owen AM., *et al.* "N-back working memory paradigm: a meta-analysis of normative functional neuroimaging studies". *Human Brain Mapping* 25.1 (2005): 46-59.

8. Turner ML and Engle RW. "Is working memory capacity task dependent?" *Journal of Memory and Language* 28.2 (1989): 127-154.

9. Ishkhanyan B., *et al.* "The meeting point: where language production and working memory share resources". *Journal of Psycholinguistic Research* (2018).

10. Mogensen J., *et al.* "Prefrontal cortical mediation of rats' place learning in a modified water maze". *Brain Research Bulletin* 38.5 (1995): 425-434.

11. Mogensen J., *et al.* "Place learning in scopolamine treated rats: the roles of distal cues and catecholaminergic mediation". *Neurobiology of Learning and Memory* 78.1 (2002): 139-166.

12. Wörtwein G., *et al.* "Place learning by fimbria-fornix transected rats in a modified water maze". *International Journal of Neuroscience* 82.1-2 (1995): 71-81.

13. Barrett HC and Kurzban R. "Modularity in cognition: Framing the debate". *Psychological Review* 113.3 (2006): 628-647.

14. Fodor JA. "The modularity of mind". Cambridge, MA: MIT Press (1983).

15. Fodor J. "The Mind Doesn't Work That Way: The Scope and Limits of Computational Psychology". Cambridge, MA: MIT Press (2000).

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16. Pinker S. "How the Mind Works". London: Penguin Books (1999).

17. McClelland JL., *et al.* "Parallel Distributed Processing: Volume 2. Psychological and Biological Models". Cambridge, MA: MIT Press (1986).

18. McLeod P., *et al.* "Introduction to Connectionist Modelling of Cognitive Processes". Oxford: Oxford University Press (1998).

19. Rogers TT and McClelland JL. "Parallel distributed processing at 25: Further explorations in the microstructure of cognition". *Cognitive Science* 38.6 (2014): 1024-1077.

20. Rumelhart D and McClelland J. "Parallel Distributed Processing". Cambridge, MA: MIT Press (1986).

21. Mogensen J and Overgaard M. "Reorganization of the connectivity between Elementary Functions - a model relating conscious states to neural connections". *Frontiers in Psychology* 8 (2017): 625.

22. Mogensen J and Overgaard M. "Neural connections and mental states: The need for a neurocognitive framework". *EC Neurology* 10 (2018): 180-194.

23. Mogensen J. "Almost unlimited potentials of a limited neural plasticity: Levels of plasticity in development and reorganization of the injured brain". *Journal of Consciousness Studies* 18.7-8 (2011): 13-45.

24. Mogensen J. "Reorganization in the injured brain: implications for studies of the neural substrate of cognition". *Frontiers in Psychology* 2 (2011): 7.

25. Mogensen J. "Reorganization of Elementary Functions (REF) after brain injury and in the intact brain: A novel understanding of neurocognitive organization and reorganization". In: J Costa and E Villalba (Eds.) Horizons in Neuroscience Research. Volume 15. New York: Nova Science Publishers, Inc. (2014): 99-140.

26. Mogensen J. "Recovery, compensation and reorganization in neuropathology - levels of conceptual and methodological challenges". In: JI Tracy, BM Hampstead and K Sathian (Eds.), *Cognitive Plasticity in Neurologic Disorders*. New York: Oxford University Press (2015): 3-28.

27. Mogensen J and Malá H. "Post-traumatic functional recovery and reorganization in animal models. A theoretical and methodological challenge". *Scandinavian Journal of Psychology* 50.6 (2009): 561-573.

28. Mogensen J and Overgaard M. "Reorganization of the connectivity between Elementary Functions as a common mechanism of phenomenal consciousness and working memory: from functions to strategies". *Philosophical Transactions of the Royal Society of London, Series B: Biological Sciences* 373.1755 (2018): 20170346.

29. Overgaard M and Mogensen J. "Visual perception from the perspective of a representational, non-reductionistic, level-dependent account of perception and conscious awareness". *Philosophical Transactions of the Royal Society of London, Series B: Biological Sciences* 369.1641 (2014): 20130209.

30. Ansaldo AI and Arguin M. "The recovery from aphasia depends on both the left and right hemispheres: three longitudinal case studies on the dynamics of language function after aphasia". *Brain and Language* 87.1 (2003): 177-178.

31. Ansaldo AI., *et al.* "The contribution of the right cerebral hemisphere to the recovery from aphasia: a single longitudinal case study". *Brain and Language* 82.2 (2002): 206-222.

32. Baumgaertner A., *et al.* "Differential recovery of aphasia and apraxia of speech in an adolescent after infarction of the left frontal lobe: longitudinal behavioral and fMRI data". *Brain and Language* 95 (2005): 211-212.

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33. Meinzer M., *et al.* "Recovery from aphasia as a function of language therapy in an early bilingual patient demonstrated by fMRI". *Neuropsychologia* 45.6 (2007): 1247-1256.

34. Meinzer M., *et al.* "Functional re-recruitment of dysfunctional brain areas predicts language recovery in chronic aphasia". *NeuroImage* 39.4 (2008): 2038-2046.

35. Paulesu E., *et al.* "A cultural effect on brain function". *Nature Neuroscience* 3.1 (2000): 91-96.

36. Perani D., *et al.* "A fMRI study of word retrieval in aphasia". *Brain and Language* 85.3 (2003): 357-368.

37. Posner MI., and Raichle ME. "*Images of mind".* New York: Scientific American Library (1994).

38. Specht K., *et al.* "Joint independent component analysis of structural and functional images reveals complex patterns of functional reorganisation in stroke aphasia". *NeuroImage* 47.4 (2009): 2057-2063.

39. Szaflarski JP., *et al.* "Poststroke aphasia recovery assessed with functional magnetic resonance imaging and a picture identification task". *Journal of Stroke and Cerebrovascular Disease* 20.4 (2011): 336-345.

40. Thomas C., *et al.* "Language processing in aphasia: changes in lateralization patterns during recovery reflect cerebral plasticity in adults". *Electroencephalography and Clinical Neurophysiology* 102.2 (1997): 86-97.

41. Thulborn KR., *et al.* "Plasticity of language-related brain function during recovery from stroke". *Stroke* 30.4 (1999): 749-754.

42. Crosson B., *et al.* "Regional changes in word-production laterality after a naming treatment designed to produce a rightward shift in frontal activity". *Brain and Language* 111.2 (2009): 73-85.

43. Ishkhanyan B., *et al.* "Grammatical and lexical pronoun dissociation in French speakers with agrammatic aphasia: a usage-based account and REF-based hypothesis". *Journal of Neurolinguistics* 44 (2017): 1-16.

44. Durbin R and Rumelhart DE. "Product units: a computationally powerful and biologically plausible extension to backpropagation networks". *Neural Computation* 1.1 (1989): 133-142.

45. O'Reilly RC. "Biologically plausible error-driven learning using local activation differences: the generalized recirculation algorithm". *Neural Computation* 8.5 (1996): 895-938.

46. O'Reilly RC. "Six principles for biologically based computational models of cortical cognition". *Trends in Cognitive Sciences* 2.11 (1998): 455-462.

47. Rumelhart DE., *et al.* "Learning internal representations by error propagation". In DE Rumelhart and J McClelland (Eds.), *Parallel Distributed Processing: Explorations in the Microstructure of Cognition I.* Cambridge, MA: MIT Press (1986): 318-362.

48. Stork DG. "Is backpropagation biologically plausible?" *Neural Networks* 2 (1989): 241-246.

49. Werbos PJ. "Beyond regression: New tools for prediction and analysis in the behavioral sciences*.* Harvard University: Applied Mathematics (1974).

50. Werbos PJ. "The roots of backpropagation: From ordered derivatives to neural networks and political forecasting*.* New York: John Wiley and Sons (1994).

51. Sluming V., *et al.* "Broca's area supports enhanced visuospatial cognition in orchestral musicians". *Journal of Neuroscience* 27.14 (2007): 3799-3806.

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52. Gram MG., *et al.* "Equal effects of typical environmental and specific social enrichment on posttraumatic cognitive functioning after fimbria-fornix transection in rats". *Brain Research* 1629 (2015): 182-195.

53. Mogensen J., *et al.* "Serotonin, locomotion, exploration, and place recall in the rat". *Pharmacology, Biochemistry and Behavior* 75.2 (2003): 381-395.

54. Dambeck N., *et al.* "Interhemispheric imbalance during visuospatial attention investigated by unilateral and bilateral TMS over human parietal cortices". *Brain Research* 1072.1 (2006): 194-199.

55. Fridriksson J., *et al.* "Transcranial direct current stimulation improves naming reaction time in fluent aphasia. A double-blind, sham-controlled study". *Stroke* 42.3 (2011): 819-821.

56. Hamilton RH., *et al.* "Mechanisms of aphasia recovery after stroke and the role of noninvasive brain stimulation". *Brain and Language* 118.1-2 (2011): 40-50.

57. Martin PI., *et al.* "Overt naming fMRI pre- and post- TMS: two nonfluent aphasia patients, with and without improved naming post-TMS". *Brain and Language* 111.1 (2009): 20-35.

58. Naeser MA., *et al.* "Improved picture naming in chronic aphasia after TMS to part of right Broca's area: an open-protocol study". *Brain and Language* 93.1 (2005): 95-105.

59. Naeser MA., *et al.* "TMS suppression of right pars triangularis, but not para opercularis, improves naming in aphasia". *Brain and Language* 119.3 (2011): 206-213.

60. Rushworth MFS., *et al.* "The left parietal and premotor cortices: motor attention and selection". *NeuroImage* 20.1 (2003): S89-S100.

61. Sack AT., *et al.* "Imaging the brain activity changes underlying impaired visuospatial judgments: simultaneous fMRI, TMS, and behavioral studies". *Cerebral Cortex* 17.12 (2007): 2841-2852.

62. Torres J., *et al.* "TMS and tDCS in post-stroke aphasia: integrating novel treatment approaches with mechanisms of plasticity". *Restorative Neurology and Neuroscience* 31.4 (2013): 501-515.

63. Weiduschat N., *et al.* "Effects of repetitive transcranial magnetic stimulation in aphasic stroke. A randomized controlled pilot study". *Stroke* 42.2 (2011): 409-415.

64. Malá H., *et al.* "Prefrontal cortex and hippocampus in behavioural flexibility and posttraumatic functional recovery: Reversal learning and set-shifting in rats". *Brain Research Bulletin* 116 (2015): 34-44.

65. Mogensen J., *et al.* "Egocentric spatial orientation in a water maze by rats subjected to transection of the fimbria-fornix and/or ablation of the prefrontal cortex". *Brain Research Bulletin* 65.1 (2005): 41-58.

66. Mogensen J., *et al.* "Prefrontal cortex and hippocampus in posttraumatic functional recovery: Spatial delayed alternation by rats subjected to transection of the fimbria-fornix and/or ablation of the prefrontal cortex". *Brain Research Bulletin* 73.1-3 (2007): 86-95.

67. Krechevsky I. "'Hypotheses' in rats". *Psychological Reviews* 39.6 (1932): 516-532.

68. Krechevsky I. "Hereditary nature of 'hypotheses'". *Journal of Comparative Psychology* 16 (1933): 99-116.

69. Schulz M-A., *et al.* "Analysing humanly generated random number sequences: a pattern-based approach". *Plos One* 7.7 (2012): e41531.

70. Rosenberg S., *et al.* "Random number generation by normal, alcoholic and schizophrenic subjects". *Psychological Medicine* 20.4 (1990): 953-960.

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71. Díaz H., *et al.* "Order and chaos in the brain: fractal time series analysis of the EEG activity during a cognitive problem solving task". *Procedia Computer Science* 55 (2015): 1410-1419.

72. Gómez C and Hornero R. "Entropy and complexity analyses in Alzheimer's disease: an MEG study". *The Open Biomedical Engineering Journal* 4 (2010): 223-235.

73. Kannathal N., *et al.* "Characterization of EEG - a comparative study". *Computer Methods and Programs in Biomedicine* 80.1 (2005): 17-23.

74. Natarajan K., *et al.* "Nonlinear analysis of EEG signals at different mental states". *BioMedical Engineering OnLine* 3.1 (2004): 7.