# Neurobiologically constrained fronto-temporal-occipital computational model explains fast semantic mapping in the cortex

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Abstract

Human beings demonstrate a remarkable cognitive ability to acquire concepts in an instant (Carey & Bartlett, 1980; Pulvermüller, Shtyrov & Hauk, 2009). Recent research has begun to shed light on the neural basis of this phenomenon. We set out to further elucidate the fundamental neural mechanism through computational cognitive neuroscience. We deployed a fronto-temporo-occipital computational model and simulated for the first time fast mapping in the cortex. Our findings support models of fast mapping through cell assembly binding in the perisylvian language cortex.

# Acknowledgements

*Maxime Carrere & Victor O.*

*Dr. Max Garagnani*.

*If you wish to read a thought, you must first simulate the brain ~ Samora Hunter*

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# 1.0 Introduction

This manuscript reports a computational cognitive neuroscientific modelling of fast mapping in the context of language acquisition.

## 1.1 A brief thesis overview, in computational cognitive neuroscience

The uniquely human faculty of language plays a remarkable role in our lives, both as a means to author our own internal mental lives, detailing and categorising information, and as one to communicate the contents of our minds to others. Natural languages vary among peoples however, they all share some components, one such critical component is that of a vocabulary. Infants appear to begin with an empty vocabulary, but rapidly acquire lexical items and their semantic referent maps in the first few years of life (Miller, 1986). Teenagers continue the learning process they began as children, acquiring about three thousand words per year (Nagy and Herman, 1987). Adult's vocabulary ranges from ten to two hundred thousand words (Brysbaert, Stevens, Mandera & Keuleers, 2016) depending on the mode of categorisation used and of course typically continues to grow throughout their life.

The broad study of how human beings come to acquire such vast vocabularies is the subject of various overlapping fields of study, including but not limited to psychology, linguistics, neuroscience, cognitive science and computational linguistics. Several other fields investigate the acquisition of vocabularies without note nor goal of understanding human psychology and neurophysiology. Machine learning, natural language processing, and other areas of artificial intelligence are some of these, there however, the concern is with the algorithmic level of analysis, independent of implementation substrata. This paper is chiefly concerned with how the human brain achieves rapid language acquisition through rapid acquisition of words and rapid formation of concepts. Since we are concerned with humans and their brains, the constraints imposed by known neurophysiology are relevant to our desired level of understanding. Since the instantiation of meaning links cognition and linguistics, we are similarly interested in cognitive models. Lastly, in so far as we are invested in simulating as a means of understanding and describing emergent phenomena we too impose some artificial intelligence desideratum on our models.

In Psychology, the prevailing view of vocabulary at birth is that it is non-extant. Numerous psychological investigations have mapped its typical development from this *tabula rasa*. Of note here in the literature is a discussion regarding a critical period, its duration and neural basis (Friederici, 2017). At a high *psychological* level, vocabulary acquisition is built on word acquisition. Word acquisition itself is underlain by a mapping between an external environment referent and a sufficiently demarcating mental representation. The mental representation maps discretely to this lexical item though some category boundary overlap is common. This conceptual framing laterally extends to semantic acquisition, as the mental representation must map to the appropriate external environment referent. At the neural level, little light has been shed on this process, though it is thought to necessarily entail the binding of the neural information theoretic representation of the word and that of the appropriate object referent (Pulvermüller, 1999). This paper partially

replicates one such neurocomputational modelling of semantic or concept acquisition under this theoretical framework (Garagnani et al. 2016).

The *raison d'être* and specifically where this paper goes beyond mere replication is in its probing of the neural processes underlying the phenomenon known as fast mapping. Fast mapping, typically contrasted with explicit mapping is best characterised by the following; The latter refers to the explicit rote learning through repeated co-presentation of target and label and thus acquisition of a lexical items correct external environment referent map. Whilst the former to a phenomena of near immediate mapping between lexical item and external environment object referent. This has been demonstrated behaviourally entailing the leveraging of semantic/contextual information under what has become known as a fast mapping paradigm (Carey & Bartlett, 1980). The present paper reports an investigation into the neural basis of the initial description of fast mapping. That is, the putative neural mechanism underlying a rapid mapping of external object referent to lexical item, without direct reference to, nor experimental constraint imposed by, a fast mapping paradigm.

Definitive theories have yet to emerge from the scientific investigation into the general acquisition of linguistic semantic maps, a critical component of vocabulary, and of the neural basis of language. The prevailing view has been that the initial neural trace is first formed quickly in the hippocampus (Gaskell, 2009), and then the neural engram trace is transferred slowly to the cortex by a process of repeated relay during sleep (Sirota & Buzsaki, 2008). The anatomically defined cortex assumed as an associative memory structure obeying correlational learning represents this information in a sparsely distributed manner in distributed functional neuronal webs (Pulvermüller, 2002). Through a process of long-term consolidation these web structures store this information permanently (Lokendra Shastri, 2001).

Two key pieces of this puzzle do not fit. On the one hand, behavioural evidence suggests it is possible to rapidly learn a new word and its semantic referent map, yet on the other, the existing memory formation and consolidation process proposed is slow (Sirota & Buzsaki, 2008). Multiple recent studies imaging fast semantic representation formation in cortex under word learning paradigms have been recently published (Borovsky, Kutas, and Elman; Shtyrov & Pulvermüller, 2011). Further, evidence for a secondary system, independent of the hippocampal formation has been recently published showing fast mapping in hippocampal lesion patients (Sharon, Moscovitch, and Gilboa, 2011).

Motivated by the paucity of evidence, our ignorance of the mechanism we set out to further elucidate the putative neural basis of fast semantic mapping. It is our intention that this study will directly inform the current scientific debate (Duff & Warren, 2014; Greve, Cooper, Henson) regarding the plausibility of such a process, its locus in cerebrum, and the nature of the neural computations that give rise to the local processes psychological manifestations. Secondarily, that the findings will inform the wider communities investigations of the neural basis of language, learning through synaptic plasticity and information representation in the cortex. Peripherally, since the human being demonstrates natural language ‘semantic’ understanding and rapid few shot learning, investigations in artificial intelligence and machine learning pertaining to these phenomena may too find this study of interest.

The human brain is probably the most complex non-linear dynamic system in the known universe (Moore, 2010, Thaggard, 2002). Dependent on the physical level of detail one

attempts to model the system at, the information density may exceed the Bekenstein black hole entropic bound (Bostrom & Sandberg, 2008). The current approaches to its study are from arguably different disciplines, only loosely tied together under ‘neuroscience’. Neuroscience is producing extraordinary number articles. However, the resultant independent and combined findings lack a common parlance to integrate the disparate findings into coherent, meaningful and powerful explanatory models that can be communicated to the wider scientific community.

We therefore demonstrate by scientific simulacrum and simulation, through computational cognitive neuroscientific modelling. In this modus we explain the putative neural basis of our high-level psychological construct of fast mapping, by neurobiologically constrained low-level computationally modelling of cortex. Fast semantic mapping, our target phenomena, then emerges organically in simulation. This allows us to draw a causal line between the model architecture, parameters and input paradigm to the emergent intelligent behaviour. We then quantify these emergent mesoscopic dynamics, and propose them as the putative neural basis of these high-level psychological phenomena. Our demonstration through simulation unites these differing levels of analysis, and shows us the plausibility of, in our case, Hebbian distributed circuits underlying the observed fast semantic mapping phenomenon.

## 2.0 Background

### 2.1 Fast mapping (Psychology & cognitive science)

Why fast mapping? There are profound unanswered philosophical questions relating to the nature of the representation, acquisition and communication of meaning. Specifically its representation in brain, mind, language and external world (Fodor, 1957, Harnad, 2000, 2007, Searle, 1980, Turing, 1950, Barsalou, 2008, Landauer, & Dumais, 1997). These problems are magnified when one considers not only how these processes occur and exist in children’s brains with little experience in the world, without an already functioning language, with only a single example and almost immediately.

Perhaps the best framing of the challenge fast mapping poses to cognitive science is Quine´s (1960) indeterminacy of translation. In this thought experiment we imagine an explorer linguist on a strange island who sees a native pointing at a rabbit saying “Gavagai”. Upon reflection, we see there are a functionally infinite number of labels one could infer. The author then draws an important parallel between this scenario and that faced and apparently overcome by children when learning words in a single instance.

The literature reports a number of investigations with often starkly incongruent findings and contradicting terminology regarding fast mapping. A review is presented here with the purpose of clarifying terminology.

We begin with early psychological studies of fast mapping in children by Carey and Bartlett (1978), the behavioural results reported suggested that after single learning trial children were able to recall at least partly correct maps between referent object and word form when tested a week later. The authors notably posit a dual mechanism for word acquisition, one of fast mapping, the rapid partial acquisition of the semantics of a lexical item, and another of a slow process of consolidation.

Distinct from the theoretical formulation of the process, a fast mapping experimental paradigm generally refers to one in which target items labels are inferred from their use in context. In doing so, some element of the semantic component of the item is thought to facilitate the immediate memory formation via integration with existing semantic knowledge. At the neuro psychological level, this would require the novel items semantic properties be integrated with existing semantic networks that already have strong neural representation. In contrast, an explicit mapping paradigm entails the rote learning of the map via repeated presentation of its components (Coutanche & Thompson-Schill, 2014).

An example of a recent developmental psychological investigation of fast mapping can be found in Stowe & Hahn (2007). The researchers report what are essentially practice effects, with greater exposure to training label and referent object pairings inducing greater recall accuracy. This seems to support the semantic integration view, with the learning improvements supported by the existing representations of similar items.

Other studies have probed the veracity of such claims (Bartlett, 1980, Borovsky, Kutas, and Elman, 2010, Woodward et al., 1994; Schafer and Plunkett, 1998; Werker et al., 1998; Friedrich and Friederici, 2008; Torkildsen et al., 2008; Cooper et al., 2018; Dollaghan, 1985 Braisby, Dockrell, Best, 2001, Mervis & Bertrand, 1994, Medina, Snedecker, Trueswell & Gleitman, 2011) though debate persists. For a good overview of word acquisition and fast mapping, see Dockrell & Campbell, 1986; Heibeck & Markman, 1987; Bloom, 2000, and children's semantic acquisition see Wagner 2010. It is of note too here, that there is evidence of the same fundamental mechanism underlying fast mapping in language, underlying other kinds of learning (Markson & Bloom, 1997).

## 2.2 Vocabulary and words (Linguistics)

Linguistics is the formal study of language. Each system of the circa seven thousand human languages is broken into component parts of phonology, phonetics, morphology, syntax, semantics and pragmatics for specialist study. Languages consist of a vocabulary and a grammar. The vocabulary or lexicon consists of the sum of lexemes of the particular language, essentially all extant words in the language. The grammar is then concerned with the rules governing the meaningful use of these words (David Crystal, 1995). The instantiation of meaning in a word is more specifically studied under the sub field of semantics (Semasiology), an area at the intersection of cognition, linguistics and philosophy.

## 2.3 Neuroscience of language (Neuroscience, physiology)

A complete overview of the neuroimaging of language is beyond the scope of this paper. The state of scientific inquiry may be found in Pulvermüller (2003), *The Neuroscience of Language: On Brain Circuits of Words and Serial Order*, (Friederici, 2017) *Language in the brain* and *Handbook Neuroscience of language* (Stemmer & Whitaker, 2008). More specifically relating to the representation of words and acquisition of language at the neural level can be found in Pulvermüller (1999) *Words in the brain*.

## 2.4 Neuroanatomy of language

The most robust finding in investigations of the functional neuroanatomy of language is that of the near ubiquitous functional lateralization of language processes to the left hemisphere of the cortex (Vigneau et al., 2010; Friederici, 2017). The earliest cortical links to auditory function underlying language are those reported by Kussmaul 1877 and Lichtheim 1885 linking pure word deafness to bilateral lesion in transverse temporal gyri of Heschl BA41. Then followed two key functionally defined regions of Broca 1861 *BA44/45* and Wernicke's 1874 *BA22*, the former associated with speech production and the latter with comprehension (Friederici, 2011). Anatomical studies have then uncovered the arcuate fascicle *AF*, a fiber tract connecting the regions (Deferine, 1895) and later studies, particularly utilising diffusion tensor imaging (Catani & Schotten, 2008) have identified two routes between these pertinent regions. These dorsal and ventral pathways form a dual stream model (Hickok, Poeppel, 2004). Similarly, gyri encompassing these regions are posited to form a network, inferior frontal gyrus, superior temporal gyrus and middle temporal gyrus (Friederici, 2011). These findings have been greatly evidenced by a combination of lesion studies of stroke patients, patients with developmental disorders, functional imaging, structural imaging, vivisection, cognitive science and computational modelling. Combined they give us the current 'Perisylvian language network' (Friederici, 2011). The collation of finer functional imaging has more specifically localized most crucial language processes to an area that has come to be known as Perisylvian cortex. This is the area about the Perisylvian fissure, and spans prefrontal, frontal temporal and parietal cortices.

## 2.5 Neuroscience of word and concept acquisition

Functional neuroimaging of word, concept and semantic acquisition has followed a similar path as other phenomena in cognitive neuroscience. It began with early techniques of electroencephalography and event related potentials Kutas & Hillyard (1980) indexed the processing of semantic components of words as what is now well known as the N400 ERP component. Later study has sought more detail; particularly regarding spatial localisation (Friedrich, Friederici . 2004, 2005, 2005; Pulvermüller, 2002). Notable recent models posit semantic hub networks governing the processing and representation of semantics (Patterson, Nestort & Rogers, 2007) and attempt to account for the various classes of lesion patient's differential patterns of cognitive deficits. For a recent review see Pulvermüller, Shtyrov understanding in an instant (2009).

## 2.6 Cognitive neuroscience and imaging of fast mapping

Several experiments report functional imaging of rapid word and concept acquisition. McLaughlin, Osterhout and Kim (2004) report N400 amplitude responses to novel foreign language words in second language learners reaching those of known words. Here the researchers suggest that at least some aspect of foreign language word acquisition is rapid, and is complemented by a slow laborious process. Mestres-Missé, Rodriguez-Fornells and Münte report N400 amplitude changes to novel words matching those of known words after just three learning presentation trials in which meaning was inferred from context. Shytrov in 2011 similarly reports ERP measurements of online rapid language acquisition, finding elicited 100ms fronto central amplitudes reaching equilibria for novel words to that of known within twenty minutes (Shytrov, 2011).

More recently and rather controversially one study reports evidence of fast mapping in profoundly amnesic patients with hippocampal medial temporal lobe lesions. The

researchers further dissociate the hippocampus as the neural locus of initial association formation from the cortex by showing the failure of fast mapping in two patients with unilateral damage to the left polar temporal neocortex (Sharon, Moscovitch, and Gilboa, 2011). This study's findings have been hotly contested

## *2.7 Hebb's postulates, cell assembly theory (Cybernetics)*

The present scientific investigations approach to neural information processing is framed in a fuzzy school of thought 'Cybernetics' that differs sufficiently from some others to warrant a brief description here. The model itself is endowed with a learning rule that is responsible for much of the emergent behaviour of the system. Ultimately, the resulting explanation of the neural basis of the phenomena is realized at this neural circuit level. It is therefore pertinent to describe too what has been termed cell assembly theory and its relation to language. It is also convenient and perhaps rightfully ought to constrain our later conclusions.

In 1949, Donald Hebb postulated a mechanism by which synaptic strengths come to change (Hebb, 1949). It is best captured by the mnemonic,

*"Cells that fire together wire together"*

Where the delta update rule to the weight between neuron A with an average firing rate of $V_A$ neuron B with an average firing rate of $V_B$, the simplest form Hebbian learning can be expressed as:

$$\Delta\omega_{BA} = \varepsilon V_B V_A$$

*EQ 1.0*

Various further formulations of this rule exist, notably a class of which allow for negative synaptic weights. This provides a stabilising mechanism, and captures the processes of both long-term potentiation and depression.

The model deployed in this investigation implements one of such learning rules, first described by Artola Bröcher and Singer (Artola, Bröcher & Singer, 1990). The ABS rule follows from an earlier BCM theory and 'BCM rule' (Bienenstock, Cooper and Munro, 1982) where synaptic weights are updated according to:

*BCM rule*

$$\Delta\omega_{ij} = \epsilon(f^{BCM}(r_i;\theta^M)(r_j) - f(\omega))$$

*EQ* 2.*0*

*Simplified ABS rule*

$$\Delta\omega_{ij} = \epsilon(f_{ABS}(r_i;\theta^-,\theta^+)sign(r_j - \theta^{pre}))$$

*EQ* 3.*0*

*Current model implementation of ABS*

$$\omega_{t+1}(x,y) = \begin{cases} \omega_t(x,y) - \Delta\omega & if\ \mathrm{O}(x,t) > \theta_{pre}\ and\ V(y,t) > \theta_{post}(LTP) \\ \omega_t(x,y) + \Delta\omega & if\ \mathrm{O}(x,t) \leq \theta_{pre}\ and\ V(y,t) > \theta_{post}(LTD) \\ \omega_t(x,y) & otherwise \end{cases}$$

*EQ 4.0*

This learning principle provides a mechanism for neurons that are repeatedly co-activated to become associated, and thus form networks of what have come to be known as cell assemblies. Several general properties of these theoretical constructs have emerged, with varying degrees of experimental and neurophysiological support. The earliest and most studied properties are reverberation, the persistence of activity after the stimulus has been withdrawn. Ignition, the reverberatory activation of cells across the network upon sufficient stimulation. Pattern completion, memory recall from incomplete information. Rivalry, competition among cell assemblies for ignition upon receipt of input (Hebb, 1959). Lateral inhibition, cells inhibiting nearby cells in loops (Milner, 1957). Lastly, phase sequence, a mechanism for connecting stimuli occurring in close temporal proximity (Pulvermüller, 1999).

### *2.8 Semantic grounding*

The study of meaning in the philosophical sense predates the inquiries of neuroscience and psychology, and perhaps study relating to language too. To this day there is much confusion obfuscating terminology in the varying disciplines that investigate it. In the study of language, meaning is synonymous with the linguistic term of semantic, and thus the study of meaning in language is the focus of the sub field of semantics. Cognitive science has then through a psychological lens tried to explain the mental representation and processing of linguistic semantics.

The predecessor to this paper cites two competing cognitive theories of the basis of meaning acquisition in this linguistic sense (Garagnani & Pulvermüller, 2016). One of '*amodal*' symbolic semantic feature interrelations between words as the basis for the representation of meaning. And another, of *semantic grounding*, in which meaning emerges from tangible reference to the external world (Barsalou, 2008). The latter approach was assumed and it is within this school of thought that that experiment was framed. In congruence, the present paper remains under this cognitive framework.

### 2.9 One shot learning (Machine learning)

Traditional approaches to a seminal problem of classification (pattern recognition) in machine learning typically require a great deal of training examples from which a mapping between input data *x* and output label *y* is eventually learned by the network. This is typically done in "supervised learning" by employing an algorithm such as back propagation (Rumelhart, Hinton & Williams, 1986). Training examples are fed into a network consisting of artificial neurons, nodes, connected by artificial synapses, weights, and through an iterative process errors are calculated by a measure of distance between the desired output,

and the networks function mapping result for each pass. The synaptic weights are then updated typically using gradient descent such that the error is gradually lessened. There has recently been a great deal of interest in so-called one shot learning in these fields. These are networks that can generalise from data in a single, or few steps.

## *3.0 Neural models of language acquisition (Computational modelling)*

The analogy of the human brain to a computer is an old idea that at least predates the relatively modern twenty first century Turing machines and Von Neumann architecture (Turing, 1950; Von Neumann, 1947). It is however a far more recent enterprise to computationally model cognitive functions, and even more recent to instantiate such models with known neuroanatomical features (Kriegeskorte & Douglas, 2018). The latest of these models combine cognitive models with computational architectures, incorporate known neuroanatomical features of the biological brains and use artificial intelligence to simulate behaviour.

First, some definitional matters are in order. Purely cognitive models describe cognitive processes at the level of information processing, without necessarily regard given to the substrate on which such processes run. They are also often purely descriptive, rather than explanatory. They too err toward symbolic representation in modules or units, rather than distributed representations across say neuronal networks. In doing so they lose many of the advantages of distributed representations such as pattern completion with degraded input, fault tolerance and many other (Hinton, McClelland, & Rumelhart, 1986).

It is useful to review a selection the evolution of model class applied in the study of the neural basis of language to contextually frame and effectively contrast the choice of model deployed here.

One of the earliest models of the earliest class of cognitive models is that proposed by Geschwind (1979). Here a series of functionally connected modules was used to model hearing a sentence and repeating it back, a supposed fundament of language comprehension. The model proposed the process was initialised by simulated linguistic signal input to auditory cortex in temporal lobe, relay from here to Wernicke's area, and then relay over anatomical highway arcuate fasciculus to Broca's area for generation of articulation motor schema (Macwhinney & Li, *Handbook of neuroscience of language*, p230).

Of additional note are a class of probabilistic models of language acquisition taking a somewhat different approach to word acquisition than that taken here, specifically in their theoretical framework of cross situational learning (Alishahi, Fazly & Stevenson, 2010; Vlach & Sandhofer; Abend, Kwiatkowski, Smith, Goldwater & Steedman 2017; Yurovsky, Yu, 2014 & Smith; Xu & Tenenbaum, 2007 ). These may be dissociated from the present approach employed given that they do not employ neural dynamic modelling; however, they are of great use in providing top down frameworks that inform the accuracy of the low-level model aspects of our approach.

## 4.0 Materials & Methods

The present investigation deployed a neurocomputational model of the Perisylvian language cortex. The model simulates neural population dynamics in several areas thought to be relevant to the acquisition of basic semantics/words/concepts. The model is an abstraction of the necessary and sufficient functional properties of the cortex thought to give rise to the phenomena of interest. This is particularly important when considering the emergent 'intelligent' behaviour it exhibits under our training paradigms.

The present investigation partially replicates a previous deployment of a neurocomputational model in the investigation of semantic grounding and concept acquisition (Garagnani & Pulvermüller, 2016). Macro and micro architectural features of the model were preserved however; the experimental paradigms and simulation parameter values have some crucial differences. Notably and pertinent to the purpose of this paper, the investigation extends beyond the previous work and advances the frontier.

The previous investigations model specification is an architectural extension of an earlier model. The earliest version of the model consisted of six identical areas (micro structural connectivity is as described here and see figure 2) with connectivity structure mimicking known anatomical connectivity structure of the left perisylvian language cortex. It was used to simulate early phonological and articulatory correlation in early language learning processes and neurophysiological response. The experimental paradigm entailed concomitantly presenting pairs of activation patterns to model input areas corresponding to primary auditory cortex and primary motor cortex. These areas were hypothesised to be coactive during early word learning "babbling" phases. Further details can be found in Garagnani, Wennekers and Pulvermüller (2007).

Later research extended the model architecture from these six areas (primary auditory cortex, auditory belt, auditory parabelt, inferior prefrontal, premotor and primary motor cortex) and 'babbling' phenomena to 12 area architecture and semantic grounding/concept acquisition phenomena(see fig 2). Here the model repeats the previous six perisylvian areas but includes six more Extrasylvian, consisting of a sensory input area model of early visual cortex, temporo-occipital, anterior temporal, dorsolateral prefrontal, premotor and primary motor cortex. As before the model area links were of identical strength, and artificial cellular dynamics were governed by the same equations.

The previous investigation then simulated the acquisition of concepts within a semantic grounding framework. In this perspective, acquisition of meaning of a basic set of words requires a referential link between the words 'form', its phonological and articulatory representation, and the type of either object or action that word is commonly used to refer to. The examples commonly given in this vein of literature are that of the action word "grasp", typically being learned in the context of the performance of the grasping action. Similarly, object words such as "table", being learned when visually identifying the object. Learning of these basic words in this framework would then require a referential link between at least some component of the word form "table" and a visual representation of that object. Most notably a mechanism to realise this link has been proposed by Pulvermüller, whom proposes a neural correlate at the *mesoscopic* cell assembly level; he terms a distributed Hebbian circuit.

The motor action word "grasp" has been shown physiologically to differentially activate motor cortex, particularly hand area motor cortex, contrasted with visual object words such as table, producing comparatively greater activity in visual occipital areas (Garagnani & Pulvermüller, 2016).

The previous investigation simulated the formation of these referential links at the cell assembly level of information representation. It did so by deploying a neurocomputational model simulating areas of cortex involved in the formation of these referential links for some basic words. Then, simulating the repeated co-activation of cells in these regions with either articulatory-phonological and motor regions receiving repeated correlated activation patterns or articulatory-phonological and visual regions receiving these activation patterns. It was shown that this neurobiologically constrained computational model under this experimental paradigm exhibited the emergence of cell assemblies exclusive to the area input regions modalities. That is to say, cell assembly topography varied as a function of the input area under training. It was also shown that the triplet modality independent activation patterns produced cell assembly extension into hub regions, heavier overlap in hub regions and formation into coherent distinct cell assemblies exhibiting the properties of the aforementioned referential links.

The present investigation deploys an identical architecture and initially identical parameter values for the initial phase, phase one. The extension of the investigation toward the frontier lies in the experimental paradigm, and nature of the phenomena investigated. Where the previous study has simulated the co-occurrence of neuronal activation patterns in regionally distinct but sparsely connected areas, we simulated first the independent formation of cell assemblies by phonological-articulatory activation and visual or motor activation, then the subsequent binding of these into a single cell assembly in a ‘one shot episode’. This was meant to simulate the fundamental mechanism underlying fast mapping.

### *Preamble and rationale*

In partial replication of prior deployment of the model and since it is our intention to simulate the fundamental mechanism not emulate it, we generated twelve activation patterns per network training instance. Patterns were randomly and arbitrarily generated upon network initialisation, however they remained fixed throughout the entire experiment.

These patterns were segmented by their training paradigm into three classes defined by the input region they were presented to, visual patterns, motor patterns and articulatory-phonological patterns. In the initial phase, six patterns fell into the class of articulatory-phonological patterns, three into ‘visual’ and three into ‘motor’.

Specific model areas were included following a similar rationale to Garagnani & Pulvermüller (2016). In this work, the area corresponding to inferior motor cortex’s $M1_i$ inclusion was justified as neuroimaging findings show its activation during the production of speech. Similarly, the area corresponding to primary auditory cortex *A1* was too included as similar imaging results show its simultaneous activation in the reception of produced speech.

We include them here and present activation patterns simultaneously to both input regions, allowing network activity to propagate about the artificial cellular links governed by the Hebbian learning implemented. This is meant to simulate the production of sounds and simultaneous hearing of these sounds, this concomitant nervous activity was expected, by Hebbian learning to form distributed circuits of strongly connected cells spanning the two regions. The two other input regions in our model at this phase receive uncorrelated randomly

generated activation patterns; this simulated the absence of a *teaching* or *grounding* signal during this 'babbling' or speech and added to the general noise inherent in the nervous system.

Again, in the prior work the area corresponding to early visual cortex *V1* inclusion was justified as neuroimaging findings show its greater activation in response to words with a greater visual component, "perception" words (Garagnani & Pulvermüller, 2016). Presentation of activation patterns to this region were then meant to simulate seeing an object. We include this area here too as we are concerned with the same semantic grounding process of concept acquisition, however at this stage we present three of twelve activation patterns under a network entrainment paradigm only to this area, whilst uncorrelated randomly generated activation patterns are presented to the remaining three input areas. This is meant to simulate seeing an object in the absence of a co-occurring auditory label.

Finally, as in the prior work, the area corresponding to dorsolateral motor cortices $M1_L$ inclusion was justified as neuroimaging findings show its greater activation in response to "action" words. Presentation of activation patterns to this region then simulate activity resulting from self-performance of a motor action. We include this area here too in partial replication of this prior study, however as with the "perception" words, we present three of twelve activation patterns under our network entrainment paradigm exclusively to this area, whilst simultaneously uncorrelated randomly generated activation patterns are presented to the remaining three input areas. These uncorrelated patterns and white noise overlay simulate the variability in inputs and general neuronal noise.

### *4.1 Phase one*

**α**: Phonological - articulatory association input areas were presented with concomitant correlated activation patterns.

$$\textbf{[Pattern] > A1 <>AB <> PB <>< } \mathbf{PF_i} \textbf{ <> } \mathbf{PM_i} \textbf{ <> } \mathbf{M1_i} \textbf{ < [Pattern]}$$

Six coupled activation patterns were presented to area A1 and simultaneously to $M1_i$ for 3000 presentations each. The remaining two input areas V1 and $M1_L$ simultaneously received uncorrelated randomly generated patterns.

*P1 α*

**β**:

$$\textbf{[Pattern] > V1 <> TO <> AT <>}$$

Three independent activation patterns were presented to area V1 for 3000 presentations each. The remaining three input areas V1, $M1_L$ and $M1_i$ simultaneously received uncorrelated randomly generated patterns.

*P1 β*

**γ**.

$$\mathbf{M1_L} \textbf{ <> } \mathbf{PM_L} \textbf{ <> } \mathbf{PF_L} \textbf{ <> [Pattern]}$$

Three independent activation patterns were presented to area $M1_i$ for a total of 3000 presentations each. The remaining three input areas V1, $M1_L$, and A1 simultaneously received uncorrelated randomly generated patterns.

Through a previously used method of quantifying cells that constitute assemblies we observed three distinct classes of cell assemblies, these were defined by their spread across the network areas. These were then quantified in a test phase by presenting the patterns to their respective input areas defined during the training phase and cells firing above a predefined threshold of 0.5 were counted. The number of active cell assembly cells are reported in the results section (See fig 1). Following phase one we halted network training and initialised the second phase.

## *4.2 Phase two*

### *One shot learning phase*

Each network instance was then subjected to the *one-shot* phase. Here only the auditory activation pattern component of the auditory/articulatory pattern couple (P1 α) and one activation pattern from either motor or sensory (P1 β or P1 γ) were presented to their respective network areas. This was meant to simulate simultaneously hearing the word and, performing the motor action or seeing the object.

**δ**:

For each network instance, three of the six-pattern sets one, three and five auditory activation pattern components utilised in training under P1 α was presented to input area A1. Simultaneously, for each individual auditory pattern component, one of three motor activation patterns trained under P1 β were presented to input area V1. The remaining input areas $M1_L$ and $M1_i$ received uncorrelated randomly generated patterns. In this phase, activation patterns were presented for two simulation time steps per presentation instance. Each pattern couple was presented for an average of 15 or so times. Global inhibition parameter and input region noise were reduced to 50 and 1 respectively.

**ε**:

For each network instance, three of the six-pattern sets two, four and six auditory activation pattern components utilised in training under P1 α was presented to input area A1. Simultaneously, for each individual auditory pattern component, one of three visual activation patterns trained under P1 γ were presented to input area $M1_L$. The remaining input areas V1 and $M1_i$ received uncorrelated randomly generated patterns. In this phase, activation patterns were presented for two simulation time steps per presentation instance. Global inhibition parameter and input region noise were reduced to 50 and 1 respectively. Each pattern couple was presented for an average of 15 or so times, though network parameter exploration showed evidence of binding with as little as five presentations. This was meant to simulate hearing the word and simultaneously seeing it. When viewing an object or hearing a sound the sensory input should persist over a matter of seconds justifying a greater than one number of simulation pattern presentations.

Cell assembly structures in this second phase were quantified by presenting only the auditory component of the phase one α activation pattern for two simulation time steps and quantifying the number of cells firing above 0.5 threshold.

### 4.3 Attention manipulation by global inhibition modulation (Computational neuroscience)

Previous research into the effects of attention on language acquisition has identified the role of global inhibition levels amongst cortical networks. (Garagnani & Pulvermüller, 2008). The model implemented here simulates global inhibition through interactions between excitatory and inhibitory cells in the dual laminar micro connectivity structure of the network. The specifics of which are detailed further in the materials section of this paper and can be found exactly in the 2008 paper.

During the initial phase of the simulation the global inhibition parameter remained fixed and at a value utilised by the researchers in 2016 to improve comparability and faithfulness of the replication. However, during the second phase in order to simulate the effects of attention during word learning we lowered the global inhibition parameter significantly. This had the effect of increasing cell assembly binding propensity and experimental exploration indicated that it was responsible for a great deal of the reduction in training presentations needed to bind the separate cell assemblies. We were interested to explore the effects of this parameter change as previous theory and experimentation (Pulvermüller, 2001, p502) suggests that the attention is not necessary to activate cortical semantic memory traces.

## 5.0 Model specification

The following is a brief description of the pertinent features of the neurocomputational model's specifications, adapted from Garagnani, Pulvermüller and Wennekers (2008) and Garagnani (2016).

The model consists of twelve identical network areas of dual layer inhibitory and excitatory 25 x 25 graded response artificial neural cells, 625 excitatory, 625 inhibitory for 15,000 cells in total.

Each *in silico* excitatory cell modelled, through mean field “graded” output response, circa 25,000 *in carbo* cortical pyramidal cells. Each of which is supposed to capture, at abstraction, the dynamics of a cortical column, functionally and anatomically bounded as a cortical computational unit. Whereas each inhibitory cell counterpart captures the dynamics of a cluster of inhibitory neurons in the same computational unit.

This made for a total *in silico* excitatory cell model of 375,000,000 *in carbo* excitatory cells. And roughly 75,000,000 inhibitory cells.

Each simulation step equates to approximately 18.75ms real time.

Activation state of each excitatory cell is modulated by its membrane potential $V(e, t)$. This captures the average of all post synaptic potentials in cortical computational unit $e$ at time $t$.

$$\tau \cdot \frac{dV(e,t)}{dt} = -V(e,t) + k_1(V_{ln}(e,t) + k_2\eta(e,t))$$

Where *V(e,t)* is the total input to cell *e* at time *t,* $\tau$ is the membrane's time constant, $k_1$ and $k_2$ are scaling constants and η(e, t) is uniformly distributed white noise applied over the interval [-0.5, 0.5].

Output (transformation function) of each excitatory *e* cell at time *t* is implemented as:

$$O(e,t) = \begin{cases} 0 & if\ V(e,t) \leq \varphi \\ (V(e,t) - \varphi\ if\ 0 < (V(e,t) - \varphi) \leq 1 \\ 1 & otherwise \end{cases}$$

O(e, t) captures the average firing rate of unit *e* at time *t.*

Forward and backward excitatory between area synaptic links, and recurrent 'within area' synaptic links were generated according to a Gaussian distribution with a maximal radius of five, decreasing to zero as a function of distance from originating cell.

Each sensorimotor pattern consisted of a set of 19 cells per primary area (57 cells in total), randomly selected amongst the 25-by-25 cells forming one area (about 3% of cells).

A full specification for the model is available in Garagnani & Pulvermüller, 2016.

### *5.1 Parameter values*

5.1.1Phase one:

Time constant (Excitatory cells): $\tau$ = 2.5 (simulation time-steps)
Time constant (Inhibitory cells): $\tau$ = 5 (simulation time steps)
Scaling factor: $\kappa_1$ = 0.01
Baseline potential $V_b = 0$
Noise scaling factor $k_2$ = 25 √48
Global inhibition strength $k_2$ = 65
(During training): $k_s = 95$
Adaptation strength: $\alpha$ = 0.01
Average output time constant
(For adaptation mechanism): $\tau_A$ = 10 (simulation time steps)
Global inhibition time constant: $\tau_S$ = 12 (simulation time steps)
Postsynaptic potential threshold
Required for synaptic change: $\theta_{post} = 0.15$
Presynaptic output activity $\theta_{pre} = 0.05$
Required for LTP
Learning rate: $\Delta\ \omega = 0.0008$

5.1.2 Phase two:

Time constant (Excitatory cells): $\tau$ = 2.5 (simulation time-steps)

Time constant (Inhibitory cells): $\tau$ = 5 (simulation time steps)
Scaling factor: $\kappa_1$ = 0.01
Baseline potential $V_b$ = 0
Noise scaling factor $k_2$ = 1 √48
Global inhibition strength $k_2$ = 65
(During training): $k_s$ = 50
Adaptation strength: $\alpha$ = 0.01
Average output time constant
(For adaptation mechanism): $\tau_A$ = 10 (simulation time steps)
Global inhibition time constant: $\tau_S$ = 12 (simulation time steps)
Postsynaptic potential threshold
Required for synaptic change: $\theta_{post}$ = 0.15
Presynaptic output activity $\theta_{pre}$ = 0.05
Required for LTP
Learning rate: $\Delta \omega$ = 0.0008

## 6.0 Results

### *6.1 Phase one*

Following the initial training phase, we presented each pattern one by one to the input region they had been previously repeatedly presented to during the training phase. This was done in the absence of uncorrelated inputs to ease quantification. The subsequent cell assembly ignition was examined on a per area basis, and was quantified by counting the number of artificial cells firing above a predefined threshold. These cells are taken to be the constituent parts of the cell assembly, the network of strongly reciprocally connected cells once postulated by Hebb.

**α**:

Under the articulatory/auditory α paradigm the greatest number of cells were found in the Perisylvian and Extrasylvian central hub regions; parabelt, prefrontal ventral, anterior temporal and prefrontal dorsal. Compared to the visual paradigms cell assembly spread across Extrasylvian areas under **γ,** fewer number of cells in the visual area network periphery early visual cortices and temporal occipital were found. Similarly compared to the motor paradigms cell assembly spread across Extrasylvian areas under **β,** fewer number of cells in motor area network periphery dorsal motor and dorsal premotor areas. Lastly, compared to both **β** and **γ**, greater number of partaking cells were found in perisylvian peripheral network areas, primary auditory, auditory belt, premotor ventral and premotor dorsal areas.

**β**:

Under the motor β paradigm the greatest number of cells were also found in the central hub regions, however a large number of cells were also found in dorsal motor and dorsal premotor. By contrast, there were far fewer cells found in early visual cortices and temporo occipital.

**γ:**

Under the visual γ paradigm the greatest number of cells were too found in the central hub regions, however a large number of cells were also found in early visual cortices and temporo occipital.

*6.2 Phase two*

Following the initial training phase, we presented concomitantly one auditory pattern component of patterns 1, 2, 3, 4, 5, 6 and simultaneously alternating between one of either motor 7, 8, 9 or visual 10, 11, 12 dorsal grounding patterns. For example auditory activation pattern component of pattern 1 from phase one α presented to auditory input area A1 simultaneously with visual activation pattern 7 presented to visual input area V1. In doing so, each pre-trained auditory-articulatory cell assembly was stimulated concomitantly with one either visual or motor cell assembly for a total of six pairs. Patterns were in this paradigm presented for only two simulation time steps per presentation; in contrast to 16 simulation time steps in phase one. Each of the pairs was presented for an average of 15 times per network instance. Results in figure 2 were averaged within network instance for cell assembly class and then averaged across 12 network instances. Pattern 1, 3 and 5 were bound with pattern 7, 8 and 9 respectively. Pattern 2, 4 and 6 were bound with pattern 10, 11 and 12 respectively.

**δ:**

Under δ motor grounding paradigm a greater total number of cells partaking in cell assemblies stimulated by patterns 1-6 were found compared to their original post pre trained state under α. This greater number is taken as good evidence that binding has occurred between the two assemblies from α and β. This is further confirmed by the greater number of cells in $PM_D$ and $M1_D$ in Extraslyvian areas under δ when compared with Extraslyvian areas under α. Further exploratory research showed no cell assembly formation when this paradigm was run from a naïve untrained network.

**ε:**

Under ε visual grounding paradigm a greater total number of cells partaking in cell assemblies stimulated by patterns 1-6 were found compared to their original post pre trained state under α. This greater number is again taken as good evidence that binding has occurred between the two assemblies from α and γ. This is further confirmed by the greater number of cells in TO and V1 in Extraslyvian areas under ε when compared with Extraslyvian areas under α.

7.0 Discussion

*7.1 Phase one*

The results from phase one support the hypothesis that cell assemblies would form topographically distinct structures across network areas dependent on input modality simulated. It is clear from figure one α that auditory-articulatory activation patterns produced cell assemblies that spanned the entirety of the perisylvian areas, exhibited high node recruitment in hub areas, and did not spread greatly into peripheral Extrasylvian areas. We predicted this based on the previous study (Garagnani & Pulvermüller, 2016), which found that uncorrelated pattern presentation inhibited cell assembly growth. In our case under α, both Extrasylvian peripheral input areas received uncorrelated 'noise' patterns. The great number of cells found in hub areas can too be explained as in the prior work, by a high probability of connections in those areas and the reverberatory behaviour of the neural networks sustaining activity post input.

The results from phase one β and γ were also as predicted; the strong peripheral node recruitment in both cases was biased toward the input region their activation patterns were presented to.

### *7.2 Phase two*

The results from phase two supported the hypothesis that the distinct pre trained cell assemblies would bind after just a few co activations, and form supramodal cell assemblies. The cell assemblies spanned perisylvian areas and their respective grounding peripheral area with striking resemblance to the topography seen in the previous 2016 research. It is of great note that the previous research required several thousand-presentation triplets to three network input areas to produce the same supramodal cell assembly topography. With just a few co-activations of auditory/articulatory cell assembly and a target semantic grounding counterpart, we see strong evidence of binding. Given the biased distribution of the independent cell assemblies' nodes to central hub regions, we speculate that this binding is greatly facilitated by the decreased proximity and thus greater probability of connectivity in these regions.

## *8.0 Concluding remarks*

Exploratory probing of these network structures revealed the cell assembly properties previously described by Hebb and later by Pulvermüller such as reverberation and pattern completion. Interestingly and masked by averaging in the figures provided was the inconsistency in pattern binding. It was apparent that at a low number of presentations in the one shot phase our cell assemblies exhibited a binary state of bound and not bound. This behaviour resembles the inconsistent learning seen in Carey and Bartlett, (1980) in which learners under fast mapping had about fifty percent recall rates. Further research could investigate this further, in search of a 'binding event horizon'.

The resultant three distinct structures of phase one could be thought of as simulating pre-existing semantic knowledge networks described by theorist's cognitive descriptions of fast mapping (Coutanche & Thompson-Schill, 2014). This is somewhat plausible for basic words, through a semantic grounding perspective and Garagnani & Pulvermüller's experimental paradigm; however, it is difficult to see how this could extend to more complex concept acquisition. Further research would do well to pursue this, as probably the most interesting and distinctly human cognitive phenomena are those of complex abstract concepts.

Regarding the binding of the cell assemblies and the phenomena of fast mapping, we can first ask whether this mechanism of leveraging existing associative memory structures for the rapid formation of new concepts, necessarily requires a fast mapping paradigm. Alternatively, whether it is just as plausible to claim it occurs under explicit learning too. The conservative view is of course that it is plausible under both paradigms, since we cannot really be sure that all words or concepts require such neat grounding such as that employed here. Further, that other types of words might utilise structures not modelled here, and as such would have different patterns of cell assembly topography. Their systems may then evolve quite differently. We do however show that this is plausible for some cases, and this in of itself is quite congruent with the inconsistent performance of humans when learning via fast mapping.

Whilst the results have been positive and predictions confirmed, some critical evaluation of the models fitness for purpose is in order. We first argue in congruence with the original study that we have sufficiently good reasons to abstract away much of the finer and more computationally expensive aspects of neural dynamics. We submit that since we are interested in the neural mechanism in its fundament, that it is superfluous to increase the macro scale of the simulation. It is also the constraint of cell assembly theory that bounds our scale to its mesoscopic level. That said, a particularly exacting view would be concerned about non linearity's and emergent phenomena in the real brain that could potentially perturb the evolution of the system as it is currently simulated if it were to be closer to emulation. These unaccounted for potential dynamics pose a threat to the validity of experiments such as this one.

On a more positive note, the model deployed here has several key advantages over others, most notably the broad class of probabilistic models of word acquisition previously mentioned. This advantage is in the neurobiological realism implemented in the architecture; this is a much more powerful explanatory mechanism than logical or probabilistic approaches. It similarly has advantages over more top down symbolic models such as that proposed by Geschwind (1979) as it accounts for the low-level features and has the high level behaviour and structures emerge from simpler connected units, rather than be hand crafted.

Further research is certainly needed given the disparities in behavioural and developmental studies of fast mapping; perhaps a large enough sample would settle the issue once and for all. There is also room for interesting analysis to be done on the finer mathematical and statistical properties of these cell assemblies, and at the intersection of neuroscience, machine learning and artificial intelligence with respect to one shot learning.

We conclude by submitting that the present results demonstrate the plausibility of fast mapping directly in the perisylvian language cortex, independent of the hippocampus, and that general neural mechanisms combined with the typical architecture of language cortex support this ability.

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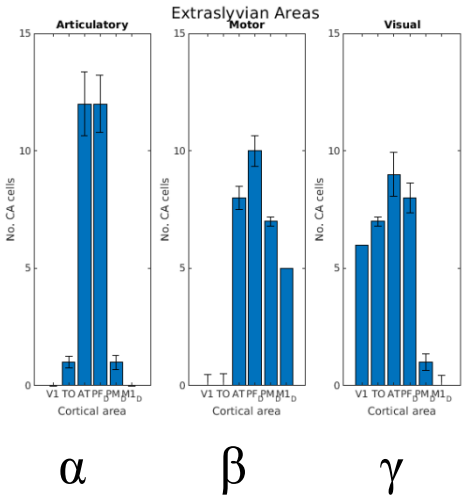
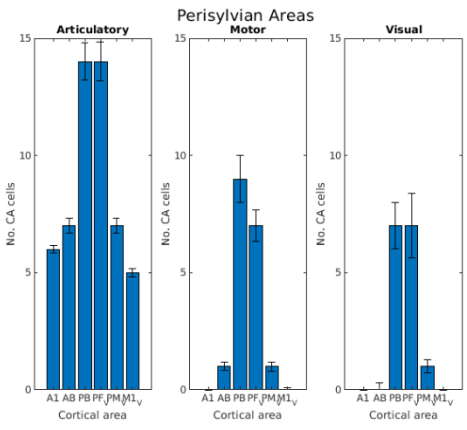
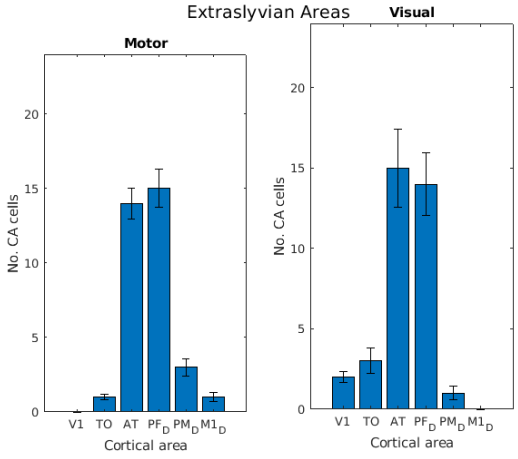
10.0 Phase one Figure 1 Appendix Figure2

Phase 1: Initial *Training*




Phase 2: *Post few shot*



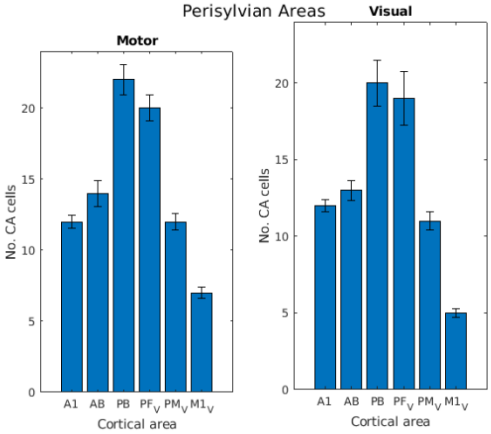



*Figure 3*

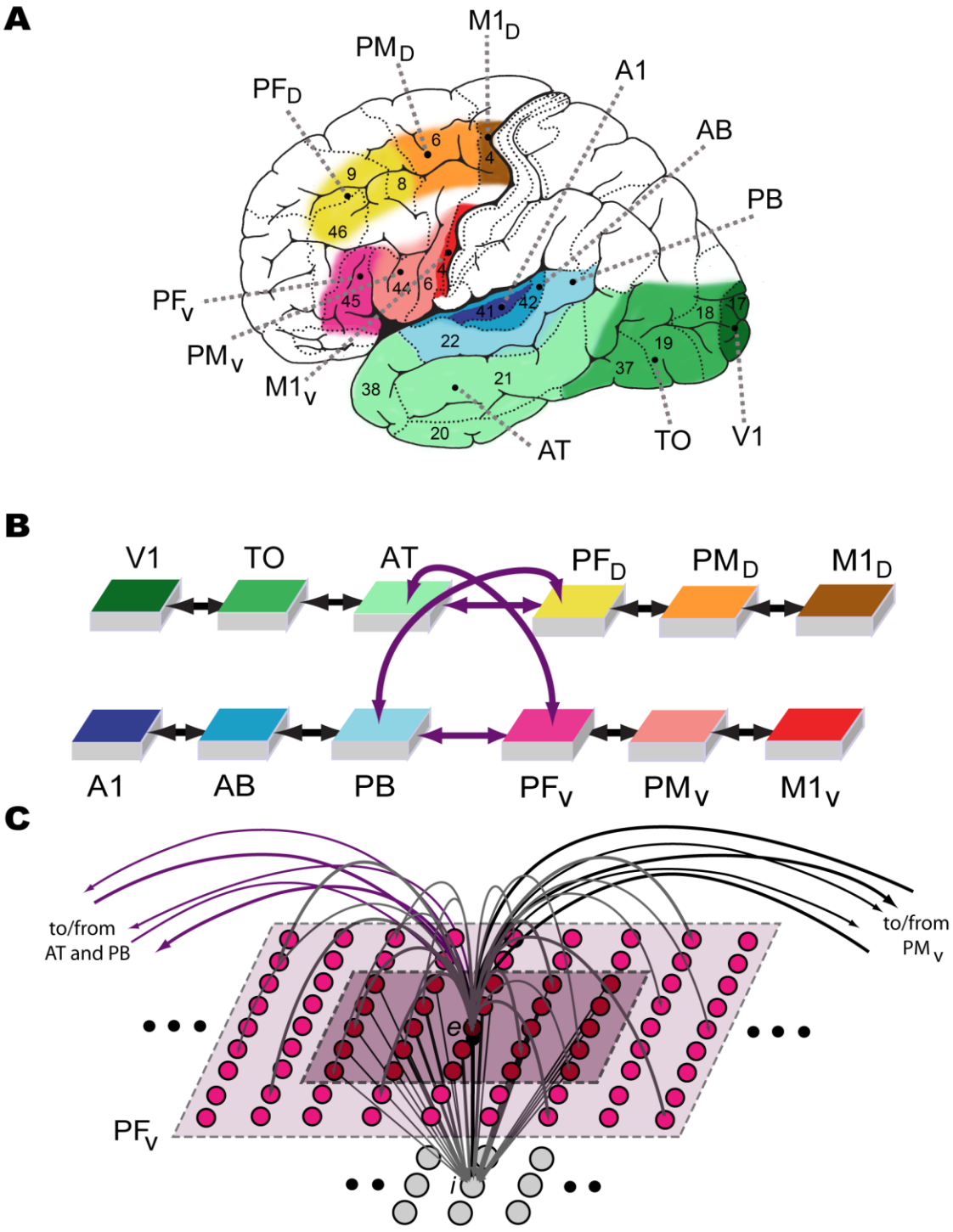



*Figure adapted from Garanani, 2016 ©