Synthesis of Neonate Connectomes for Artificial Sentience and Common Sense

Michael Bihn
Dept. of Computer Science
University of Colorado at Colorado Springs
Colorado Springs, USA
email: mbihn@uccs.edu

Rory Lewis
Dept. of Computer Science
University of Colorado at Colorado Springs
Colorado Springs, USA
email: rlewis5@uccs.edu

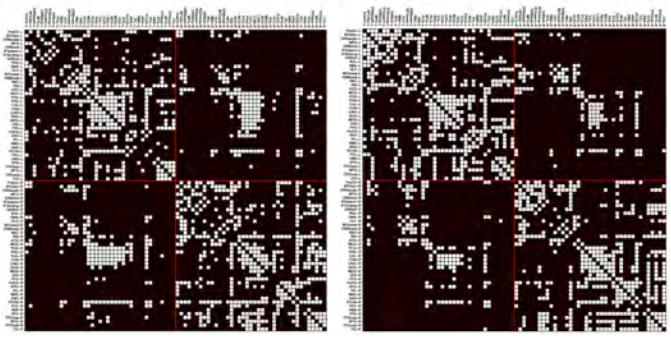
***Abstract*—In the ongoing research effort of synthesizing sentience into artificial intelligence, we propose a modular network that emulates neurological synaptic evolution in the neonate brain. Our hypothesis is that if one were to successfully develop a synthesized emulation of human's six-month hippocampus as it initializes adult-like glucose usage and synaptic density which is generally accepted in the domain of neuroscience as being the foundation of human sentience, then so can human sentience be injected into the synthesized replication of said six-month hippocampus. Accordingly, we present a theoretical proposition that facilitates a significant step towards overcoming the commonsense challenge that state-of-the-art artificial intelligence systems are still grappling with today; where even the most powerful artificial intelligence systems are void of the common sense of a three year old: That lemons are sour, that things fall towards the ground and that they, as children, can pretend to be somebody else. Herein, we present a methodology to efficiently promulgate the research goal of integrating sentience and common sense reasoning into artificial intelligence, taking a neurological rather than a psychological approach.**

***Keywords*—Sentience, Common Sense, AI.**

I. Introduction

In 1766, Immanuel Kant theorized that human knowledge is a combination of priori knowledge where knowledge is acquired independently of any particular experience, and posteriori knowledge, which is derived from experience that we reason from our senses being affected by our surrounds [1]. Nowadays, it is accepted that to achieve common sense processing, an entirely new method will need to be invented [2] [3] and, that this new method will require priori and posteriori knowledge [4]- [5]. The design of a posteriori knowledge component shall seamlessly communicate with the artificial intelligence system. In 2020, Shanahan *et al.* [6] examined the common sense of animals and concluded that there must exist, in each animal, some innate knowledge that allows them to learn without words.

We postulate that, because common sense in humans and animals require priori and posteriori knowledge, so should we design sentient machines. Our premise is that, to emulate human and animal common sense, one needs to mathematically emulate developments in neuroscience which will include discovering that the directionality of brain waves in the cortical regions of the brain form different frequency bands [7], that functions emerge from the flow of information linking distant cortical regions [8] that a modular network topology is present in the brain from the first days of life. [9] [10] and that the complexity of our multi-connected connectome network [11] has been decoded [12].



(a) (b)

Fig. 1. Connectivity Matrice: Mapping connectome evolution in 78 cortical regions, excluding subcortical and cerebellar regions, in the brains of a) two-week, and (b) one-year old infant humans [13]. The white squares represent connectivity between the lobes of the horizontal and vertical axis.

A. Neuronal Pathway

We focus on the neuronal network between regions of the brain, as defined by Automated Anatomical Labeling (AAL) for length and local efficiency [13] where Yap *et al.* used a connectivity matrix to group neuronal regions into three distinct communities, as shown in Fig. 1. When comparing the synaptic evolution on the matrices from a cohort of two week old and one year old children, one observes a complex neuronal mesh comprised of multiple additions and pruning of the network. Fig. 2 illustrates how integrating the Kamada-Kawai layout with the Pajek software package [14] shows three distinct neuronal communities [13].

Recently, Fornito *et al.* showed that connectomes have an inheritable complex topology that suggests a genome-wide association that can be either excitatory or inhibitive [16]. Rosenburg *et al.* showed that at ~6 months, the hippocampus has adult like glucose use and synaptic density [17] and Szalkai *et al.* built four consensus brain graphs from a cohort of 106 individual brain graphs and set directions by popular

 ISBN: 978-1-61208-885-3

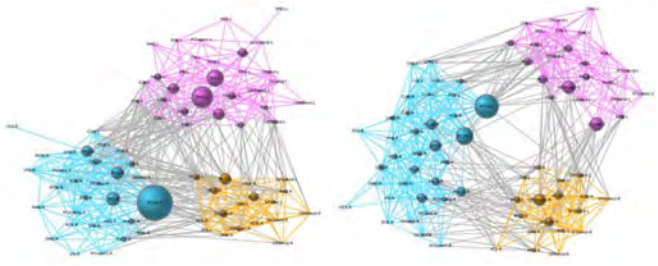


Fig. 2. Spring Embedding Visualization: Where the sizes of the vertices are weighted by Yap *et al.* using Freeman's logarithmically scaled node betweenness algorithm, [13] [15] .

TABLE I
THE PATTERNS OF CHANGE

Pattern	Number		Pattern	Number
000	2120		100	161
001	64		101	28
010	53		110	39
011	136		111	402

vote and in so doing, proved that i) axons have directionality from the soma to the end of the axon, and that, ii) 82% of the directions were the same for all four groups [18].

B. Neuronal Pathway Insights

At the biological level, the authors first accept that neuronal pathways are constantly changing over time with rapid myelination occurring in the first year of life [13]. Secondly, the authors acknowledge that brain modules (highly connected groupings of brain lobes) at two weeks old and one year old are not the same. Thirdly, the authors accept that using connectomes is the state-of-the-art for mapping these pathways yet, unfortunately, it is evident that, in essence, using a two dimensional array, that has no geometric similarity with the brain, at all, to track these neurological events is a shortcoming. It is here that the authors found their motivation to procure what is in this paper; a more robust and precise means to emulate, represent and measure in a computer the neuronal pathway evolution that occurs at the biological level. It should also be noted that Yap *et al's* 3-D representations of the neuronal pathways suggest that i) there are hubs inside the modules that are most likely connected to many lobes in that module, that ii) there are bridges with edges which connect the hubs of different modules, and that iii) a non hub leaf node in one module may never connect to a non hub leaf node in another module.

C. Neuronal Instantiation

The authors note that because lobe-to-lobe connections are initialed as early as two weeks, only to be turned off at one year, and then turned back on again at two years, while others are turned off at two weeks, turned on at one year, and then off again at two years [13], the authors have focused on studying whether this seemingly random and chaotic process has patterns that when found will enlighten researchers in this domain as to how human sentience is formed, and have said formation replicated synthetically in a machine. The numbers inherent in each of these patterns are represented in Table I. The first bit of the pattern is dependent on the two week connectome, the second bit represents the one year connectome and the third represents the two year connectome. We note that there are 2120 possible connections that never occur and 402 connections that never break. Additionally, it is of interest that the connectomes have 630 connections and that the number of connections added from two weeks to one year is 189, which is also the number disconnections from two weeks to one year. The number of connections added from one year to two years is 92, which is also the number of disconnections from one year to two years. This reflects the slowdown of myelination over time. Yap *et al.* have found the growing efficiency of the brain in [13].

The rest of the paper is structured as follows. In Section 2, we present our research objectives. In Section 3, we present our experiments. In Section 4, we present how we plan to move from a prototype to the real model. We make our conclusion in Section 5.

II. RESEARCH OBJECTIVES

The aforementioned research has lead the authors to answer four questions. 1) What is the purpose of the changing lobe to lobe connections? 2) Is there a development phase to the neuronal pathways? 3) Is there an initialization phase? 4) At which connectome do humans start independent thinking and if so, what pathways are crucial to this independent thinking?

A. Transversal Definition

We start by defining the Transversal propagation of connectivity and pruning by focusing on forming a means to measure the path distance from one lobe to the next and present (1) and (2):

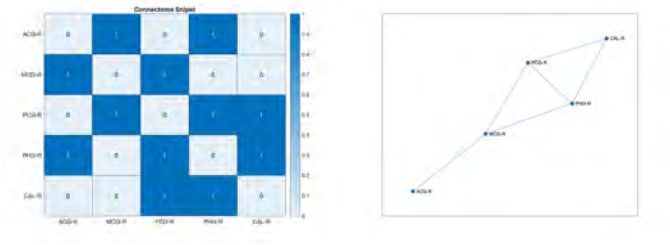
$$SSP = SingleShortestPath(lobe_i, lobe_j) \quad (1)$$

$$T_i = \sum_{j=1, j\neq i}^{78} SSP(i,j) \quad (2)$$

where $lobe(i)$ and $lobe(j)$ are the brain lobes as indexed by i and j from the automatic anatomical labels provided by Montreal Neurological Institute [13]. The Single Shortest Path is the length of the single shortest path defined in the connectome graph preliminaries. Additionally, we define Ti, transversal of the ith node, as the sum of all the shortest paths from the ith lobe to the jth lobe excluding the path to from the ith lobe to itself. 78 is the number of brain lobes in our connectome. Each of the 78 brain lobes is fully connected to all the other 77 lobes in the brain. Accordingly, we reference Fig.3 (b) and determine the single shortest path from the right Anterior Cingulate Gyrus lobe (ACG-R) to the right Median Cingulate Gyrus lobe (MCG-R) is one.

The single shortest path from ACG-R to the right Posterior Cingulate gyrus lobe (PCG-R) is two. For ACG-R to the right

 ISBN: 978-1-61208-885-3



(a) (b)

Fig. 3. Connectivity: This shows a snippet of the connectome and the graph produced. (a) connectome, and (b) graph

Parahippocampal gyrus lobe (PHG-R) it is also two. However, for the ACG-R to the right Calcarine cortex (CAL-R) there are multiple paths, the shortest being a length of three. T_ACG-R is the transversal of node ACG-R and is defined by 2 as the sum of all the SSP of A, therefore T_ACG-R=1+2+2+3=8.

$$MaxTransversal = \max(T_i, i = 1..78) \quad (3)$$

$$MinTransversal = \min(T_i, i = 1..78) \quad (4)$$

where the $MaxTransversal$ is defined as the maximum of traversals, see (2), of all the nodes transversal, i stepping through the lobes from 1 to 78 lobes, as shown in(3). $MinTransversal$ is defined as the minimum of all the nodes transversals, defined in (2), as shown in (4). Once again this calculation looks at the lobes i from lobe 1 through lobe 78, similarly to the $MaxTransversal$.

B. Defining Maximum Traversal Length

We have determined that a common denominator to correctly replicating human sentient neurological evolution in a machine, lasers in on how accurately one can define and measure the maximum traversal length. The maximum traversal length on the two week connectome is 257; see 3. The maximum traversal for the one year connectome is 219, and 212 for the two year connectome. We examine the maximal transversal of 257 at two weeks, 219 at one year and 212 at two years. Preliminary findings show that, while the connectome is reducing the maximum length path over time, *at the same time*, the minimal transversal path is increasing from 107, at two weeks, to 127 at one year, and 128 at two years. It is also interesting and not understood why the number of connections remains stable at 630 during this period.

C. Optimizing Maximum Traversal Length

The optimization of maximum transition length with the constraints of pairs of connections (connection, disconnection) over time and stable 630 connections over time could lead to a model that predicts the connectome development. Of course, more data and analysis is necessary to order the transitions of the connectome. This gives us an optimization problem, namely, minimize the maximum transition length, with the constraint of stable number of 630 connections. This optimization is how we propose to predict which transitions will occur. Left unbounded, the model would keep pruning and connecting, thus we shall just look at the best 189 from two weeks old to one year old and compare our predictions to the actual changes that occur. We will then start with the one year old and execute the same optimization, this time only taking the best 92 pairs of disconnect and connect.

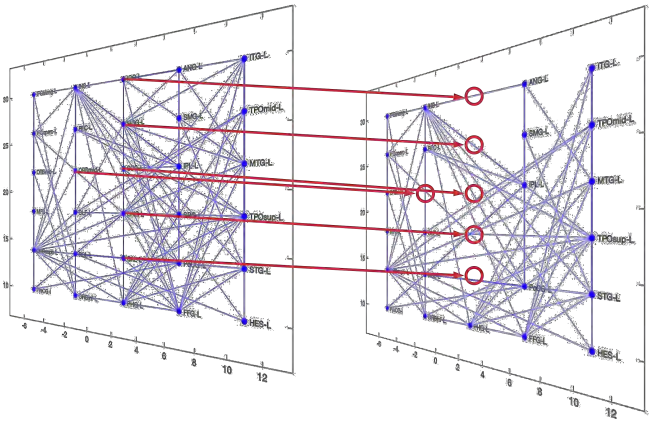


Fig. 4. The red lines emphasize a few lobes moving out of Module one

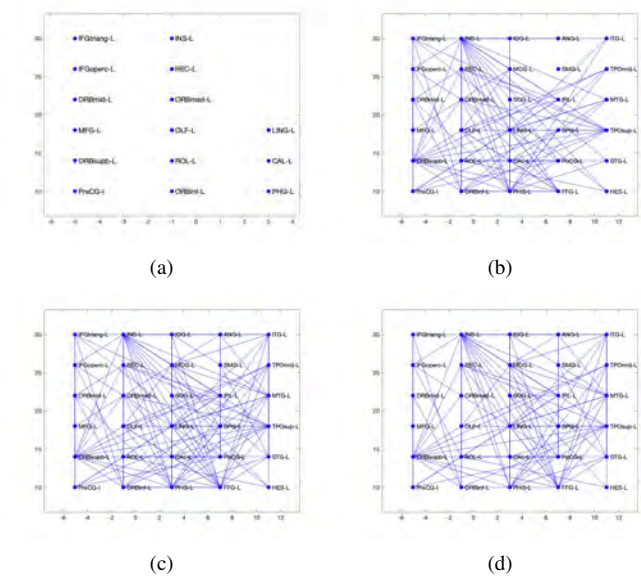
A further challenge is matching the modules at different ages. Fig. 4 shows how certain brain lobes will move from one module of the brain to another. This occurs due to the changing connections those lobes have with other brain lobes. This figure provides the reader with insight to view the changes in the figures of the next section. In the next section, we examine the development of the connectome from conception to one year old. Noting the changes that occur just inside the first lobe for simplicity of explanation. The changes that occur across all 78 lobes, modifying from 3 brain modules to four brain modules at two years old are too complex to put on one sheet of paper. The actual lobes involved in particular changes are details to be reconciled by our predictions.

III. Experiments

The authors hypothesize that it is possible to measure the path from any node to another node in terms of the number of edges traversed through the connectome. Our justification for this proposition is that because we know that as the brain develops from three to four modules, it is logical to expect that the total number of edges traversed is decreasing. To formulate our methodology we base our experiments off of the following determinate. Fig. 4 shows the detail that should be noted as stepping through the determinate.

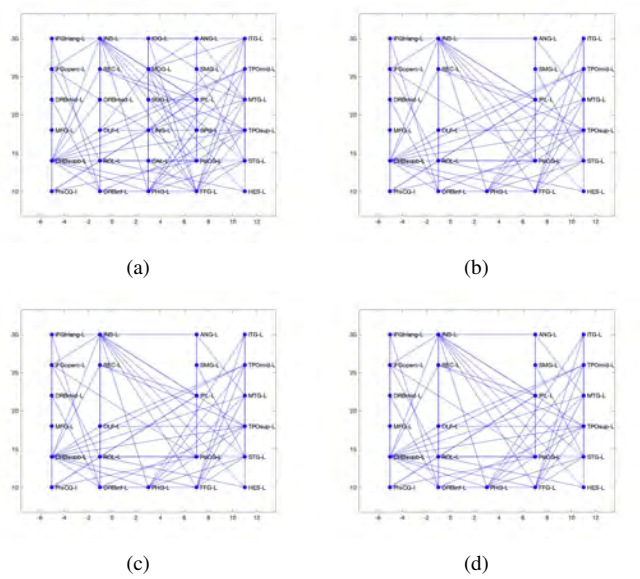
1) At conception no brain lobes exist.
2) Over time, the brain lobes form and connections are established.
3) Fig. 5(a) shows the first brain lobes appearing in module one of the brain.
4) Fig. 5(b) shows the neuronal pathways being formed between some of the existing lobes in module one of the brain. The same growth is occurring in the second and third modules of the brain.

 ISBN: 978-1-61208-885-3



(a) (b) (c) (d)

Fig. 5. Connectome Proliferation: Isolating the first highly connected lobes of the module 1 of the brain, we see the following stages (a) Module one shows initial lobes appearing. (b) all lobes in module one appear with first synapses, (c) first brain module complete at two weeks, and (d) after two weeks, initial edge pruning occurs.



(a) (b) (c) (d)

Fig. 6. Connectome Pruning (a) After two weeks, edge pruning completed. (b) After two weeks, some nodes break away from module 1. (c) After two weeks, all nodes to break away from module 1, have done so., and (d) At one year, the first new node moves into module 1.

5) Fig. 5(c) shows the the connectome lobe 1 completed at two weeks of age.
6) Once the two week connectome is formed, we know that connections will be broken and others formed leading to the one year connectome. Fig. 5(d) shows synaptic connections being broken. While the prototype shows multiple edges dropping, we note i) that this is only module one of the brain and ii) it is highly likely, that while a synapse/edge is disconnected, another synapse is formed, maintaining the full connectedness of the connectome and the 630 connections.
7) Fig. 6(a) shows all the edges dropped that are not in the one year connectome. While this gives the reader an overview of how the connectomes are changing over time. One should note that even though it may appear that all the lost edges are dropped before adding nodes and edges to achieve the one year connectome, evidence shows that this is *not* what happens in the human brain. The lobes *do not go away* but rather connect to another module of the brain. The edge modifications are most likely intermingled. We need to know if a neuronal pathway is dropped if the myeline becomes available to form another synapse/edge.
8) Fig. 6(b) shows nodes moving out of module 1 between two weeks and one year. A node switches modules when it becomes more connected to the nodes/lobes of the new module then its current module.
9) Fig. 6(c) shows the rest of the nodes moved out of module 1 and into another module. This is the final node/lobe state that is expressed in the one year connectome.
10) Fig. 6(d) shows a node from another module moving into module 1.
11) Fig. 7 shows how at one year lobes from other modules have become more connected to the module 1.

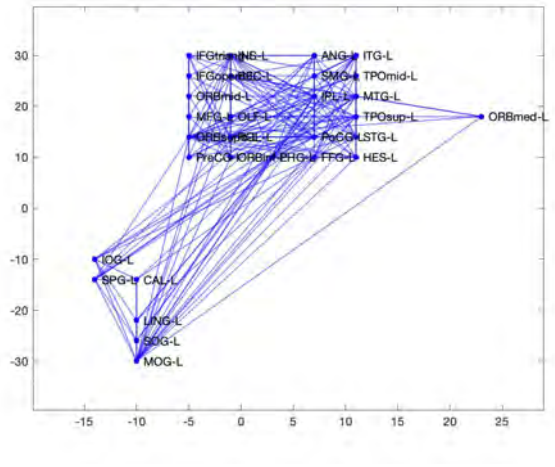


Fig. 7. Nodes and edges are added to complete module 1 for the one year connectome

The ordering of these modifications is most definitely not occurring en masse, not all edge deletions, then all node deletions, then all node additions, then all edge additions. As already stated the lobes do not go away, but rather become connected to one of the other modules. Therefore, the nodes should not go away, but rather be connected to lobes in another module of the brain. Noting that from one year old to two years old a forth module appears. The graph with all 78 lobes/nodes and 630 synapsis/edges is incomprehensible. The connections are created when the synapse strength had crossed a certain

threshold. The connection is broken when the synapse strength drops below that threshold.

The rapid myelination in the first year eventually slows down. We need some measure of this myelination availability and usage in forming a synapse to be able to predict when the next neuronal pathway can be formed. If the myeline is not available, the synapse and neuronal pathway will not be formed. We know what connections exist at two weeks old. With enough data we should be able to measure the total length of these pathways and determine the myeline needed to form them. This could lead to a side study on what mechanism provides the myeline and what factors such as nutrition, O2, and time influence said mechanism. Simultaneous growth in the three lobes could provide competition for resources. We need to measure the myeline production and usage over time. Only then can we use regression analysis (Time Series) to plot the brains resources availability and use over time. Once these curves can be predicted, then they can be used to spot deviation in individuals brain growth. These deviations can then be catalogued, accumulated and used for finding disease or giftedness.

Another possibility is to find an arbitrary variable for the synaptic length growth potential over time. Rather then measure the myeline availability measure the total synapse length over time. [13] provides average synapsis length over time, an average implies a sum divided by a count. If this data is available, we should be able to regress the growth in number and growth of length of the synapsis over time. These growth rates along with a scripted ordering of synapsis connectivity will then predict when connections are built.

Fornito *et al.* [16] suggest that the DNA explicitly controls the brains growth. This infers that if there was a resource problem the brain may not have formed properly, potentially altering the expectations of a DNA study. In which case, it may be better to compare the connectome development to disease expression, rather than the DNA to disease expressions. This leads the authors to conclude that it raises the importance of researching *how* the brain is formed rather than studying the DNA process at the neurological level.

IV. From Prototype to the Real Model

o model the real connectome development we dynamically mimic four constraints, 1) the maximal traversal minimization over time, 3) that there are always 630 connections, 3) that the average synapse length is always increasing, and 4) that the connectome remains fully connected.

In order to accomplish this, we shall implement connectome changes in pairs, one disconnect paired with a connect. These pairs shall be created by ordering all the connects in increasing synapse length and the disconnects in increasing synapse length. Then, pairing the smallest connect with the smallest disconnect, to give the list of pairs to be implemented over time. We then shall ensure that the connectome remains fully connected over time. The pairs of actions(connect/disconnect) will need to be examined for orphan creation, where a node/lobe has a degree of 1 and that link is being deleted.

TABLE II
The Action Table

Action	the action to be taken connect or disconnect
LobeFrom and To	the lobes being connected or disconnected
Day and Time	the projected day and time the action will occur
Length	the length of the connection
TimeToComplete	the time necessary to complete the action

Since we know that the connectome stays fully connected, we know there is another connect in the list that reconnects that node/lobe. The two actions shall then swap partners, such that the disconnect and connect will maintain that particular node/lobe inclusion in the connectome, and maintain the fully connectedness of the connectome. To maintain the full connectedness, the connection will be created before the disconnect.

Once the actions list is refined, the actions can then be implemented over time. The rate of increase of myelin availability applied to project when the next action will occur. That is, when there exist enough myeline available to satisfy the net gain of pathway creation. We shall build the action table, see Table II, to provide the time ordered list of connectivity growth.

The length of synapsis should be known. [13] shows the average synapse length is increasing over time. The average is the sum of all the lengths divided by the number of synapsis. In future experiments we may find the synapse growth rate could replace the myelin availability function, or provide the myeline available function with another variable. Considering a biological process is not instantaneous, we shall set a length of time to complete the action.

The rate of myeline availability or rate of synapsis length growth shall be determined by regression analysis. We have the data points that from two weeks to one year there are 189 pairs of actions taken. While we do not know what the pairs are, we do know what the 189 disconnects are, and we do know what the 189 connects are. From one year to two years there are 92 pairs of action, likewise we know the 92 connects, and the 92 disconnects. With only two points we can only have a linear function. Research into the ConnectomeDB will provide more defined pairs of actions over time, thus more data points for the regression. Several regression attempts (linear, quadratic, polynomial, exponential, logarithmic) shall be executed to find the best correlation coefficient, r.

Assessment of the Accuracy of the algorithms developed will be possible when more connectome data becomes available. The National Institute of Health (NIH) is sponsoring a Baby Connectome Project that began in 2016 where the data will be available to NIH sponsored researchers [19].

V. Conclusion and Future Work

In considering where neuroscience research has lead we reiterate our original concept which was to produce a minimal ontology, recognizing that a toddler has minimal common sense and must learn it from experiences. In pursuing the neuroscience, we find the connectome, a representation of the

 ISBN: 978-1-61208-885-3

brains fully connected network between 78 cortical regions. But rather than having a stable connectome as a child, the connectome is more stable as an adult. What we have run into is the innate process of building the networked brain. The neuroscience shows us the result of the innate process of building the brain, but does not, as of yet, show us how it is built.

A systematic approach to define the requirements of synthesizing the brain needs to be taken. Mapping known capabilities to cortical regions and corresponding tool that has been built or needs to be built. Evaluating all existing tools for input, processing and output. All inputs shall be received from the network that simulates the connectome. All outputs shall be delivered to other cortical consumers through the synthesized connectome. The collective process shall mimic our notion of common sense. What started as an attempt to minimize the scope of the common sense problem has led us to the extensiveness of the brains innate development in the first years of life. What we set upon to build is not stable, but rather time dependent, adding a fourth dimension as we observe the connectome evolve to its adult stability.

This research has revealed that the max traversal shortens with time. In essence, the shorter max traversal provides a shorter path from one brain lobe to all the other brain lobes. This quicker transmission of brain signals provides humans with an increasing brain speed as we age. This in turn provides us a mathematical means to differentiate between "*quick wittedness*" versus "*not the sharpest tool in the shed*". Both of these phrases are common judgments of a person's level of common sense. We have determined that a common denominator to correctly replicating human sentient neurological evolution in a machine, lasers in on how accurately one can define and measure the maximum traversal length.

For our future research we will be studying how Prescott *et al.* [20] have built their humanoid robot, named ICub, based on psychological division. Prescott proposes using the human cognitive architecture, yet they take a psychological self approach to their brain inspired control architecture. However, they make no mention of connectomes, or the latest neural networks of the brain. Conversely, the authors have full faith that studying and replicating the neurological approach is more realistic and shall prove more fruitful in the long term.

REFERENCES

[1] I. Kant, "The possibility of experience," *Journey into Philosophy: An Introduction with Classic and Contemporary Readings*, p. 42, 2016.

[2] E. Davis and G. Marcus, "Commonsense reasoning and commonsense knowledge in artificial intelligence.," *Commun. ACM*, vol. 58, no. 9, pp. 92–103, 2015.

[3] R. Zellers, Y. Bisk, R. Schwartz, and Y. Choi, "Swag: A large-scale adversarial dataset for grounded commonsense inference," *arXiv preprint arXiv:1808.05326*, 2018.

[4] S. A. Bini, "Artificial intelligence, machine learning, deep learning, and cognitive computing: what do these terms mean and how will they impact health care?," *The Journal of arthroplasty*, vol. 33, no. 8, pp. 2358–2361, 2018.

[5] A. Talmor, J. Herzig, N. Lourie, and J. Berant, "Commonsenseqa: A question answering challenge targeting commonsense knowledge," *arXiv preprint arXiv:1811.00937*, 2018.

[6] M. Shanahan, M. Crosby, B. Beyret, and L. Cheke, "Artificial intelligence and the common sense of animals," *Trends in Cognitive Sciences*, 2020.

[7] F. Babiloni *et al.*, "Estimation of the cortical functional connectivity with the multimodal integration of high-resolution eeg and fmri data by directed transfer function," *Neuroimage*, vol. 24, no. 1, pp. 118–131, 2005.

[8] P. Bartolomeo, "The quest for the critical lesion site in cognitive deficits: problems and perspectives," *Cortex*, vol. 47, no. 8, pp. 1010–1012, 2011.

[9] P. Hagmann, M. Kurant, X. Gigandet, P. Thiran, V. J. Wedeen, R. Meuli, and J.-P. Thiran, "Mapping human whole-brain structural networks with diffusion mri," *PloS one*, vol. 2, no. 7, p. e597, 2007.

[10] O. Sporns, *Networks of the Brain*. MIT press, 2010.

[11] A. Sokolov Arseny, G. Cristina, F. Elda, P. Giulia, R. Philippe, V. Dimitri, and K. J. Friston, "Brain network analyses in clinical neuroscience," *Swiss Archives of Neurology, Psychiatry and Psychotherapy*, vol. 170, no. 6, 2019.

[12] E. Fischi-Gomez, E. Muñoz-Moreno, L. Vasung, A. Griffa, C. Borradori-Tolsa, M. Monnier, F. Lazeyras, J.-P. Thiran, and P. S. Hüppi, "Brain network characterization of high-risk preterm-born school-age children," *NeuroImage: Clinical*, vol. 11, pp. 195–209, 2016.

[13] P.-T. Yap, Y. Fan, Y. Chen, J. H. Gilmore, W. Lin, and D. Shen, "Development trends of white matter connectivity in the first years of life," *PloS one*, vol. 6, no. 9, p. e24678, 2011.

[14] V. Batagelj and A. Mrvar, "Pajek-program for large network analysis," *Connections*, vol. 21, no. 2, pp. 47–57, 1998.

[15] L. C. Freeman, "A set of measures of centrality based on betweenness," *Sociometry*, pp. 35–41, 1977.

[16] A. Fornito, A. Arnatkevičiūtė, and B. D. Fulcher, "Bridging the gap between connectome and transcriptome," *Trends in Cognitive Sciences*, vol. 23, no. 1, pp. 34–50, 2019.

[17] A. Rosenberg, *How history gets things wrong: The neuroscience of our addiction to stories*. MIT Press, 2019.

[18] B. Szalkai, C. Kerepesi, B. Varga, and V. Grolmusz, "High-resolution directed human connectomes and the consensus connectome dynamics," *PloS one*, vol. 14, no. 4, p. e0215473, 2019.

[19] B. R. Howell, M. A. Styner, W. Gao, P.-T. Yap, L. Wang, K. Baluyot, E. Yacoub, G. Chen, T. Potts, A. Salzwedel, *et al.*, "The unc/umn baby connectome project (bcp): An overview of the study design and protocol development," *NeuroImage*, vol. 185, pp. 891–905, 2019.

[20] T. J. Prescott and D. Camilleri, "The synthetic psychology of the self," in *Cognitive architectures*, pp. 85–104, Springer, 2019.

 ISBN: 978-1-61208-885-3