Dimensions of Kernel Abstractions
A Theory on Neurobiology and Cognitive Functioning

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Abstract

The ability to create abstract categorizations to be reused in different contexts is one of the most critical abilities for intelligent behavior. In this letter, we present a proposal for how neurobiological structure plays a role in this ability. We introduce two terms: kernel and kernel dimension. The first to describe what the abstraction is and the second to numerically quantify the first. Based on the common abnormalities in both biological structure and cognitive functioning of two psychiatric disorders, autism and schizophrenia, we hypothesize that the density of the dendritic spines in the pyramidal

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neurons of the Prefrontal Cortex determines the dimension. We show the computational validity of our claims by making two demonstrations with artificial intelligence - one to show that structural density can effect kernel dimension and the other to show that cognitive functioning is effected by the dimension of the kernel.

Introduction

Anatomically, the Prefrontal Cortex (PFC) is located at the front of the brain and has connections to all of the brain’s major subdivisions and their constituents (Kandel et al., 2012; Miller & Cohen, 2000) and can communicate with each of them (Fuster, 1999). Due to this, the PFC has been hypothesized by many as being the site of the brain that is responsible for the abilities that allow for intelligent behavior. Miller & Cohen (2000) described the PFC as being a railroad operator that is in charge of deciding how to move the existing tracks during different circumstances, rather then being responsible for creating the tracks. As a necessary means to being able to achieve this, the PFC must learn how to reuse lessons from past experiences in new and different contexts. This ability has been described by many in terms such as categorization, representation, or abstraction (Genovesio et al., 2005; White & Wise, 1999; Cromer et al., 2010; Antzoulatos & Miller, 2014, 2011; Miller & Cohen, 2000). Although this concept has been widely researched, the much about the specific mechanisms behind it are not understood. To borrow from the metaphor used by Miller & Cohen (2000), there has been little progress in understanding how the conductor decides the number of tracks that are moved for a given train. In this paper, we propose a theory for explaining this, which is
that certain structural properties of the PFC - the density of the dendritic spines of the pyramidal cells - determine how much information is encoded into an abstract category, or the kernel. We introduce two new terms for formally analysing abstractions, kernel and kernel dimension.

**Kernel and Kernel Dimension**

As part of our proposal, we define the terms kernel, and kernel dimension, as a means to formally quantify abstract visual categorization that can be used in different contexts to carry over and reuse learned properties, rules, and restrictions. Continuing on the railroad metaphor, the kernel could be described as being the set of tracks that the conductor moves for a train. The dimension of a kernel as being the amount of information that is encoded into the kernel. In terms of the railroad metaphor, the kernel dimension could be described as being the number of tracks that the conductor must move for a given train. We define the value of the dimension as being between 0.0 and 1.0, with 1.0 being the exact representation of the visual stimulus and 0.0 being nonexistence.

**Example**

A park bench, a library desk, and a car seat are all three distinct objects. Despite the differences in appearances and the differences in the contexts that each are encountered in, all three objects are identified as being chairs. The same learned properties, rules, and restrictions of a chair is carried over to these distinct objects in different contexts. Though they were all identified as being chairs, they all have their own nuances. For example, in a car, it is customary to look for a seat belt after sitting down. This is
something that is not shared with the other 2 objects, despite the fact that they were all identified as being used for sitting on. This nuance, is therefore not part of the kernel that comprises a seat. The commonalities between the three objects that are carried over across all three in different contexts make up the kernel of a seat. The kernel - the population that represents the categorization and corresponding learned rules of seats - is readily able to be used in the different settings and with different objects that share the common features of a seat, without having to relearn what a what a seat is in every new setting. And the amount of detail that is necessary for an object to be recognized as belonging to a specific category is the dimensional value. If an individual learned what each of these objects were individually without being able to spot commonality between them, then the kernel that represents the category of each specific object would have a higher dimensional value then a kernel that represents a category that could connect all three to one another.

**Dendritic Spine Density**

Though the specific role of dendritic spines are not known, they have been speculated to play a functional role in input specific learning rules for neural circuits (Yuste, 2010). There are five main reasons for this speculation. The first is that the dendritic spines receive almost all excitatory inputs in all regions in the brain, and though pyramidal cells do not have any excitatory input, they do have dendritic spines, which implies that the spines have other functions. The second is that the physical properties of the spines match their surrounding synapses, which implies that they are created for a functional purpose, which has led to speculation that it the structure is such that the spines can
adapt over time by changing synapses. The third reason is that the durability of spines leads it to playing a role in long term memories. The fourth reason is that the biochemical properties of spines may mean that they play a role in plasticity. And the final reason is that spines may have an electrical function and that a spine can experience a specific voltage independent of their parent dendrites (Yuste, 2010; Spruston, 2008; Elston et al., 2005; Prather et al., 1998). The pyramidal cells of the PFC have been reported to contain the largest amount of dendritic spine density (density) in the entire brain (Prather et al., 1998).

The abilities that these circuits have to solve organization, categorization, classification, or optimization problems, or to generate multistable dynamical states and temporal patterning of the activity, or to enable the association of sets of inputs, or to implement Boolean logic, could be based on the fact that they use spines. In this view, spines would not be an accidental design feature, but the functional components of these circuits, and precisely the ones that endow them with the properties that support their computational powers. Yuste (2010, p. 210).

Due to the amount of speculation regarding the role that spine play in cognitive functioning, in our proposed theory, we use spines as a contributing source that determines the kernel and the density of the spine to determine the dimension. To examine the functional role that density has on the dimension of a kernel, we examine two psychiatric disorders, schizophrenia and autism, both of which have been found to have alterations in the spine density of PFC pyramidal cells. These disorders can shed light on the role that spine density plays on cognition because these disorders are characterized
for behavioral defects that arise from cognitive functioning rather than from anatomical alterations, meaning that the differences in density can be mapped to the cognitive deficiencies of these disorders.

**Autism**

Anatomically, the PFC of individuals with autism has been found to have a far greater density in the dendritic spines of layer II and layer V pyramidal cells (Hutsler & Zhang 2010; Uhlhaas & Singer 2012). Behaviorally, this disorder is formally characterized by restricted behavior, poor communication, and deficits in social-emotional reciprocity (Smith 2017). All three of these behaviors imply defects in ability to generalize and reuse experience, and thus we hypothesize that this disorder is the partially the result of difficulty in reducing the kernel dimensions.

**Schizophrenia**

Individuals with Schizophrenia (SZ) have also been found to have abnormalities in their PFC. Anatomically, the region appears to be thin, and this factor is most pronounced in the region that is responsible for working memory (Kandel et al. 2012; Lewis 2000) and there is visible reduction in the amount of tissue in the region (Mccarley et al. 1999). Despite the reduction in tissue, the SZ PFC has been found to contain a normal number of neurons. The difference, however, is in the structure of the pyramidal cells (Mirmics et al. 2000; Berman 1986). Among the differences that have been found is that there is a significant decrease in dendritic spine density of the layer III neurons (Glantz & Lewis 2000) and layer IV neurons (Hashimoto et al. 2003). Behaviorally,
SZ is also characterized by disorganized speech, as well as diminished emotional expressions, disorganization, and hallucinations (Abuse & Administration, n.d.). All of these behaviors also imply defects in ability to generalize and reuse experience, but in the opposite manor as autism. We hypothesize that this disorder is partially the result of difficulty in connecting information to make kernels of normal dimensions.

**Proposed Theory**

Given the fact that two different psychiatric disorders that characterized by behavioral defects that can be attributed to trouble in generalizing information, are also known to have abnormalities in the density of certain pyramidal cells’ dendritic spines in their PFC, we propose a hypothesis for how a kernel is formed based on these structural and behavioral abnormalities: that a kernel’s dimension directly correlates with the dendritic spine density of pyramidal cells of the PFC. As previously stated, dendritic spines have long been hypothesized for playing a functional role in cognitive functioning and there are many competing theories for their specific role. Since there is no uniformed consensus of their specific role and since many of the current theories of them range many different and unrelated functional properties of neurons, we do not hypothesize a mechanism. Rather, we aim to build on the existing theories about abstract category formation in the PFC by bridging them to unrelated studies related to psychiatric disorders.
Equation

As stated, we are not proposing the mechanism for how dendritic spines produce categories due to how little consensus there is about the specific functional role that they play in cognition. As such, we show a kernel expressed as a relation to density in mathematical form as the following:

\[ \| \delta \| = \frac{\partial \chi}{\partial \rho} + E \]  (1)

where \( \delta \) represents the kernel, \( \| \delta \| \) represents the dimension, \( \chi \) represents the visual stimulus, \( \rho \) represents the density value of the dendritic spine, and \( E \) represents extraneous factors.

Methods

Since our proposed theory pieces together the structural property of the cell anatomy on a micro-scale to the cognitive abilities on a much broader-scale, there is no straightforward way to test this in a single computational simulation, which is why we use two separate tests: one to test whether the structural component, dendritic spine density, correlates with kernel dimension and another to test whether kernel dimension correlates with cognitive functioning.

Test 1: Kernel Dimension as a Factor of Density

The motif of this test to determine whether density in the connections of cells plays a role in determining a kernel’s dimension. For this purpose, a batch of Convolutions Neural Networks (CNN) was prepared. Each CNN in the batch was given 900 neurons.
each, but each with a different number of hidden layers to manipulate the amount of connectivity among layers. The density property that we measured was the amount of connectivity between the input layer and the first hidden layer in proportion. Each CNN in the batch was trained on the classic MNIST data-set (The MNIST database (n.d.)). Given the fact that each CNN had the same total number of neurons, the most plausible explanation for any variation in the results would be differences in the connectivity of each model. The experiment was inspired by the results that were reported in the book *Deep Learning* (Goodfellow et al., 2016, pgs. 198-200), in which the topic of accuracy correlating with number of layers in a CNN was discussed.

The following table shows the number of connections between input and hidden layers for the 6 models:

| Number of Layers | Input to Hidden Connections | Density (Connections/Total) |
|------------------|-----------------------------|-----------------------------|
| 1                | 900                         | 1.0                         |
| 2                | 450                         | 0.5                         |
| 3                | 300                         | 0.33                        |
| 4                | 225                         | 0.25                        |
| 5                | 180                         | 0.2                         |
| 6                | 150                         | 0.167                       |
Test 2: Cognitive Functioning as a Factor of Kernel Dimension

The motif of this test was to determine whether a kernel’s dimension can produce the behavioral defects that characterize the two psychiatric disorders, autism and SZ. For this purpose, we chose to use emotional intelligence (EQ) - defined by Peter (2010) as being the "cognitive ability to correctly perceive, use, understand, and regulate emotions in the self and others" (Peter, 2010, pg. 1) - as the metric to quantify behavior with. Individuals with both disorders have been found to have lower then average EQ (Mcintosh et al., 2006; Kimmy S. Kee, 2009), which is not surprising given the earlier stated deficits in cognitive functioning. "Loosely, then, one could argue that a variety of individuals with mental health problems lack [EQ]" (Zeidner et al., 2011, p. 9). The most common way to test EQ is with the Mayer-Salovey-Caruso Emotional Intelligence Test. The test involves many different components, including a section on labeling emotions based on facial expression (Brackett & Salovey, 2013). To test EQ as a factor of kernel dimension, we trained a batch of different CNNs on the Cohn-Kanade dataset (Kanade et al., n.d.; Lucey et al., 2010), a series of images that shows different faces of individuals with different facial expressions, which correspond to different emotions. Each CNN was trained on the same data-set, but with one modification. We added a bit of blur with a Gaussian filter, with each model being trained with a different level of blur. Our rational for this was to remove precision in an incremental manner.
Figure 1: Same sample image from each set. © Jeffrey Cohn

Results

Test 1

The results for the experiment are as follows:
### Test 2

The results for the experiment are as follows:

| Density | Accuracy | Time to Train |
|---------|----------|---------------|
| 1.0     | .9717    | 161 seconds   |
| 0.5     | .9731    | 81 seconds    |
| 0.33    | .9724    | 71 seconds    |
| 0.250   | .9701    | 61 seconds    |
| 0.2     | .9690    | 48 seconds    |
| 0.167   | .9657    | 38 seconds    |

![Plot of the Time taken to train each CNN](image_url)
The above shows the results of the same CNN architecture trained on the Cohn-Kanade with different levels of Gaussian blur (Sigma) applied.
Conclusion

In the first test, we found that all of the models converged to the nearly the same accuracy. Although we expected to use model accuracy for our analysis, the near uniform convergence meant that the factor of accuracy was not significant enough in the simulation that we designed to draw any conclusions from. An interesting finding, however, was that despite that fact that the each model was able to learn and perform roughly the same, we found that there was a significant difference in the amount of time that it took each model to train. We found that that the greater the density between the input and first hidden layer, the longer it took for the model to learn. This could imply that as the density value of the CNN increases, the ability to learn to generalize information decreases. By our theory, each model performed differently because each processed information into kernels of different dimensions, which demonstrates what we intended to show - density plays a role in kernel dimension. The results of the second experiment are as expected. They show that the models had difficulty with learning to generalize subtle facial expressions when trained with either too much or too little precision. This implies that either functioning with very high dimension kernels or functioning with very low dimension kernels has adverse effects on cognitive functioning that result in behavioral deficits similar to those that categorize autism and SZ.

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Appendix

The code that was used to run the simulations can be found at www.github.com/prashantcraju/Kernel-Abstractions.

References

Abuse, S., & Administration, M. H. S. (n.d.). Table 3.22, dsm-iv to dsm-5 schizophrenia comparison - impact of the dsm-iv to dsm-5 changes on the national survey on drug use and health - ncbi bookshelf. https://www.ncbi.nlm.nih.gov/books/NBK519704/table/ch3.t22/. U.S. National Library of Medicine.

Antzoulatos, E. G., & Miller, E. K. (2011). Differences between neural activity in prefrontal cortex and striatum during learning of novel abstract categories. Neuron, 71(2), 243-9. doi: 10.1016/j.neuron.2011.05.040

Antzoulatos, E. G., & Miller, E. K. (2014). Increases in functional connectivity between prefrontal cortex and striatum during category learning. Neuron, 83(1), 216-225. doi:
Berman, K. F. (1986). Physiologic dysfunction of dorsolateral prefrontal cortex in schizophrenia. Archives of General Psychiatry, 43(2), 126. doi: 10.1001/archpsyc.1986.01800020032005

Brackett, M., & Salovey, P. (2013). Emotional intelligence. Oxford Bibliographies Online Datasets. doi: 10.1093/obo/9780199828340-0047

Cromer, J. A., Roy, J. E., & Miller, E. K. (2010). Representation of multiple, independent categories in the primate prefrontal cortex. Neuron. doi: 10.1016/j.neuron.2010.05.005

Elston, G. N., Elston, A., Casagrande, V., & Kaas, J. H. (2005). Pyramidal neurons of granular prefrontal cortex of the galago: Complexity in evolution of the psychic cell in primates. The Anatomical Record Part A: Discoveries in Molecular, Cellular, and Evolutionary Biology, 285A(1), 610–618. doi: 10.1002/ar.a.20198

Fuster, J. M. (1999). Memory in the cerebral cortex: an empirical approach to neural networks in the human and nonhuman primate. MIT.

Genovesio, A., Brasted, P. J., Mitz, A. R., & Wise, S. P. (2005). Prefrontal cortex activity related to abstract response strategies. Neuron, 47(2), 307–320. doi: 10.1016/j.neuron.2005.06.006

Glantz, L. A., & Lewis, D. A. (2000). Decreased dendritic spine density on prefrontal cortical pyramidal neurons in schizophrenia. Archives of General Psychiatry, 57(1), 65. doi: 10.1001/archpsyc.57.1.65
Goodfellow, I., Bengio, Y., & Courville, A. (2016). Deep learning. MIT Press. (http://www.deeplearningbook.org)

Hashimoto, T., Volk, D. W., Eggan, S. M., Mirnics, K., Pierri, J. N., Sun, Z., . . . Lewis, D. A. (2003). Gene expression deficits in a subclass of gaba neurons in the prefrontal cortex of subjects with schizophrenia. The Journal of Neuroscience, 23(15), 6315-26. doi: 10.1523/jneurosci.23-15-06315.2003

Hutsler, J. J., & Zhang, H. (2010). Increased dendritic spine densities on cortical projection neurons in autism spectrum disorders. Brain Research, 1309, 83-94. doi: 10.1016/j.brainres.2009.09.120

Kanade, T., Cohn, J., & Tian, Y. (n.d.). Comprehensive database for facial expression analysis. Proceedings Fourth IEEE International Conference on Automatic Face and Gesture Recognition (Cat. No. PR00580). doi: 10.1109/afgr.2000.840611

Kandel, E. R., Schwartz, J. H., & Jessell, T. M. (2012). Principles of neural science, fifth edition. McGraw-Hill.

Kimmy S. Kee, e. a., Kimmy S. (2009). Emotional intelligence in schizophrenia. Schizophrenia Research, 107(1), 61–68. doi: 10.1016/j.schres.2008.08.016

Lewis, D. A. (2000). Is there a neuropathology of schizophrenia? recent findings converge on altered thalamic-prefrontal cortical connectivity. The Neuroscientist, 6(3), 208–218. doi: 10.1177/107385840000600311

Lucey, P., Cohn, J. F., Kanade, T., Saragih, J., Ambadar, Z., & Matthews, I. (2010). The extended cohn-kanade dataset (ck): A complete dataset for action unit and emotion-
specified expression. 2010 IEEE Computer Society Conference on Computer Vision and Pattern Recognition - Workshops. doi: 10.1109/cvprw.2010.5543262

Mccarley, R. W., Wible, C. G., Frumin, M., Hirayasu, Y., Levitt, J. J., Fischer, I. A., & Shenton, M. E. (1999). Mri anatomy of schizophrenia. Biological Psychiatry, 45(9), 1099–1119. doi: 10.1016/s0006-3223(99)00018-9

Mcintosh, D. N., Reichmann-Decker, A., Winkielman, P., & Wilbarger, J. L. (2006). When the social mirror breaks: deficits in automatic, but not voluntary, mimicry of emotional facial expressions in autism. Developmental Science, 9(3), 295-302. doi: 10.1111/j.1467-7687.2006.00492.x

Miller, E. K., & Cohen, J. D. (2000). The prefrontal cortex and cognitive control. Nature Reviews Neuroscience, 1(1), 59-65. doi: 10.1038/35036228

Mirnics, K., Middleton, F. A., Marquez, A., Lewis, D. A., & Levitt, P. (2000). Molecular characterization of schizophrenia viewed by microarray analysis of gene expression in prefrontal cortex. Neuron, 28(1), 53–67. doi: 10.1016/s0896-6273(00)00085-4

The mnist database. (n.d.). Retrieved from http://yann.lecun.com/exdb/mnist/

Peter, P. C. (2010). Emotional intelligence. Wiley International Encyclopedia of Marketing. doi: 10.1002/9781444316568.wiem04017
Prather, M., Schall, M., & Jacobs, B. (1998). Regional differences in dendritic and spine complexity: A quantitative golgi analysis of human cerebral cortex. Psi Chi Journal of Psychological Research, 3(4), 151–162. doi: 10.24839/1089-4136.jn3.4.151

Smith, I. C. (2017). Dsm-5 and autism spectrum disorder. Encyclopedia of Autism Spectrum Disorders, 1–6. doi: /978-1-4614-6435-8\_102158-1

Spruston, N. (2008). Pyramidal neurons: dendritic structure and synaptic integration. Nature Reviews Neuroscience, 9(3), 206–221. doi: 10.1038/nrn2286

Uhlhaas, P. J., & Singer, W. (2012). Neuronal dynamics and neuropsychiatric disorders: Toward a translational paradigm for dysfunctional large-scale networks. Neuron, 75(6), 963-80. doi: 10.1016/j.neuron.2012.09.004

White, I. M., & Wise, S. P. (1999). Rule-dependent neuronal activity in the pre-frontal cortex. Experimental Brain Research, 126(3), 315–335. doi: 10.1007/s002210050740

Yuste, R. (2010). Dendritic spines. MIT Press.

Zeidner, M., Matthews, G., & Roberts, R. D. (2011). The emotional intelligence, health, and well-being nexus: What have we learned and what have we missed? Applied Psychology: Health and Well-Being, 4(1), 1–30. doi: 10.1111/j.1758-0854.2011.01062.x