Interview with Two Professors

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Introduction

While psychology and neuroscience have a long history, this knowledge is not set in stone, nor is it all-encompassing. New findings are always being presented, sometimes disproving generally accepted principles. The scientific method allows scientists to constantly be testing and retesting hypotheses, and if these hypotheses are wrong, then sometimes the solution is questioning the foundation on which these hypotheses are built upon. Asking questions is the entire foundation of science. The scientific model requires questioning what is known. The importance of asking these questions cannot be overstated, as investigation into these hypotheses can benefit the field and humanity at large. At the university level, professors have the opportunity to choose what they teach, including their own hypotheses. At the Psychology department at the University of Illinois, a variety of perspectives and hypotheses can be observed, coloring the general understanding of the topic of psychology and its concentrations, such as Neuroscience and Clinical Psychology.

Professor Justin Rhodes

Professor Justin Rhodes is a Professor at the Beckman Institute for Advanced Science and Technology and an Affiliate at the Carl R. Woese Institute for Genomic Biology. He earned his bachelor's degree in biology from Stanford and his PhD in zoology from the University of Wisconsin-Madison. Professor Rhodes has a research focus in cognitiveneuroscience and is researching how genes and the environment can affect behavior. His current lab research investigates how exercise can affect neurogenesis (the creation of new neurons) in the hippocampus. Rhodes frequently discusses these concepts in the classes he teaches, which are PSYC/NEUR 302 (Applied Neuroscience) and PSYC/NEUR/PHIL 433 (Evolutionary Neuroscience), which is also cross coded as IB 436.

Prof. Rhodes often collaborates with colleagues and wrote a chapter in the book of his doctoral advisor, Dr. Theodore Garland Jr., on behavior and neurobiology. Rhodes described the importance of splitting duties based on expertise when collaborating on academic writing, writing sections of papers dedicated to concepts he has the most experience in, and leaving space for collaborators with more experience in other topics. Recently, he wrote a paper with Dr. Ki Yun Lee and Dr. Taher Saif about the involvement of astrocytes in the muscle fiber contraction-hippocampal development network, which showed that astrocytes may mediate this relationship. Prof. Rhodes expressed his interest in the unknown roles of glia in the brain, highlighting how microglia are seen to have specific responses to different levels of an organism engaging in physical activity. Cumulatively, the function of microglia is altered by the effect of an organism running. He would like to see an investigation into the role of signaling in the blood in this effect. Rhodes has conducted an abundance of research on

the hippocampus and has some of his own propositions to its functions.

During intense physical activity, it has been observed that the hippocampus, typically associated with learning and memory, becomes activated. The level of activation in the hippocampus appears to have a positive relationship with the level of intensity of the activity, further indicating that these two are related. This phenomenon is a subject of curiosity and debate among neuroscientists, as the current understanding of the hippocampus and intense physical activity seem to be independent processes. There is evidence to show that physical activity could encourage neurogenesis in the hippocampus. But this evidence is not sufficient to explain how the level of intensity seems to correlate with the level of activation in the hippocampus.

The classic interpretation behind this activation is that the hippocampus is acting as a part of the sensory system. This hypothesis assumes that the hippocampus activates in response to speed, because if the animal is moving through space faster, they will need to sample their environment faster to keep their spatial map up to date. The spatial map is the brain's representation of the spatial environment surrounding it, which is used to support functioning in the environment and relating to the space one is in. The mechanism used to explain this activation is that during hippocampal activation, the hippocampus creates waves of activity, with the most salient, or powerful and effective, learning occurring at the peaks and troughs of these waves. This supposes that to increase the rate of learning, the hippocampus would need to increase the frequency of these waves to maximize learning potential.

Prof. Rhodes believes that the hippocampus is serving a different purpose in this intense activity system, acting as a motivator of this system, instead of serving as a reactionary sensory organ. He hypothesizes that the hippocampus acts as an intensity generator, allowing the animal to move at intense speeds by motivating the body to move. It should be noted that this form of motivation is not of the higher order of consciousness, where people choose to move intensely because of their understanding of the health benefits of such activity or because they want to escape something. Instead, Prof. Rhodes and his colleagues believe that the hippocampus is the organ needed to allow the body to move at such intense speeds when necessary. There have been several studies conducted testing this hypothesis, and a wealth of evidence collected to support it.

Recordings taken in the entorhinal cortex reveal that hippocampal activity precedes intense movement. This evidence could not be explained by the popular hypothesis of the hippocampus acting as a reaction to the activity, as this movement could not cause neural activity that precedes it.

The sensory hypothesis also does not explain why this hippocampal activation does not occur when a subject is being moved through an environment at high speeds whenthey are not running. In a study where test mice were placed in transportation carts (analogous to a car for a human) and moved at a rapid rate, the level of activation seen in intense activity is not observed in the hippocampus, despite the need to rapidly update the spatial map still being present. However, this is a potential side effect of human evolution adjusting far slower than human technological development. This lack of activation when moving quickly through technological means could be explained by human evolution not progressing past the stage where the only way to get around quickly was through intense activity such as running. These recordings of neural activity support the hypothesis that Prof. Rhodes believes. Stimulating these areas, instead of simply observing them, could also generate evidence to support this theory.

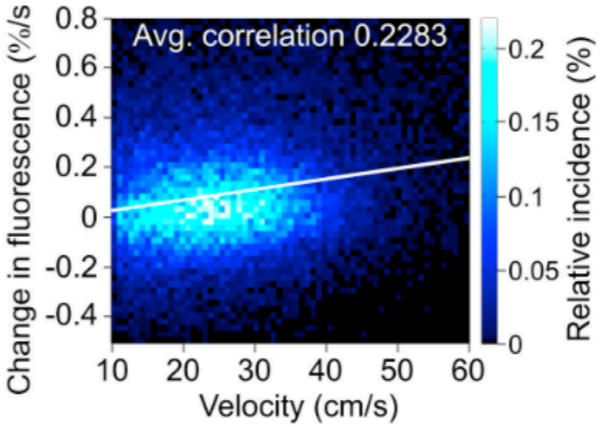

Figure 1. Positive correlation of GCaMP5 fluorescence slope and velocity.

Prof. Rhodes cited a study conducted with Dr. Stefan Remy, a professor at the University of Bonn in Germany. This study was conducted in mice using optogenetics, a revolutionary new technique that allows for the stimulation of specific cells using light. The study used optogenetics to stimulate the hippocampus of mice while on a treadmill. When the hippocampus was stimulated, the mice began to move around and run at an intensity that matched the level of activation in the hippocampus. This evidence supports the idea that the hippocampus supports motivation and ability to move quickly.

This theory has its detractors, however, who call on the current understanding of the motor system and clinical history. The hippocampus is not traditionally connected to the movement system in the field of neuroscience. Patient H.M., a famous patient in the history of psychology, was known for having large portions of his temporal lobe removed from his brain, including his hippocampus. While Patient H.M. is widely known to have lost many of the abilities traditionally associated with the hippocampus, such as the ability to form new memories, he was able to move quickly or engage in intense movement after his operation.

Though, this possibility was not officially studied with HM. The hypothesis believed by Prof. Rhodes of hippocampal involvement provides evidence for why this may have been. This data has also been seen in studies, as the loss of the ability for intense movement when the hippocampus is lesioned has also been observed in experiments where rats have their hippocampus suppressed via anesthetics. The combination of evidence discussed, through studies of recording and stimulation in the hippocampus as well as lesion studies, supports Rhodes's motivator hypothesis. If hippocampal activation precedes intense movement, then it cannot be caused by intense movement, nor can it explain why similar hippocampal activation in response to non-neural-motor forms of rapid movement is not seen. In addition, his idea supports why directly stimulating the hippocampus induces intense motor activity. Finally, while removal of the hippocampus does not have all-encompassing effects on the motor system, it seems to remove the organism's ability to engage in intense movement. The commonly held sensory hypothesis of the relationship between hippocampal activation and intense physical activity does not explain this evidence. Discussions such as these encourage neuroscientists to look deeper into the validity of these hypotheses.

Prof. Rhodes's research and hypotheses of the hippocampus can aid in growing the scientific understanding of the brain region associated with some of the most damaging effects of aging. The hippocampus is the first part of the brain to begin deteriorating as humans age, losing about 1% of it every year after the age of 20. The hippocampus is the brain region most associated with dementia, but it is also one of only two brain regions that is able to create new neurons throughout the human lifetime. Studies have shown that hippocampal exercise may be beneficial for retaining hippocampal mass as humans age as regular strenuous exercise can promote neurogenesis in the hippocampus. Evaluating the current knowledge of the hippocampus, especially in relation to exercise, could grant a revolutionary tool in the treatment of neurodegenerative diseases, as well as the regular struggles of aging and memory impairment.

Professor Thomas Kwapli

Professor Thomas Kwapil is the Director of Clinical Training and an Associate Head of the Psychology Department at the University of Illinois. He describes his role as being responsible for ensuring that the Clinical-Community program runs smoothly, as well as acting as a liaison with the department head. The Clinical-Community program comes with extra responsibilities, as it requires accreditation, which requires reporting to outside agencies. He received his bachelor's degree in psychology from Louisiana State University, and his M.S. in psychology and PhD in clinical psychology from the University of Wisconsin-Madison. Prof. Kwapil is the current supervising professor for PSYC 238 (Psychopathology and Problems in Living) and PSYC 239 (Community Psych).

This is a role that rotates among different professors in the department. Instead of being the instructor for the course, the supervising professor supports and supervises the graduate students who teach the course. Professor Kwapil also regularly teaches a PSYC 496 (Adv Current Topics in Psych) course on schizophrenia-spectrum disorders, which is his area of interest.

Schizophrenia-spectrum disorders include schizophrenia and other psychotic disorders, including schizophreniform disorder and brief psychotic disorder, as well as schizotypal personality disorder. It is seen through abnormalities in "positive" symptoms (including delusions and hallucinations), disorganized symptoms (such as disorganized thinking, speech, and behavior), and "negative" symptoms (such as avolition and diminished emotional expression). An estimated 3-4% of the population suffers from schizophrenia spectrum disorders. Many psychologists, especially those whose perceptions are colored by the categorical nature of the Diagnostic and Statistical Manual of Mental Disorders (DSM), would see these as categorical disorders, following a yes/no binary. Prof. Kwapil disagrees with this notion.

Figure 2. SEQ Figure * ARABIC 2. Schizophrenia Spectrum Disorders. Giovanni, V. (2015).

It is widely believed that other disorders, such as depression and anxiety, exist on a spectrum of intensity and subclinical presentations, yet this line of thinking is not often extended to schizophrenia-spectrum disorders. Many people report experiencing anxiety or depression. With some, a more clinical expression is seen. With others, these symptoms are less intense, do not last as long, and/or cause less impairment. All are experiencing these symptoms; some are simply experiencing them on a subclinical level. The same can be true for schizophrenia-spectrum psychopathology. Even when it comes to clinical schizophrenia-spectrum disorder presentations, categorical measures fall short to dimensional measures. Those with schizophrenia may experience psychotic episodes, which remit and return, similar to depressive episodes seen in Major Depressive Disorder. An all-or-none categorical form of thinking, in which a subject is either in or out of an episode, may be easier to conceptualize, but is not entirely accurate to describe this phenomenon. Often when people remit from episodes, their expression of symptoms doesn't go from disordered to regularly functioning, but rather a less intense symptomology or degree of impairment. Where the categorical model fails to encapsulate these observations, Prof. Kwapil has a more fitting explanation.

Prof. Kwapil and his colleagues support a dimensional model, in which schizophrenia-spectrum disorders and subclinical presentations of schizophrenia exist on a continuum, called schizotypy. Prof. Kwapil and his colleagues are pursuing an effort to reconceptualize schizophrenia and related disorders as not simply extreme and rare disorders, but also milder presentations. Often conceptualized as extreme manifestations in clinical cases, milder forms of positive, disorganized, and negative symptoms can also be seen. These subclinical symptoms can develop and change in a variety of ways, if at all. Some of these milder symptoms go on to develop into a clinical, disordered presentation. Some continue to have these symptoms, but they do not get worse or interfere with functioning. Others will have these symptoms come and go, sometimes never appearing again. A key difference between milder symptoms and clinical symptoms when it comes to, for example, delusional thinking, is in conviction. Someone with a clinical expression of delusional thinking may be convinced that someone is stealing their thoughts or threatening them, whereas someone with a subclinical expression of delusional thinking may wonder or suspect if it may be possible but are unsure.

Prof. Kwapil's method for studying the dimensional model involves a complex process that he has implemented into his research. First, development of a conceptual model for understanding the dimensions of schizotypy is needed. Models have been developed to measure the dimensions of positive, disorganized, and negative schizotypy in order to identify people who exist on this scale and how their symptoms should present. When observed in subclinical expressions, which do not cause impairment, those being studied are not seen as patients, but rather personality traits. People can be elevated on one, two, or all three dimensions. For example, someone who may be high in the positive dimension, experiencing magical beliefs or strange perceptual experiences, may be low in negative and disorganized dimensions, such that they would not be experiencing the symptoms such as flattened affect, anhedonia, or difficulty organizing and executing thoughts and emotions. Participants for a study of subclinical presentations of schizotypy are typically found through extensive interview studies or questionnaire studies focusing on schizotypal traits, personality, and emotion. One of the testing methods favored by Prof. Kwapil is Experience Sampling Methodology, or ESM. ESM is a measurement that uses a system of daily surveys sent out multiple times each day that assesses the dynamic system of emotional and psychological phenomena throughout the day. This is a more effective measurement system for seeing a larger picture of a participant's life by measuring them over a larger period of time than a single lab interview session, making it a fitting choice for studying subclinical presentations of psychological disorders.

Schizotypy, like many dimensional models of clinical disorders, offers a useful conceptual model of schizophrenia spectrum disorders that has advantages over traditional categorical models. Human behavior and emotions have a complexity and nuance that cannot be entirely captured through a yes/no binary. Similar to anxiety and depression, which are generally accepted to be experienced both clinically and subclinically, symptoms of schizophrenia spectrum disorder can be observed on a lower impairment level among those who do not fit the criteria for schizophrenia spectrum disorders. Prof. Kwapil has been studying schizotypy for years and has developed an effective model of study of the dimensions of schizotypy. With models such as these, the scientific community can move on to a better understanding of the truly dimensional model of psychological disorders and their subclinical presentations.

Prof. Kwapil's proposed model of schizotypy can advance the understanding of schizotypy, allowing for an improved early detection system and potentially negating some of the stigma associated with these disorders. An estimated 10% of people experience subclinical expressions of schizotypy, which is a large portion of the population and merits investigating the presentation and mechanism behind these symptoms. Identifying these individuals could be instrumental in instituting early intervention services for those who will eventually develop a clinical presentation of schizotypy. However, those with schizophrenia spectrum disorders have a heavy stigma against them among the general public, and it is important to acknowledge this during research. When trying to identify potential prodromal symptoms, which can help with preventing these symptoms from developing to a clinical level, those identified with subclinical symptoms could put at risk of losing social support systems, losing their jobs, and potentially being prevented from being covered by insurance because of the association with these disorders. Being identified as being at risk of developing heavily stigmatized disorders such as schizophrenia or its related disorders carries a much difference social weight than those identified as being at risk for the development of less stigmatized illnesses, such as breast cancer. As those with schizophrenia are often mischaracterized as dangerous or violent. However, as the understanding of schizotypy grows through research, it is possible for education about schizotypy to be spread outside of the scientific community, placing logic and understanding in place of fear.

About the Author

Brianna Mae is a Junior at the University of Illinois majoring in Clinical/Community Psychology. She became involved in Brain Matters to gain more experience researching and writing about the current research in Neuroscience. When she is not writing for Brain Matters, she is also involved in Dr. Kwapil's Project on Life Experiences Lab, and is the Treasurer for the Psychology Research and Community Club (PRACC). Brianna Mae is hoping to pursue a PhD in Clinical Neuropsychology and conduct research about the neurological basis behind different clinical disorders.