The New York University Integrative Psychology Review

Fall 2021 || Volume I || No. 1
New York University

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Acknowledgements

The New York University Integrative Psychology Review would like to thank its administrative director, Dr. Andrew Hilford, and its faculty advisor, Dr. Lawrence Ian Reed for their contributions.

Additionally, NYU IPR would like to thank the members of its editorial team for their work and dedication.

Finally, the journal would like to thank New York University for allowing NYU IPR to provide a platform for undergraduate students from various disciplines to contribute to the interdisciplinary field of psychology.

Statement of Purpose

The New York University Integrative Psychology Review is a publication seeking to help students across all disciplines work together under the umbrella of psychology by providing a peer-reviewed platform that fosters a collaborative learning environment. Psychology is an incredibly interdisciplinary field, and we believe that it deserves a forum that exemplifies this diversity.

NYU IPR aims to empower undergraduate students of all disciplines and fields to engage in both empirical research and literature review and facilitate discussion and inquiry within the domain of psychology. We believe that creating a strong foundational framework of research literacy and collaboration across multiple disciplines and fields of study at an undergraduate level is critical to foster a truly comprehensive understanding of psychology and its applications for future generations of researchers and academics.

Preface

Welcome to the NYU IPR!

The peer-reviewed journal is a foundational component of the advancement of knowledge. It serves to maintain the strength and validity of empirical pursuits. A measure of quality is established by requiring potential published works to obtain the approval of an editorial board in order to appear printed for all to see and benefit from. The analysis and examination of candidate articles by an objective group of learned and critical minds provides a degree of confidence in the value of content being put forward. Because of this, there is worth and prestige in the articles presented in the NYU IPR. It is these articles that can be considered among those most worthy of a reader's time and intellectual commitment.

Without publication engaging concepts, fresh ideas, novel findings – from any domain – languish if not given the opportunity to be read. Discoveries, epiphanies and unexpected reinterpretations only reach their potential when new audiences are tapped and new minds found. In providing a vehicle for communicating knowledge, this journal aids in the expansion of thought and understanding. Absent of a platform, new and exciting information can wither and fade and vital and dynamic minds can go unfulfilled. The transmission of fresh and vibrant content is a key aspect of advancement. With journal in hand, or on screen, the reader is offered the opportunity to experience novel concepts, challenge old ideas, or build upon previously consolidated learning.

The NYU IPR journal extends its intellectual purview to any and all corners of academic pursuits related to the study of psychology. By design, this not only provides diversity of topics, but allows for unexpected exposure to varied and challenging perspectives.

Ultimately, we hope that the NYU IPR journal provides you with engaging and interesting readings, but also gives rise to new thoughts, ideas and opinions.

Enjoy!

Andy Hilford
Associate Director of Graduate and Undergraduate Studies, Psychology
New York University

Note from the Editors-In-Chief

Dear readers,

We are proud to present the first volume of the New York University Integrative Psychology Review. This edition is the result of months of work from authors, our staff editors and faculty mentors throughout the past year.

With the uncertainty of COVID-19 and challenges it brought to our local and global communities, we are grateful and humbled to have the opportunity to publish this review. We are delighted to present nine budding researchers who, during this time, have worked tirelessly to produce the featured empirical research and literature reviews.

We hope that NYU IPR inspires undergraduate researchers to continue pursuing their passions for psychological investigation, and we look forward to producing future editions in the years to come.

Sincerely,

Sanjana Dixit
Founder and Co-Editor in Chief

Samantha J. Gordon
Co-Editor in Chief

Validating Instruments to Screen for Psychopathy In a Non-Institutionalized Population

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Author contributions: PW designed the study and analyzed the data. TB, IA & JR drafted the manuscript. TB, IA, JR, CEB, DF, SS, HY, MA, JZ, SG, SM, JP, PL, AG, SH, AE, HY, VT, DT, LC, AF, NV, JF, SD, AK, BB, HJTS, GI, APL recorded the data. All authors reviewed and revised the manuscript and approved the final version.

Acknowledgements

We would like to acknowledge New York University, the Department of Psychology, and Andy Hilford for supporting our research. We would also like to thank the senior administration of the College of Arts and Sciences for sponsoring our research through the Dean's Undergraduate Research Fund.

Abstract

We wanted to validate commonly used instruments to measure psychopathic tendencies in a college student population. To do so, we administered both the "Dark Triad Dirty Dozen" and the "Levenson Self-Report Psychopathy scale" to a high-powered sample of college students. Participants also performed a social discounting task to measure generosity. We correlated all of these measures and found that both instruments correlate well and negatively with generosity. We take these findings to indicate that both instruments are valid measures of psychopathic tendencies in non-institutionalized populations.

Keywords: Levenson Self-Report Psychopathy Scale (LSRP), Dark Triad Dirty Dozen, Psychopathy, Narcissism, Social Discounting, Generosity

Introduction

Psychopathy is characterized by shallow affect, amoral conduct, poor impulse control, and lack of empathy (Psederska et al., 2021). Behaviors associated with this condition include manipulativeness, disregard for moral principles, and pathological lying (Sperry, 2016). The present study aims to validate commonly used self-report screening tools, specifically the Levenson Self-Report Psychopathy Scale (LSRP, Levenson, Kiehl, & Fitzpatrick, 1995) and the Dark Triad Dirty Dozen (DTDD, Jonason & Webster, 2010).

Despite significant advances in what is known about psychopathy, considerable gaps in the literature remain. Up to this point, research has focused on psychopathy in prison populations rather than the general population. While there is

participants in an interleaved fashion, with a presentation order that was randomized for each participant to avoid order effects.

Dark Triad Dirty Dozen Test

The Dark Triad Dirty Dozen Test (DTDD) is a 12-item personality inventory that consists of three facets: Machiavellianism, narcissism and psychopathy (Jonason & Webster, 2010). Participants rate each item on a 7-point Likert scale, with 1 implying strong disagreement and 7 strong agreement. Despite the brevity of the test, the DTDD is considered to be reliable and valid (Jonason & Webster, 2010).

Levenson Self-Report Psychopathy Scale

The Levenson Self-Report Psychopathy Scale (LSRP) is a 26-item personality inventory that measures psychopathy on a 5-point Likert scale (Levenson, Kiehl, & Fitzpatrick, 1995). The LSRP supposedly consists of two underlying facets - primary and secondary psychopathy, with primary psychopathy referring to lifestyle choices and secondary psychopathy to emotional responses (Vaughn et al., 2009). The LSRP is also considered to be reliable and valid (Bowling, 2005; Brinkley et al.; Falkenbach et al., 2007; Fritz, & Lim, 2018; Gummelt, Anestis, & Carbonell, 2012; Henrich, Heine, & Norenzayan, 2010).

Generosity

Participants were asked to give responses in a social decision-making task. In this task, participants choose whether they prefer receiving or losing a certain amount of hypothetical money or someone else receiving/losing a more significant monetary amount, sensu Jones & Rachlin (2006). There are six social distances, six monetary amounts (from $20 to $105) and receiving/losing the money, fully crossed - for a total of 72 unique trials. We classify responses that make you gain money or someone else lose money as selfish responses and where someone else gains money and you lose money as selfless. We then compute a generosity index. For instance, if a participant picks the selfish choice 72 times, we would classify them as 0% generous. If they choose the selfless choice 72 times, we would classify them as 100% generous, with everything in between. Notably, the presentation location of which button represents the selfish choice and which button represents the selfless choice was randomized between left and right for each participant and trial to prevent the impact of potential response biases.

Analysis

We analyzed the data recorded using the methods above by computing Pearson correlation coefficients between all of these measures as well as between generosity and the facets of the DDTD. Data were analyzed using MATLAB 2019b (Mathworks, Natick, MA). As we assess several such correlations, and to prevent alpha-inflation, we adopt a conservative alpha-level of 0.005 (Benjamin et al., 2018).

Results

What is the relationship between responses on the Dirty Dozen (DTDD) and Levenson Self-Report Psychopathy Scale (LSRP)?

In order to assess whether Psychopathy is a valid construct that can be reliably assessed with screening instruments, we correlated the scores on the DTDD and the LSRP. We found them to be highly positively and significantly correlated to each other, $r = .70$, $p = 5.1381e\text{-}97$, see figure 1.

What is the relationship between DTDD and generosity?

In order to assess whether DTDD scores are related to behaviors, we correlated them with generosity scores in the task described above. We found that DTDD scores correlate significantly negatively with generosity, r = = -0.25, $p = 1.2015e\text{-}10$, see figure 2.

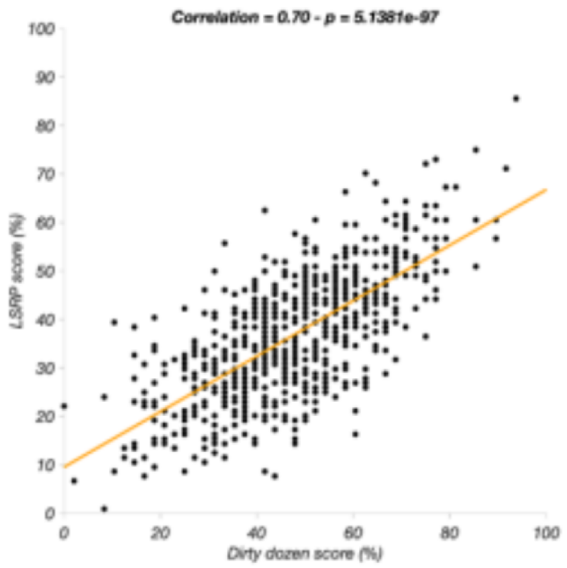

Figure 1. DTDD vs. LSRP scores. The x-axis represents the DTDD score of participants as a percentage. The y-axis represents the LSRP score of participants as a percentage. Each black dot represents the scores of an individual participant. The orange line is the line of best fit.

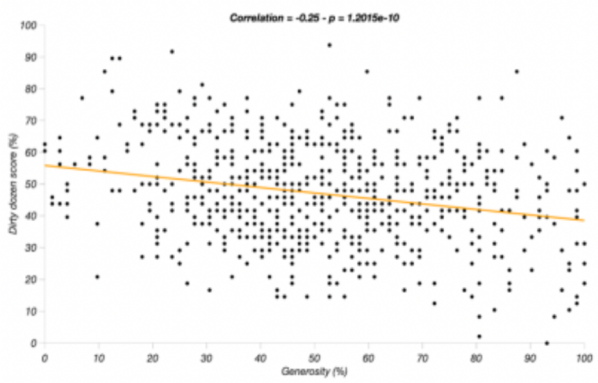

Figure 2. Generosity vs. DTDD scores. The x-axis represents the generosity score of participants as a percentage. The y-axis represents the DTDD score of participants as a percentage. Each black dot represents the scores of an individual participant. The orange line is the line of best fit.

What is the relationship between LSRP vs. Generosity?

In order to assess whether LSRP scores are related to behaviors, we correlated them with generosity scores in the task described above. We found that LSRP scores correlate significantly negatively with generosity, r *= -0.25, p = 8.6838e-11*, see figure 3.

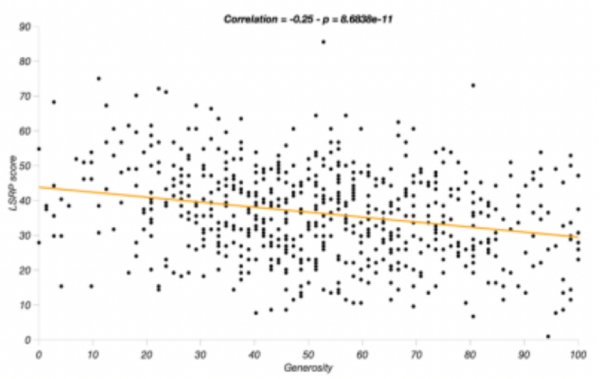

Figure 3. Generosity vs. LSRP scores. The x-axis represents the generosity score of participants as a percentage. The y-axis represents the LSRP score of participants as a percentage. Each black dot represents the scores of an individual participant. The orange line is the line of best fit.

What is the relationship between the three facets of the DD (Machiavellianism, Psychopathy and Narcissism) and Generosity?

Conceptually, we would expect psychopathy and narcissism to correlate with selfishness, but not necessarily Machiavellianism. However, when correlated with generosity, all three facets of the DTDD correlated negatively with generosity, *r = -0.21, p = 3.3434e-08; r = -0.17, p = 1.3818e-05; r = -0.19, p = 6.9252e-07, respectively*, see figure 4.

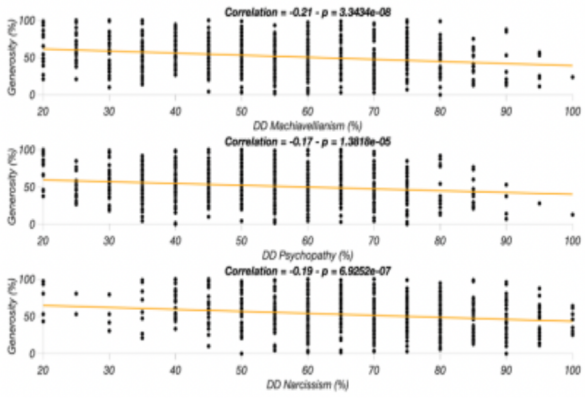

Figure 4. Facets of the DTDD vs generosity. The panels represent the facets of the DTDD, from top to bottom: Machiavellianism, Psychopathy and Narcissism. The x-axis represents the score of that subfacet. The y-axis represents generosity as a percentage. Each black dot represents the scores of an individual participant. The orange lines are the line of best fit.

What is the intercorrelation between all measures in the study?

To assess the intercorrelation between all measures in our study, we present the correlation matrix in Table 1

	LSRP	DTDD Machiavelli Psychopathy	Narcissism
LRSP	1	0.696*** 0.617*** 0.641***	0.359***
DTDD		1 0.846*** 0.741***	0.733***
Machiavelli		1 0.491***	0.452***
Psychopathy		1	0.250***
Narcissism			1

Table 1: Pearson correlation coefficients between all measures in our study. "Machiavelli", "Psychopathy" and "Narcissism" refer to the facets of the DTDD, whereas "LSRP" and "DTDD" refer to the scores of the whole instrument. ***: significant at alpha < 0.005.

Discussion

We found a strong correlation between DTDD scores and LSRP scores. We found negative correlations between both DTDD as well as LSRP scores and generosity We also found negative correlations between all facets of the DTDD and generosity. We found that the facets Machiavellianism and Psychopathy correlate strongly with LSRP scores while narcissism does only weakly. The facets of Psychopathy and Narcissism correlate moderately with the facet of Machiavellianism, but Narcissism correlates only weakly with the facet of Psychopathy. All of these findings were significant.

We take this pattern of results to mean that both the Levenson Self-Report Psychopathy scale and the Dirty Dozen Dark Triad are valid instruments to measure psychopathy in a college population. Interestingly, all facets of the DTDD correlated significantly negatively - at about the same magnitude with generosity, suggesting that - despite the theoretical differences between Machiavellianism, psychopathy and narcissism, the overall construct of psychopathy is captured well by all facets of the DTDD.

However, looking closer at the intercorrelations between the facets of the DTDD and the correlations between these facets and the overall LSRP scores reveals a very curious insight: Machiavellianism seems to be as strongly correlated with psychopathy as measured by the LSRP as the DTDD facet "Psychopathy", whereas Narcissism (as measured by the DTDD) is a clearly distinct construct, even if it is equally negatively correlated with generosity.

This suggests - as is also evinced by the low correlation between psychopathy scores and generosity - that psychopathic tendencies are only loosely related to generosity. It is likely that generosity is determined by many factors, psychopathic tendencies accounting only for a rather small portion of the variance - between 4 and 5 percent.

We believe that this is a realistic proposition. If Psychology is to be a mature science, we need to get beyond the notion that we can account for complex macroscopic behaviors like generosity by single factors. It is more plausible that these complex traits are determined by a large number of distinct factors, like Psychopathy, Narcissism, Affluence, Religion, Ideology, Mood, Philosophical outlook, and many others - each of which likely only accounts for a small single digit percentage of the variance in the trait. Thus, it is imperative that studies are powered well enough to be able to detect these minute effects.

References

Benjamin, D.J., Berger, J.O., Johannesson, M. *et al.* Redefine statistical significance. *Nat Hum Behav* 2, 6–10 (2018).

Bowling, A. (2005). Mode of questionnaire administration can have serious effects on data quality. *Journal of Public Health, 27 (3)*, 287.

Brinkley, C. A., Schmitt, W. A., Smith, S. S., & Newman, J. P. (2000). Construct validation of a self-report psychopathy scale: does Levenson's self-report psychopathy scale measure the same constructs as Hare's psychopathy checklist-revised? *Personality and Individual Differences, 31 (7)*, 1025.

Driessen, J. M. A., Brazil, I. A., Lorenzo, E. D., Herwaarden, A. E., Olthaar, A. J., Potamianou, H., & Glennon, J. C. (2021). Psychopathic traits influence threat avoidance in a community sample independent of testosterone. *Personality Disorders: Theory, Research, and Treatment, 12*(5), 428–436.

Falkenbach, D., Poythress, N., Falki, M., & Manchak, S. (2007). Reliability and Validity of Two Self-Report Measures of Psychopathy. *Assessment, 14 (4)*, 344–345.

Forth, A. E., Brown, S. L., Hart, S. D., & Hare, R. D. (1996). The assessment of psychopathy in male and female noncriminals: Reliability and validity. *Personality and Individual Differences, 20*(5), 531–543.

Fritz, K. N., & Lim, N. K. (2018). Selection Bias. *The SAGE Encyclopedia of Educational Research, Measurement, and Evaluation*, 1490–1491.

Gummelt, H. D., Anestis, J. C., & Carbonell, J. L. (2012). Examining the Levenson Self Report Psychopathy Scale using a Graded Response Model. *Personality and Individual Differences, 53 (8)*, 1002–1006.

Henrich, J., Heine, S. J. & Norenzayan, A. (2010). *The weirdest people in the world? Behavioral and Brain Sciences, 33 (2–3)*, 61-135.

Jonason, P. K., & Webster, G. D. (2010). The dirty dozen: A concise measure of the dark triad. *Psychological Assessment, 22(2)*, 420–432.

Jones, B., & Rachlin, H. (2006). Social discounting. *Psychological Science*, *17*(4), 283-286.

Park, S., Kahnt, T., Dogan, A. *et al.* A neural link between generosity and happiness. *Nat Commun* 8, 15964 (2017).

Psederska E, Thomson ND, Bozgunov K, Nedelchev D, Vasilev G and Vassileva J (2021) Effects of Psychopathy on Neurocognitive Domains of Impulsivity in Abstinent Opiate and Stimulant Users. *Front. Psychiatry* 12:660810.

Levenson, M. R., Kiehl, K. A., & Fitzpatrick, C. M. (1995). Assessing Psychopathic Attributes in a Noninstitutionalized Population. *Journal of Personality and Social Psychology. 68 (1)*, 151–152.

Lilienfeld, S. O., Gershon, J., Duke, M., Marino, L., & de Waal, F. B. M. (1999). A preliminary investigation of the construct of psychopathic personality (psychopathy) in chimpanzees (Pan troglodytes). *Journal of Comparative Psychology, 113*(4), 365–375.

Lynam, D. R. (2010). Child and adolescent psychopathy and personality. In R. T. Salekin & D. R. Lynam (Eds.), *Handbook of Child and Adolescent Psychopathy* (pp. 179–201). The Guilford Press.

Sperry, L. (2016). Handbook of Diagnosis and Treatment of DSM-5 Personality Disorders: Assessment, Case Conceptualization, and Treatment (3rd ed.). Routledge.

Vaughn, M. G., Edens, J. F., Howard, M. O., & Smith, S. T. (2009). An Investigation of Primary and Secondary Psychopathy in a Statewide Sample of Incarcerated Youth. *Youth Violence and Juvenile Injustice. 7 (3)*, 173.

Wallisch, P. (2015). Brighter than the sun: Powerscape visualizations illustrate power needs in neuroscience and psychology. arXiv preprint arXiv:1512.09368.

White, B. A. (2014). Who cares when nobody is watching? Psychopathic traits and empathy in prosocial behaviors. *Personality and Individual Differences, 56*, 116–121.

Coronavirus Cognition Paper

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Introduction

The emergence of SARS-Cov-2 is a cognitive problem because it interrupts normal processing on multiple levels of cognition. Even though there have been global pandemics in the past, the SARS-Cov-2 pandemic is on a much greater scale globally compared to that of the 2009 H1N1 outbreak. The protocols to combat the pandemic include, but are not limited to, self-isolation, social distancing, use of surgical masks when out in public, and two-week periods of quarantine for individuals who may have been exposed to the virus. The daily routine for people who live in large cities has changed greatly due to the viral outbreak. From working at home to not being able to visit friends and family, life has not been easy for many individuals during this time. The SARS-Cov-2 pandemic poses problems for cognition because it has forced people into unique situations. For different communities and individuals, there have been noticeable changes in behaviors and, as a result, this impacts neuropsychological processes.

How is this a cognitive challenge for communities?

The article, "What Does Covid-19 Do to Your Brain?" by Megan Molten describes some relatively recent findings on the neural effects of the virus. One patient who contracted SARS-Cov-2 was tested by doctors at Henry Ford Hospital in Detroit. She exhibited swelling in the brain and the images were captured by CT and MRI scans. In addition to the swelling, the doctors found dead and dying neurons and diagnosed the woman with acute necrotizing hemorrhagic encephalopathy. Even though scientists are still trying to determine whether such effects are linked to SARS-Cov-2, this type of brain damage accompanies viral infections because inflammation induces the production of cytokines. Other neurological symptoms found in some SARS-Cov-2 patients include the inability to smell or taste, strokes, and prolonged seizures (Molten 2020). The extent of neurological effects of SARS-Cov-2 is not known, but according to Stanley Perlman, a microbiologist, it is very possible for the virus to cause severe harm to the brain. On a test done with mice, a small amount of SARS-Cov-2 that was put into their noses went straight to their brains and damaged their nerves (Molten 2020). While the effects of the virus discussed in this paper have more to do with physical impacts, the pandemic has definitely altered cognitive processes among those who have not been directly exposed to SARS-Cov-2 itself.

In accordance with typical perception, all senses are critical. Perception is when stimuli from the environment are processed by organs such as the eyes and ears. Certain information is prioritized over others and creates each individual's own model of the world. Senses make it possible for information to enter the neurological system (Gazzaniga, Ivry, Mangun 2014). Although individuals still need to go outside to do essential tasks like shopping for groceries or work, their model of the world is altered. For many communities, schools and small businesses have been closed indefinitely. Perception is extremely useful, but the model of the world that individuals conjure in their brains does not accurately represent reality. Because models are already imperfect and prone to relevancy biases, being in quarantine greatly impacts one's perception because the state of the world has changed. With more reliance on technology and online contact, the model of the world is limited to what people see on their screens and within their own homes. When people

can only look at their screens to know what is going on outside, it is a matter of visual perception. On electronics, the 3D world is displayed in 2D and that is how the world is projected on the retina. Through different cues like depth and prior knowledge, the brain constructs a specific model for each person.

The different levels of processing that are involved in attention include the stimulus environment, the information that individuals are consciously aware of, what is remembered, and what is acted on. Attention is the cognitive process by which information from one's surroundings is filtered and processed by relevance. It is an important function because not everything is relevant. When people are able to focus on a certain aspect out of many, they are able to form a deeper understanding (Wallisch 2020). By dividing attention, or inaccurately coined as 'multitasking,' one's capability to execute tasks worsens. Within communities where the viral outbreak is more intense, people might be focusing on the latest updates about the pandemic instead of their jobs or other priorities. If more people are prioritizing news about SARS-Cov-2 over other aspects of their life, then it will be hard to focus on anything else. Based on the logic of selective attention, people are theoretically able to attend to certain things while ignoring others (Gazzinga et. al. 2014). This might do more harm than good because although the emergence of the virus has altered the daily routine, it would not be practical to neglect other priorities such as education and work. The virus has inflicted fear upon many individuals, but there are better ways to be cautious than staying updated every minute.

Decision-making might be the cognitive function that has the most influence on life during the pandemic. The ability to make decisions implies a degree of choice because decisions are designated outputs based on sensory inputs or one's perception. Through the decisions that people make, they transform the information into a behavior (Wallisch 2020). During the pandemic, government leaders have been making more profound decisions for the general population. Some of the few ways political leaders have attempted to control the spread of the virus was through curfews and going on "pauses." In New Jersey, for example, people in some cities could only go outside from five in the morning until eight at night. The curfew was lifted recently and the only things that are still enforced are social distancing and wearing surgical masks in public. New York used to be busy every day, but it was announced by Governor Cuomo that the "pause" would be extended until May 28. In the realm of problem solving, the SARS-Cov-2 issue is far from well defined. There are multiple styles to solve problems, two of which are through trial and error, and insight. Because there is no accessible or definite cure for the virus, the best ways to combat its spread are through social distancing, treating illness with other medications, and continuous research (Wallisch 2020). There have been terrible setbacks for the economy and other social systems, but humans have had to make use of decision-making during the pandemic. With insight from previous pandemics like H1N1 and SARS as well as trying different solutions to see what helps, political leaders are actively exercising their decision-making.

Action, in many aspects, is the whole point of having cognitive processes. There are costs for indecisiveness as natural drives like hunger and thirst build up in the brain. When people are inactive, there are consequences. At some point, cognition becomes pointless when people do not act and bring about change in their lives. Evolution usually decides what the necessary action is based on survival and genetics. In real life, there is a speed accuracy tradeoff, but there is also a general outlook that people can get better at certain skills if done continuously. Practice is necessary but not sufficient (Wallisch 2020). During the pandemic, a lot of people are practicing social distancing. Logically, things will get easier when individuals stay home, but it can also be argued that sticking to one routine for a long time will cause more harm than good. "What Coronavirus Isolation Could Do to Your Mind (and Body)" by Emma Grey Ellis is an article that

discusses how individuals' cognition may be affected while self-isolating. When practicing social distancing and quarantine, feelings of loneliness, depression, and anxiety might be of concern. However, actively partaking in social isolation can improve health because people become more accustomed to the routine. The people who have a higher risk while in isolation are those who have a limited network, i.e. marginalized individuals who do not have contact with friends, family, or others. For people living in bigger cities like New York, the transition to social distancing was extremely abrupt and the shift in the daily routine changed immensely. However, it is possible for people to get used to or even thrive while social distancing (Ellis 2020).

Emotional homeostasis is the idea that people have a set point for their emotions and can keep themselves emotionally stable. The pandemic might disturb this balance even though, in principle, change is unlikely. Emotions are hard to measure and they are a controversial subject within science. As stated previously, action makes cognition useful, and emotion is a shortcut for action. In the modern environment, emotions can lead to the right path, but there is also a chance for cognitive override in the form of paralysis (Wallisch 2020). It is likely that decisions made during the pandemic have been emotionally charged, and there might be an offset in embryonal stability for people who lost their jobs, are essential workers, or know of someone who passed away during this time. The number of cases and deaths for SARS-Cov-2 is continuously escalating, which could cause a lot of people great stress. Within the brain, it is speculated that the amygdala is the group of neurons that process emotions (Gazzaniga et. al. 2014). Emotions can influence other cognitive processes including memory, perception, and attention. If more people are driven to act based on their emotions, this may lead them to make decisions they would not normally consider. In addition, since emotions affect the proficiency of other aspects of cognition, it should be very important for people to do things that keep their stress levels low. Social distancing gives people an opportunity to take care of themselves on their own terms. By learning how to handle emotions in a situation like this, individuals can be better equipped for future calamities.

The awareness of SARS-Cov-2 increased dramatically as the number of confirmed cases and deaths increased. Thinking back to the first few days it broke onto the scene, the awareness surrounding SARS-Cov-2 was relatively contained. People knew about it and there was some media coverage, but basic knowledge has since increased almost exponentially. In addition, the issue of denial was a concern. Now, general awareness of the virus is vast, and there are daily updates on multiple news outlets. Although the average citizen knows that SARS-Cov-2 is a real virus, there are still tests being conducted to figure out how to combat it. The awareness between individuals varies greatly, even more so if they are not actively tuning into the news. Regardless, the awareness surrounding the virus has certainly changed ever since it spread throughout the country. Unfortunately, there is uncertainty in regards to when the pandemic will subside or when a cure will be available. For now, some research has determined hydroxychloroquine to be promising, but it is not definite (Wallisch 2020). Despite the amount of pandemic-related news there is, it is ultimately up to communities and each individual to do what they can to keep their cognition in check. People can keep themselves notified very easily, but not all sources are reliable. Awareness is important and the news coverage is a testament to how much people want to know about the virus and the state of the world. Whether it is anticipation for the day when the cure is found and the pandemic can end, awareness can lead societies to be more conscious of what is going on.

How did the pandemic affect my cognition?

Ever since I was three years old, I have only learned in a traditional classroom setting. After nearly two decades of going to school for

nine months a year, this is the first time in my life I had no choice but to take classes online. Even though there were only seven weeks left in the semester, my learning was drastically impacted by SARS-Cov-2. While attending university, I did not use any electronics for my classes. After switching to remote learning, I was only able to learn on my computer. It was extremely inefficient as I was easily distracted by what was going on around me, especially because I was never alone in my house. Furthermore, I found myself checking my phone more often for updates on social media. Taking notes for some of my classes was no longer a possibility and this could be detrimental for future learning. Studies have shown long-term retention of information is reduced when students use electronic devices (Glass & Kang 2019). By rapidly switching from lectures to one's laptop or phone, exam performance is very negatively impacted. Although most exams for my classes were canceled because of the pandemic, I still had one class that gave two exams. My attentiveness during lectures was generally good and focused, but this did not mean my class performance and long-term retention would not suffer. Remote learning is a bit of a conundrum because the electronic devices become the source of learning as well as the source of distraction.

Being at home every day for almost three months has greatly decreased my motivation to do a lot of things. For example, I found myself procrastinating more and spent more time on my phone than I did before. This past semester has challenged me on multiple levels, but most of all on my motivation. Keeping up with the assignments and tuning into lectures felt draining at times. I would not consider myself a good student, but I worked hard and usually my work would pay off. Models on motivation explain the possibilities of how people determine what they want. The Rubicon model proposed by Heinz Heckhausen and Peter M. Gollwitzer in 1987 is a reference to the time of the Roman Republic when armies were banned from the empire. In the past, when armies crossed the Rubicon, it was interpreted to be a declaration of war. By crossing the metaphorical Rubicon in motivation, people realize what they want and can take action on these goals. The primary steps in this motivation model are deliberation, planning, action, and evaluation (Heckhausen & Gollwitzer 1987; Wallisch 2020). Another model of motivation introduced by Gabriele Oettingen in 2014, known as WOOP, follows a certain trajectory: wish, outcome, obstacle, then plan. The logic behind WOOP is that wishful thinking can help individuals with short term and long term goals. When I think about my motivation during this pandemic, I think it fits more with WOOP because I am stuck on the "obstacle" stage. When I took Cognition last semester, I was taught that wishful thinking can actually prevent individuals from achieving their goals (Oettingen 2014; Wallisch 2019). This made more sense to me ever since the university switched to remote because the only obstacles I faced were online due dates. Since most of my lectures were recorded and I had access to the Internet for all my assignments, I wrongly assumed it was possible to do well in my classes just because of the available resources.

I was home for a majority of the time because of the pandemic and there were very few things I could do. For about three weeks, I was not allowed to go outside, even to take a short walk around the neighborhood. Although I still had lectures during that time, my range of action was truncated compared to when I lived in the city. When being at home, I had to be accustomed to staying in one place and following a very basic routine. This loosely reminded me of the concept of sensorimotor adaptation because my daily life was modified by the pandemic (Gazzaniga et. al. 2014). Of course, this form of learning has more to do with a specific skill and a change from the environment, but I think the ability to adapt is extremely necessary to stay sane during the pandemic. I had limited options to keep myself busy and decided to work on knitting. After doing it almost every day for about six hours each session, I learned how to knit while watching television. I would not consider myself a knitting expert because I only know how to knit scarves,

but I am very familiar with the different stitches and was able to complete a scarf in just three days. The continuous practice has strengthened my knitting immensely. It is not the most practical thing to know, but this is one way that I exercised sensorimotor adaptation while being at home.

According to the general understanding of emotion and decision-making, the three types of emotional states that have an influence are current, anticipatory, and expectant (Gazzaniga et. al. 2014). As stated before, emotions can greatly impact individuals' ability to make decisions. I am majoring in psychology, but the main reason I applied to university in the first place was to fulfill the necessary pre-med requirements. Before the pandemic, I was fairly certain I wanted to become a doctor. It was the career path that made the most sense to me because most of my family members are nurses, doctors, and other medicine-related professionals. Ever since the emergence of SARS-Cov-2, I have pondered whether or not this would be the right career choice for me. There have been many days in which my emotions would fluctuate. Depending on what I had going on during the week, in terms of assignments or house duties, I would often rethink my decision to pursue a medical degree and try to figure out how to get out of it without completely abandoning the track that I am currently on. When I think about where I am in five years, I do not want to look back and regret emotion-driven decisions. At this point, however, I fully intend to complete the pre-health requirements. As long as I complete my major, I would not consider it a loss to take classes on organic chemistry and physics. While the pandemic is still going on and I still have two years left of my undergraduate career, I feel as if it is too soon to make major decisions like whether or not I should go to medical school.

My awareness of the virus increased while under quarantine because three weeks after the university switched to remote learning, both of my parents tested positive for SARS-Cov-2. The symptoms varied greatly between my mother and father, but I did not learn of their condition until after the fact. They both kept their distance by staying in the basement and limiting any contact to maybe an hour a day. Additionally, I learned that one of my family members also tested positive for the virus because someone who worked with her was a carrier. Even though I received new updates on my phone about the virus and tuned into televised news weekly, I do not think I was fully processing what I read and heard with regards to SARS-Cov-2. There was a general understanding that it was highly contagious, but I was naïve to think that I would never have to worry about the virus as long as I kept my distance. When I found out multiple people in my life tested positive, it was as if SARS-Cov-2 became a real thing for me. Awareness was not enough before these events happened and it may have been because I thought it would be something I did not have to worry about. After their health improved I stopped worrying as much as I initially did, but I am definitely more alert because of what happened while they were sick. My mother lost her sense of smell and taste which was not the worst thing, but my father had more severe symptoms. He refused to eat because he physically could not handle having food in his system and there was about a week when he was unable to talk or even stay up for long periods of time. The one impact that the virus had on my father that I thought was the most fascinating was in regards to his memory.

People are able to modify behavior, accumulate experiences, and create a record of knowledge during their lifetime because of memory. It is the ability to store, retrieve, and utilize information over time (Wallisch 2020). I thought it was very interesting, albeit terrifying, how my father was essentially oblivious to what was going on around him when he contracted the virus. Also, he was only able to recall the names of a few people he knew when his physical health deteriorated greatly. Fortunately, he recovered and his memory seems to be fine now. When thinking about memory and its neural basis, the brain is the major organ that makes memory storage possible. The brain is not a static organ and has use-dependent plasticity.

The microstructures change often based on how they are used. As a result, all individuals are responsible for their own cognitive health through their behaviors (Wallisch 2020). My father had a temporary relapse in his memory proficiency and I suspect, in his case, that it was a retrieval issue because when he got better, it was as if he did not contract the virus in the first place. One of the first things he did when his health improved was play the guitar, and this is considered to be a learned behavior. Based on what happened to my father, I think SARS-Cov-2 can have an effect on memory. It is definitely an interesting time to be alive, despite the many tragedies of living in a pandemic.

Relatively speaking, I consider my experiences living in the pandemic quite idiosyncratic. I was not severely impacted when the university closed or when my state enforced protocols like curfews and social distancing. Thinking about my friends and family who live in different places, most of them were in the same situation as I am. They are living at home with other people and only going outside for necessities like food or toiletries. What I learned from living in this pandemic was that cognition never takes a pause, especially when the world is faced with a dire situation like the SARS-Cov-2 outbreak. This is a truly challenging time and there is great uncertainty in the outcome. However, I am sure that healthy cognition is more important than ever. It is something that people take for granted or are not even fully aware of, but it will be present for individuals at all points in their lives.

References

Ellis, E. G. (2020, March 25). What Coronavirus Isolation Could Do to Your Mind (and Body). *Wired*. https:// www.wired.com/story/coronavirus-covid-19-isolation-psychology/

Gazzaniga, M. S., Ivry, R. B., Mangun, G. R. (2014). Cognitive Neuroscience: The Biology of the Mind. *W. W. Norton & Company, Inc.* Fourth Edition.

Glass, A. L. & Kang, M. Dividing attention in the classroom reduces exam performance. *Educational Psychology*, Vol. 39, No. 3, 395-408.

Heckhausen, H., Gollwitzer, P. M. (1987). Thought contents and cognitive functioning in motivational versus volitional states of mind. *Motiv Emot* 11, 103. https://doi.org/10.1007/BF00992338

Molten, M. (2020, April 15). What Does Covid-19 Do to Your Brain?. *Wired*. https:// www. wired.com/story/what-does-covid-19-do-to-your-brain/

Oettingen, G. (2014). WOOP: A scientific strategy to find and fulfill wishes https:// woopmylife.org/en/science

Wallisch, P. (2019-2020). Cognition & Cognitive Neuroscience. New York University

Effects of Gender Bias in Workplace on Female Leadership Attainment

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Despite women's increasing participation in the labor force, there is still a huge gender gap in leadership positions, with women making up only 5% of all CEO-level positions (Elliott & Smith, 2004; Kaiser & Wallace, 2016). The most prominent factor influencing working women's success as leaders appears to be a gender bias against women, which includes negative evaluations and treatment that stem from negative female stereotypes (Heilman, 2001, 2012). Specifically, gender bias mainly affects their leadership attainment in two aspects: entry into leadership positions and further development upon entry (Kaiser & Wallace, 2016; Samuelson et al., 2019). Bias influences evaluations of candidates and decisions of new hires and promotions thereafter, which determines women's entry to leadership (Heilman, 2001; Eagly & Karau, 2002). Additionally, bias against women holding leadership positions prevents them from excelling in these positions (Elsesser, 2016; Brescoll & Uhlmann, 2008). In order to close the gender gap, there must be a more robust understanding of how bias influences female leaders' entry and development opportunities in the leadership attainment process. This leads to the question: How does gender bias against female leaders influence their opportunities for leadership attainment?

Gender Bias and Hiring and Promotion Opportunities

Gender bias is largely influential in the evaluation of female leaders in hiring and promotion processes (Bosak & Sczesny, 2011; Gorman, 2005). The evaluations, in turn, influence hiring and promotion decisions of leadership positions through the lack of fit model (Heilman, 2001). According to this model, decision-makers (e.g., hiring managers) tend to give biased and negative evaluations when female candidates show attributes that match female gender stereotypes (e.g., being communal) rather than the typical characteristics of a successful male leader (Heilman, 2001; Ryan et al., 2011). Furthermore, the mismatch between traditional attributes of female gender roles and the attributes of strong leaders allows decision makers to form a habit of adopting less-positive attitudes when evaluating female candidates compared to male candidates (Eagly & Karau, 2002). Even when female candidates enact behaviors that fulfill the expectations of a leader role, decision-makers acknowledge their behaviors less than they do for similar behaviors enacted by male candidates (Bosak & Sczesny, 2011; Eagly & Karau, 2002; Gorman, 2005). As a result, women who are equally capable leaders as their male counterparts get more biased and disadvantaged evaluations, hindering them from the acquisition of leadership positions over men (Bosak & Sczesny, 2011; Gorman, 2005; Isaac et al., 2009).

On a societal level, the biased evaluations and rejections of potential female leaders in hires and promotions are further perpetuated because of the same-gender preference in hiring and promotion (Fitzsimmons et al., 2014; Reskin, 2001). As male decision-makers are more conscious about the lack of fit between stereotypical female attributes and traditional leaders' attributes, they favor men over women when hiring (Reskin, 2001; Gorman, 2005). With the combination of a large number of male decision-makers in society and their same-gender preferences, the gender bias against female leaders

is deeply rooted and remains dominant across industries and settings, leading to fewer successful hires and promotions of female leaders across many industries in the long term (Bosak & Sczesny, 2011; Gorman, 2005; Reskin, 2001).

Gender Bias and Leadership Development

Another aspect of leadership attainment that gender bias influences is leadership development. The most pervasive belief about women in terms of leadership development is that women need to be protected by men and cannot excel in most tasks that are essential in leadership roles (Elsesser, 2016; Glick & Fiske, 1999). Supervisors who hold this bias, in turn, are less likely to provide female leaders with the most helpful opportunities to develop their leadership competence (De Pater et al., 2010). According to recent studies, female leaders receive less challenging and prestigious tasks in addition to fewer networking opportunities with important business partners as compared to their male counterparts (Fitzsimmons et al., 2014; King et al., 2012). As a result, it is a longer, more difficult process for women to further elevate themselves in the organizational hierarchy (Samuelson et al., 2019).

Gender bias also influences leadership development and attainment by negatively impacting the legitimacy of female leaders' power in work environments (Vial et al., 2016). Specifically, it is difficult for female leaders to gain authority and leadership effectiveness because their subordinates show less respect for them out of their biased evaluation that women cannot lead well due to stereotypical female characteristics (Eagly & Karau, 2002; Vial et al., 2016). For example, Brescoll & Uhlmann (2008) have shown that female leaders receive more negative evaluations from subordinates than male leaders do for being more emotionally expressive (a stereotypical description of women), which leads to fragile workplace relationships and professional networks. Furthermore, female leaders' further elevation is hindered by the social and economic penalties from subordinates and colleagues who do not acknowledge the leaders' legitimacy (Phelan & Rudman, 2010; Vial et al., 2016). Studies have shown that female leaders suffering from penalties (e.g., receiving lower salaries and lower desire of cooperation from colleagues) become disadvantaged in succeeding in current positions and lose competitiveness for future promotions (Heilman & Okimoto, 2007; Fitzsimmons et al., 2014; Brescoll & Uhlmann, 2008). Clearly, due to subordinates' bias, female leaders suffer from illegitimation of their power and status as they navigate through their leadership paths (Brescoll & Uhlmann, 2008; Heilman, 2012; Vial et al., 2016).

Conclusion

Gender bias against working women largely influences the significant gender gap in leadership attainment in the workplace by creating and persisting obstacles for entry to positions and leadership development (Elliott & Smith, 2004; Elsesser, 2016; Samuelson et al., 2019). Understanding the possible models of thoughts helps to clarify how bias leads to practical problems for female leaders in the work environment (Heilman, 2001; Eagly & Karau, 2002; Glick & Fiske, 1999). As discussed previously, problems caused by bias include rejections of qualified female leaders' candidates by hiring and promotion decision makers, scarce opportunities for developing leadership competence from supervisors, and illegitimation of power due to biased colleagues (Bosak & Sczesny, 2011; De Pater et al., 2010; Vial et al., 2016). Although there has been extensive research examining bias against working women from the perspectives of different groups around female leaders, there is mixed evidence showing that levels of bias detected in experimental conditions differ from those detected in real workplace settings (Elsesser, 2016). As research findings aim to minimize and eliminate the gender gap in organizational leadership powers, future research should focus more on examining the influence of

bias while ensuring externally valid findings and results. Furthermore, future research should examine how bias could be reduced through changes in organizational structures and processes and explore the possibility of interventions in organizations.

References

Bosak, J., & Sczesny, S. (2011). Gender bias in leader selection? Evidence from a hiring simulation study. *Sex Roles*, *65*, 234–242. https://doi.org/10.1007/s11199-011-0012-7

Brescoll, V. L., & Uhlmann, E. L. (2008). Can an angry woman get ahead? Status conferral, gender, and expression of emotion in the workplace. *Psychological Science*, *19*(3), 268–275. http:// doi.org/10.1111/j.1467-9280.2008.02079.x

De Pater, I. E., Van Vianen, A. E., & Bechtoldt, M. N. (2010). Gender differences in job challenge: A matter of task allocation. *Gender, Work and Organization*, *17*(4), 433–453. https://doi.org/10.1111/j.1468-0432.2009.00477.x

Eagly, A. H., & Karau, S. J. (2002). Role congruity theory of prejudice toward female leaders. *Psychological Review*, *109*(3), 573–598. https://doi.org/10.1037/0033-295X.109.3.573

Elliott, J. R., & Smith, R. A. (2004). Race, gender, and workplace power. *American Sociological Review*, *69*(3), 365–386. https://doi.org/10.1177/000312240406900303

Elsesser, K. M. (2016). Gender bias against female leaders: A review. In M. L. Connerley, & J. Wu (Eds.), *Handbook on Well-Being of Working Women* (pp 161–173). https:// doi.org/10.1007/978-94-017-9897-6_10

Fitzsimmons, T. W., Callan, V. J., & Paulsen, N. (2014). Gender disparity in the C-suite: Do male and female CEOs differ in how they reached the top? *The Leadership Quarterly*, *25*(2), 245–266. http://doi.org/10.1016/j.leaqua.2013.08.005

Glick, P., & Fiske, S. T. (1999). *Sexism and other "isms": Independence, status, and the ambivalent content of stereotypes.* In W. B. Swann, Jr., J. H. Langlois, & L. A. Gilbert (Eds.), *Sexism and stereotypes in modern society: The gender science of Janet Taylor Spence* (pp. 193–221). American Psychological Association. https:// doi.org/10.1037/10277-008

Gorman, E. H. (2005). Gender stereotypes, same-gender preferences, and organizational variation in the hiring of women: Evidence from law firms. *American Sociological Review*, *70*(4), 702–728. https://doi.org/10.1177/000312240507000408

Heilman, M. E. (2001). Description and prescription: How gender stereotypes prevent women's ascent up the organizational ladder. *Journal of Social Issues*, *57*(4), 657–674. https:// doi.org/10.1111/0022-4537.00234

Heilman, M. E. (2012). Gender stereotypes and workplace bias. *Research in Organizational Behavior*, *32*, 113–135. https:// doi.org/10.1016/j.riob.2012.11.003

Heilman, M. E., & Okimoto, T. G. (2007). Why are women penalized for success at male tasks? The implied communality deficit. *Journal of Applied Psychology*, *92*(1), 81–92. http://doi.org/10.1037/0021-9010.92.1.81

Isaac, C., Lee, B., & Carnes, M. (2009). Interventions that affect gender bias in hiring: A systematic review. *Academic Medicine: Journal of the Association of American Medical Colleges*, *84*(10), 1440–1446. http://doi.org/10.1097/ACM.0b013e3181b6ba00

Kaiser, R. B., & Wallace, W. T. (2016). Gender bias and substantive differences in ratings of leadership behavior: Toward a new narrative. *Consulting Psychology Journal: Practice and Research*, *68*(1), 72–98. http:// doi.org/10.1037/cpb0000059

King, E. B., Botsford, W., Hebl, M. R., Kazama, S., Dawson, J. F., & Perkins, A. (2012). Benevolent sexism at work: Gender differences in the distribution of challenging developmental experiences. *Journal of Management*, *38*(6), 1835–1866. https://doi.org/10.1177/0149206310365902

Reskin, B. F. (2001). Employment discrimination and its remedies. In I. Berg & A. L. Kalleberg (Eds.), *Sourcebook of Labor Markets* (pp. 567–599). Springer. https:// doi.org/10.1007/978-1-4615-1225-7_23

Ryan, M. K., Haslam, S. A., Hersby, M. D., & Bongiorno, R. (2011). Think crisis–think female: The glass cliff and contextual variation in the think manager–think male stereotype. *Journal of Applied Psychology*, *96*(3), 470–484. https://doi.org/10.1037/a0022133

Phelan, J. E., & Rudman, L. A. (2010). Prejudice toward female leaders: Backlash effects and women's impression management dilemma. *Social and Personality Psychology Compass*, *4*(10), 807–820. https:// doi.org/10.1111/j.1751-9004.2010.00306.x

Samuelson H. L., Levine, B. R., Barth, S. E., Wessel, J. L., & Grand, J. A. (2019). Exploring women's leadership labyrinth: Effects of hiring and developmental opportunities on gender stratification. *The Leadership Quarterly*, *30*(6), Article 101314. https://doi.org/10.1016/j.leaqua.2019.101314

Vial, A. C., Napier, J. L., & Brescoll, V. L. (2016). A bed of thorns: Female leaders and the self-reinforcing cycle of illegitimacy. *The Leadership Quarterly*, *27*(3),400–414. https://doi.org/10.1016/j.leaqua.2015.12.004

Upgrading Study Abroad and Cultural Exchange Programs

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Study abroad programs are intended to widen students' worldviews by taking them to different countries. Lilli and John Engle articulated this idea well in their research from 2003:

> Study abroad is not about providing immediate comfort and services to clients, safe and familiar cultural bubbles, moving bodies around geographically, [or] simple changes of scenery. It's about recognizing the challenge that true involvement in an unfamiliar world represents and choosing the hard, progressive road to understanding what Hall calls the 'inherent logic' of a foreign culture. (Engle & Engle 2003)

Given our continuously globalizing world and the increasing value of being globally informed, students going into cultural exchange programs have increased. Globalization has also made mass education much more popular over the past millennia; hence, studying abroad has become an educational norm that may have caused a downward shift in quality. Education abroad looks excellent on paper since it encourages students to open their minds and look at the world differently, perhaps with more empathy given the myriad experiences one can explore abroad. However, the reality for many of these programs looks as if students abroad are just attending regular school in a different country. Study abroad programs have been one of the most "understudied areas" in recent years and have become a focus of study for many researchers trying to discover their benefits (Streitweiser 2012). I want to add to that discussion by arguing that study abroad programs can be more beneficial for students if they are designed using more effective learning theories, planned and implemented by the correct international actors, and if their duration is optimized for student needs.

By intentionally planning and utilizing Experiential Learning Theory and Transformative Learning Theory in tandem with one another, study abroad programs could provide more valued experiences to participants. Experiential learning is defined as "the type of education whereby knowledge and meaning are contextualized in actual experiences" (Strange 2017). In contrast, Transformative learning incorporates "reflection, active learning, and placing ourselves in an uncomfortable situation [... in order] to develop our understanding of the world and of ourselves" (Strange 2017). In Mezirow's 2003 study of these two combined theories, he stated that:

> Since the outcomes of both experiential and transformative learning are in alignment with those desired in study abroad, it is appropriate to use them both as frameworks to assess the effectiveness of a variety of study abroad models (Mezirow 2003 as cited in Strange 2017).

As of now, study abroad programs typically let the combination of these theories happen by chance, as students learn different cultural norms or languages. However, these two learning theories must be conjoined in study abroad curriculums since they allow students to approach learning intuitively. For example, suppose students were instructed to note down the struggles of adapting to the country. In that case, they are studying in (experiential learning), and then with guidance, instructed to reflect on

how to overcome the hardships (transformative learning), since the student's initial view changed through a live experience equates to successful learning abroad. These two theories complement one another, and they lay out a plan for how students approach new experiences and reflect on them. Additionally, this kind of learning process may produce by-products of adaptability and cultural sensitivity since compelling study abroad programs force students to learn and then reflect on situations within this environment. Hannah Strange's research on study abroad in 2017 incorporated interviews with students, and when she asked them about their experiential learning, she received mixed reviews. One student claimed," 'I would have liked more interaction with the local community as a part of the program'" (Strange 2017). About "half of the responses reported that the field trips and interactions with the community were the most influential aspects of their program, making comments like 'tours of public hospitals had the most impact.'" (Strange 2017). As for transformative learning when applied to study abroad programs yielded "the creation of a new frame of reference that promotes cultural pluralism (Berwick & Whalley 2000 as cited in Strange 2017). As one can observe, study abroad programs are still not necessarily hitting all of the experiential and transformative learning targets they may want. The components for successful study abroad programs are present to make them truly effective and beneficial to the participants. Aligning these two learning theories will allow study abroad programs to complete the learning arc for students and make these programs more effective.

An area many study abroad programs ignore is the back-end of the program that requires support from different organizations, and an untapped source of support could be the international actors to improve programs in many various aspects. International actors include research institutes, universities, NGOs, and UN organizations (like UNESCO and UNICEF). However, one kind of actor that has been neglected is the policymaker, "who commonly seek information about [educational] models." (Bray 2014). Through study abroad programs, policymakers can use students as live subjects to assess the effectiveness of particular educational systems. For example, if a developing country wanted to see true educational reform, they would send students from their country to an industrialized country like Finland. Finland has "persistently outperform[ed] other nations" on standardized exams and is a key point of emulation by the education systems of other countries (Sahlberg 2011). The policymakers could then interview and study those students since they already experienced specific policies they may borrow from the more developed country. Essentially, the policymaker would use educational comparison and study abroad to improve educational policy. If policymakers drove this concept of study abroad and educational policy, it would give the study abroad programs more legitimacy, focus, and purpose. Hypothetically, a program like this would have a goal of finding specific teaching methods which were effective and which the government of one country could apply to their system. For these reasons, policymakers are one international actor that must be brought in to support study abroad programs. The second kind of actor is academics in comparative education. These academics "are concerned with conceptual and theoretical work. Sometimes they collaborate with policymakers and international agencies in the analysis of data" (Bray 2014). Bringing on academics to support study abroad will legitimize their argument for students to participate. Their support for programs will be invaluable because they can improve these cultural exchange programs through learning theories and further research. Moreover, academics will be a crucial focal point of support as more and more studies continue to look at the study abroad field. Study abroad programs must engage with educational policymakers and academics to see how these actors can utilize and plan abroad programs.

The last part of study abroad programs that need improvement is their duration.

Currently, both long and short study abroad programs face a problem between balancing cost-effectivity and learning outcomes. This has become an issue because it "is possible that short-term programs are the only realistic option for many students" (Strange 2017). That is why I would like to present an alternative that hits the "sweet spot" of duration during an abroad program. According to Lilli and John Engle's study in 2003, they believe that a "typical four- to six-week [...] course allows students a first exposure to language and civilization in its cultural setting" (Engle 2003). This evidence demonstrates a target of duration that balances out cost efficiency with good learning outcomes. For example, Anderson et al. "found that in a group of study abroad students there was a considerable increase in their intercultural sensitivity despite only participating in a four-week program" (Anderson 2006 as cited in Strange 2017). Furthermore, if study abroad programs were honed in on this four to six-week mark, it would allow students not to waste time abroad, away from their primary institution. Also, studying abroad will no longer be a critical decision with a shortened programming duration. Some students may even feel more financially and personally comfortable studying in multiple abroad sites during their college career. Regardless of the optimized length of these programs, the only disadvantage is that there is significantly less leeway for lesson planning. Faculty will need highly structured activities to achieve the learning outcomes. As found by Gerald W. Fry in 2009, a professor of international development education, "states, 'If it's done right, if it's done with intensity of learning, a short-term program can have impact'" (Ritz 2011). Shorter abroad programs, hitting the sweet spot of four to six weeks with the correct planning, are the exact solution, which students with a budget-sensitive academic college plan can use to go abroad. As of now, "at many colleges, students participating in any study abroad program retain their status as full-time students and pay regular tuition fees" (Heitmann 2008). Many students' only option is to attend school for a semester or year, whereas a shorter program could be much cheaper. Additionally, students pay lower tuition to compensate for not being at their primary institution. If it is too daunting, students will have an intense academic plan laid out for them to adapt, with plenty of guidance. By making more time-efficient study abroad, programs will be revolutionary since they cater to the student needs without sacrificing the desired learning outcomes of institutions.

Study abroad programs can be incredible experiences where revolutionary learning theories educate students; they can be pathbreaking for different research-based international actors, and their duration could be optimized to fit all students' needs. If cultural study abroad programs are improved upon in educational policy, they could be the next big academic step as our world becomes more and more connected. Studying abroad could even solve US-centric social problems in the next generation's students. The experiences when a student travels to another country are priceless, and with proper planning, these experiences could genuinely prepare the students of tomorrow to enter the complex global society. Globalization is not something that plans on stopping, and education will have to adapt to keep up, and I believe studying abroad is at the helm of this educational evolution.

References

Bray, M. (2014). Actors and Purposes in Comparative Education. *Comparative Education Research*, 19-46. doi:10.1007/978-3-319-05594-7_1

Engle, L., & Engle, J. (2003). Study Abroad Levels: Toward a Classification of Program Types. *Frontiers: The Interdisciplinary Journal of Study Abroad, 9*(1), 1-20. doi:10.36366/frontiers.v9i1.113

Heitmann, G. (2007). The Opportunity Cost of Study Abroad Programs: An Economics-Based Analysis. *Frontiers: The Interdisciplinary Journal of Study Abroad, 15*(1), 57-66. doi:10.36366/frontiers.v15i1.216

Ritz, A. A. (2011). The Educational Value of Short-Term Study Abroad Programs as Course Components. *Journal of Teaching in Travel & Tourism, 11*(2), 164-178. doi:10.1080/15313220.2010.525968

Sahlberg, P. (2011). The Fourth Way of Finland. *Journal of Educational Change, 12*(2), 173-185. doi:10.1007/s10833-011-9157-y

Strange, H., & Gibson, H. (2017). An investigation of experiential and transformative learning in study abroad programs. *Frontiers: The Interdisciplinary Journal of Study Abroad, 29*(1), 85-100. doi:10.36366/frontiers.v29i1.387

Streitwieser, B. T., Le, E., & Rust, V. (2012). Research on Study Abroad, Mobility, and Student Exchange in Comparative Education Scholarship. *Research in Comparative and International Education, 7*(1), 5-19. doi:10.2304/rcie.2012.7.1.5

Poisson-Gamma Neural Variability In The Visual Cortex

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In neural coding, a population of sensory neurons responds to the same repeated stimuli with differing spiking variability. The stochasticity in the neural responses is characterized as "trial-to-trial variability". A classical model in neural encoding captures the response variability using the poisson rate model:

$$N \sim \text{Poisson}(\lambda), \lambda \geq 0,$$

where $\lambda = \mu\Delta t$ is the rate parameter, μ denotes the mean spike rate, Δt denotes the duration of the counting window, and N represents the spike count (Goris et al., 2014). So we have the spike count distribution:

$$P(N = n|\lambda) = \frac{\lambda^n}{n!}\exp(-\lambda)$$

with equal mean and variance, i. e. $E[N] = Var[N] = \lambda$. In sensory system and particularly in visual cortex, there is usually higher variance than the mean in the population responses, a phenomenon called as "overdispersion" (Taouali et al., 2016). Overdispersion in neural data represents modulatory influences (attention, adaptation etc.) coming from non-sensory sources, and the response variability increases along the visual pathways from LGN, V1, V2, to MT (Goris et al., 2014).

Poisson-Gamma Model

To capture the overdispersion, this project paper implements a doubly-stochastic poisson-gamma model by Goris et al (2014). We define the modulatory influence, or gain signal, as G. Given the poisson-gamma framework, the mean spike rate μ is the product of a stimuli-independent gain signal G and a function of the stimuli f(S):

$$\mu = f(S)G \Rightarrow \lambda = f(S)G\Delta t$$

The gain signal follows a gamma distribution:

$$G \sim \text{gamma}(r, s),$$

where the gamma distribution is parameterized by the shape parameter r and the scale parameter s. Here we set $E[G] = rs = 1$, $Var[G] = rs^2 = s$, where $s = \sigma^2 G$, $r = 1\ \sigma^2 G$ (later in the numerical simulation we will explore the decoding accuracy of both $[r, s] = [\sigma^2 G, \sigma 2\ G]$ and $[r, s] = [\ 1\ \sigma^2 G\ , \sigma 2\ G]$). Here $\sigma^2 G$ denotes the variances of the gain signal G. Now since $\lambda = f(S)G\Delta t$ where $f(S)\Delta t$ is a constant, λ is now a random variable Λ with a gamma distribution:

$$\Lambda \sim \text{gamma}(r, sf(S)\Delta t)$$

$$P(\Lambda = \lambda) = \frac{\lambda^{r-1}\exp(-\lambda/[s(f(S)\Delta t)])}{\Gamma(r)[s(f(S)\Delta t)]^r}$$

So for $P(N|\Lambda)$ and $P(\Lambda)$ as a poisson-gamma mixture, the marginal distribution of N should be negative-binomial (Gelman et al., 2013, p. 44).

$$\text{Neg-Bin}(n|r, s) = \int Poisson\,(n|\lambda)\text{Gamma}(\lambda|r, s)d\lambda.$$

Now we can compute the marginal distribution of N:

$$= \int_0^\infty \frac{\lambda^n}{n!}\exp(-\lambda)\frac{\lambda^{r-1}\exp(-\lambda/[s(f(S)\Delta t)])}{\Gamma(r)[s(f(S)\Delta t)]^r}d\lambda.$$

Let $\alpha = s(f(S)\Delta t)$, we have.

$$\int_0^\infty \frac{\lambda^n}{n!}\exp(-\lambda)\frac{\lambda^{r-1}\exp(-\lambda/\alpha)}{\Gamma(r)\alpha^r}d\lambda$$
$$= \int_0^\infty \frac{\alpha^{-r}}{n!\,\Gamma(r)}\lambda^{n+r-1}e^{-(1+\frac{1}{\alpha})\lambda}\,d\lambda$$
$$= \frac{\alpha^{-r}}{n!\,\Gamma(r)}\Gamma(n+r)\left(\frac{\alpha}{\alpha+1}\right)^{n+r}\int_0^\infty \frac{1}{\Gamma(n+r)\left(\frac{\alpha}{\alpha+1}\right)^{n+r}}\lambda^{n+r-1}e^{-(1+\frac{1}{\alpha})\lambda}\,d\lambda$$
$$= \frac{\alpha^{-r}}{n!\,\Gamma(r)}\Gamma(n+r)\left(\frac{\alpha}{\alpha+1}\right)^{n+r} = \frac{\Gamma(n+r)}{\Gamma(N+1)\,\Gamma(r)}\alpha^{-r}\left(\frac{\alpha}{\alpha+1}\right)^{n+r}$$
$$= \frac{\Gamma(n+r)}{\Gamma(N+1)\,\Gamma(r)}\alpha^{-r}\left(\frac{\alpha}{\alpha+1}\right)^{n+r} = \frac{\Gamma(n+r)}{\Gamma(N+1)\,\Gamma(r)}\left(\frac{1}{\alpha+1}\right)^{r}\left(\frac{\alpha}{\alpha+1}\right)^{n}.$$

So we have the spike count N following a negative-binomial, or poisson-gamma mixture, distribution:

$$N \sim \text{Neg-Bin}(r,\alpha),\ \text{where}$$
$$P(N=n) = \frac{\Gamma(n+r)}{\Gamma(N+1)\,\Gamma(r)}\left(\frac{1}{\alpha+1}\right)^{r}\left(\frac{\alpha}{\alpha+1}\right)^{n},$$

where

$$E[N] = r\alpha = rs(f(S)\Delta t) = f(S)\Delta t$$
$$\text{Var}[N] = r\alpha + r\alpha^2 = f(S)\Delta t + (\sigma_G^2)(f(S)\Delta t)^2.$$

To quantify the extent of overdispersion, we use Fano Factor F, defined as the variance over the mean. For poisson rate model, $F_{Poi} = 1$. For the negative binomial model, we have

$$F_{\text{Neg-Bin}} = \frac{\text{Var}[N]}{E[N]} = 1 + \alpha = 1 + \sigma_G^2(f(S)\Delta t).$$

(Notice that FNeg-Bin is not changed for either $[r, s] = [\sigma^2 G, \sigma^2 G]$ or $[r, s] = [\frac{1}{\sigma^2 G}, \sigma^2 G]$). So the overdispersion in the negative-binomial neural population relative to the poisson population is

$$F' = F_{\text{Neg-Bin}} - F_{\text{Poi}} = \sigma_G^2(f(S)\Delta t),$$

where F' is determined by the product of the gamma parameter $\sigma^2 G$ and the stimulus tuning.

Encoding

A numerical simulation is performed to determine the fluctuations in the Fano Factor with varying stimulus values and gamma parameters. For simplicity, we assume Δt to be 1. A gaussian tuning curve with $g = 15$, $b = 0.1$, $\sigma = 5$ is chosen with the preferred orientations of 20 to 40 and the true stimuli values ranged from 0 to 60 for a population of 50 neurons. The gamma gain signal G is parametrized by the scale parameter s and the shape parameter r from 1 to 81 in 50 steps. The gamma values are generated by 100 trials for every different scale parameter s and put through a poisson random number generator to create a poisson-gamma distribution for every different gamma parameters and stimulus values. Then fano factors are plotted for varying stimulus values and gamma parameters.

$$[r, s] = [\tfrac{1}{\sigma_G^2}, \sigma_G^2]$$

If we set $r = s^{-1}$, we will have the following figure

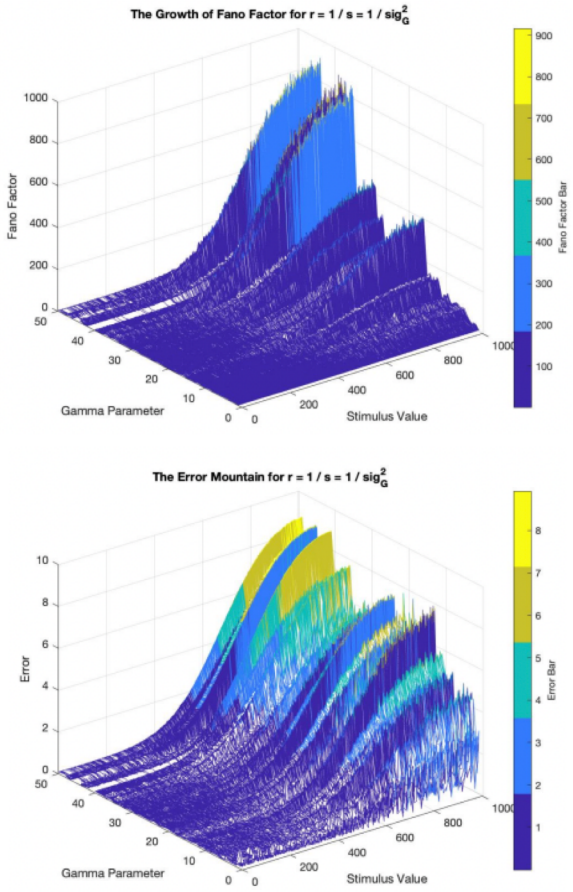

Figure 1: Encoding Performance for $[r, s] = [\frac{1}{\sigma_G^2}, \sigma_G^2]$.

It can be seen that fano factor generally increase for increasing gamma parameters and stimulus values, but there are some fluctuations. This fluctuability could be due to matlab underflow since there might be a parameter combination such as [r, s] = [$\frac{1}{81}$, 81] that is not numerically stable. To determine the extent to which the growth in fano factor is not coming from the product of $\sigma^2 G$ and $f(S)\Delta t$, we define the error term

$$\mathcal{E}_F = |f(S)\Delta t - f(\hat{S})\Delta t| \text{ ,where } f(\hat{S})\Delta t = \frac{F_{\text{Neg-Bin}} - 1}{\sigma_G^2},$$

$$\text{for } F_{\text{Neg-Bin}} = 1 + \sigma_G^2 (f(S)\Delta t).$$

Plot εF and we get the Error Mountain above. Since εF fluctuates on a small scale in general, it can be concluded that the excess in fano factors comes from the product between $\sigma^2 G$ and $f(S)\Delta t$, as our derivation predicted.

$$[r, s] = [\sigma_G^2, \sigma_G^2]$$

Set $r = s = \sigma^2 G$ and plot the fano graph. From the figure, it is obvious that the fano factors increase as gamma parameters and stimulus values increase. The error is also small and it corresponds to our initial hypothesis.

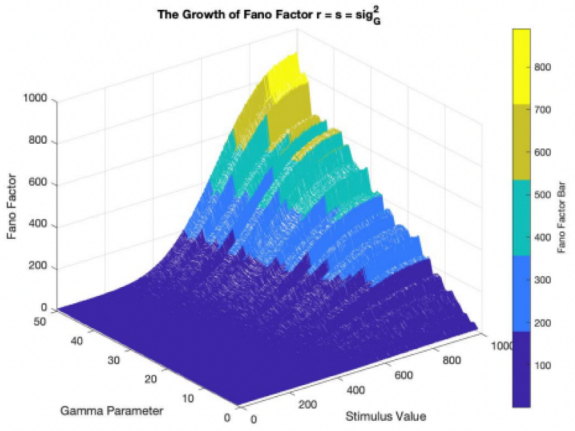

Figure 2: Encoding Performance for $[r, s] = [\sigma_G^2, \sigma_G^2]$.

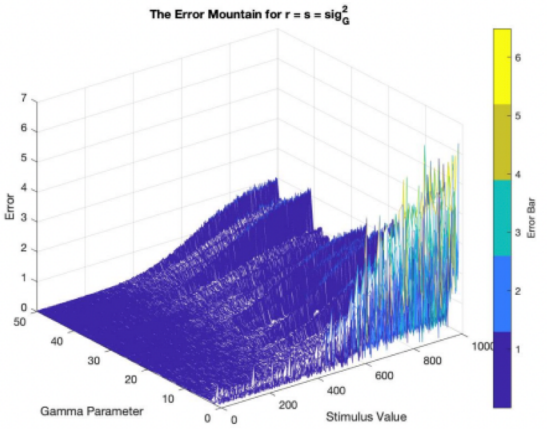

Figure 2: Encoding Performance for $[r, s] = [\sigma_G^2, \sigma_G^2]$.

Decoding

During the encoding process, the parameters of the gain signal G is parametrized as $r = \sigma^2 G$, $s = \sigma^2 G$. For decoding, both $[r, s] = [\sigma^2 G, \sigma^2 G]$ and $[r, s] = [\frac{1}{\sigma^2 G}, \sigma^2 G]$ is explored. Here we will do $[r, s] = [\frac{1}{\sigma^2 G}, \sigma^2 G]$ first (different from the encoding part above, where $[r, s] = [\sigma^2 G, \sigma^2 G]$).

$$[r, s] = [\tfrac{1}{\sigma_G^2}, \sigma_G^2]$$

Rewrite r as s^{-1}, α as $s(f(S)\Delta t)$ we get:

$$P(N = n) = \frac{\Gamma(n + s^{-1})}{\Gamma(N + 1)\,\Gamma(s^{-1})} \left(1 + \alpha\right)^{-s^{-1}} \left(\frac{\alpha}{\alpha + 1}\right)^n$$

$$= \frac{\Gamma(n + s^{-1})}{\Gamma(N + 1)\,\Gamma(s^{-1})} \left(1 + s(f(S)\Delta t)\right)^{-s^{-1}} \left(\frac{s(f(S)\Delta t)}{s(f(S)\Delta t) + 1}\right)^n,$$

where

$$N \sim \text{Neg-Bin}(s, f(S)\Delta t).$$

The log likelihood is then

$$l(s, f(S)\Delta t) = \log\left(\prod_{i=1}^{N} \frac{\Gamma(n_i + s^{-1})}{n_i!\,\Gamma(s^{-1})} \left(1 + s(f(S)\Delta t)\right)^{-s^{-1}} \left(\frac{s(f(S)\Delta t)}{s(f(S)\Delta t) + 1}\right)^{n_i}\right)$$

Simplify then we get the log likelihood equation (Piegorsch, 1990):

$$l(s, f(S)\Delta t) \propto \frac{1}{m} \sum_{i=1}^{m} \sum_{v=0}^{n_i - 1} \log\{1 + sv\} + \bar{n} \log\{f(S)\Delta t\} - (\bar{n} + s^{-1}) \log\{1 + s f(S)\Delta t\}.$$

Take the partial derivative of l with respect to $f(S)\Delta t$ we get

$$\nabla_{f(S)\Delta t}\, l = \frac{\bar{n}}{f(S)\Delta t} - \frac{(\bar{n} + s^{-1}) \times s}{1 + sf(S)\Delta t}$$

$$= \frac{\bar{n}}{f(S)\Delta t} - \frac{1 + s\bar{n}}{1 + sf(S)\Delta t}$$

Now set ∇f(S)Δt l = 0, then we have ˆ f(S)Δt = ¯n (Piegorsch, 1990). The ML estimator for the parameter f(S)Δt is just the sample mean ¯n, i. e. the mean spike count (Anscombe, 1950). This result from statistical literature implies that the stimulus value f(S) can be decoded independently regardless of the values of the gain signal. It is consistent with the claim that "the precision of decoding of the PM and the NBM under the nontuned dispersion hypothesis are very similar" (Taouali et al., 2016, p. 441).

Population decoding is performed in nbm decoding.m. To prevent unexpected fluctuations in the population responses, the Matlab command nbinrnd is used to encode a negative binomial population instead of nesting gamrnd inside poissrnd. Maximum likelihood estimation is performed on the encoding population. There are trials where the sample variance exceeds the sample mean such that the parameter estimation is not possible for the maximum likelihood estimation of the negative binomial parameters. Given that this project wants to evaluate the decoding accuracy of a negative binomial population, it is legitimate to throw away trials where certain parameter combinations can't generate an adequate amount of sample variance over the sample mean for the population response to be "negative binomial". The missing data is then imputed using the KNN imputation package for the purpose of plotting the decoding bias colormap without missing values. After the data imputation, the decoding bias is plotted (decoding variance is not included in here as all variances are close to 0).

A visual inspection tells us that there might be some but not significant increases in the decoding bias as we increase the gamma parameters. A two-sample Kolmogorov-Smirnov test is performed over 50 samples of decoding bias, each corresponding to a given gamma parameter, for every two columns. There are no p-value smaller than 0.05 and we fail to reject the null hypothesis that 50 samples of decoding bias are coming from the same distribution. In other words, the decoding bias is not changed with respect to the variations in the gamma parameters, confirming the theoretical derivation that the decoding accuracy is not affected by the gain signal G.

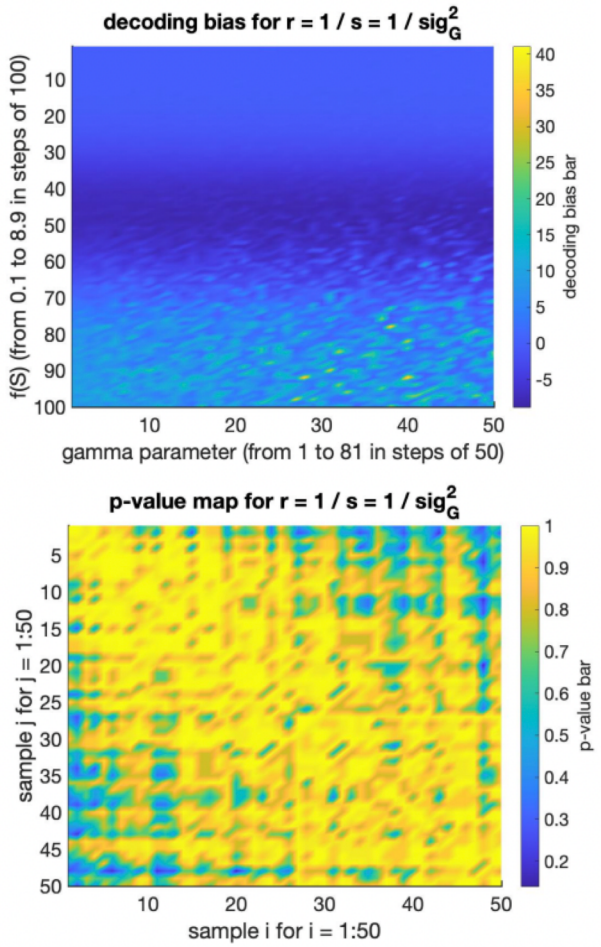

Figure 3: Decoding Metrics for $[r, s] = [\frac{1}{\sigma_G^2}, \sigma_G^2]$.

$$[r, s] = [\sigma_G^2, \sigma_G^2]$$

Rewrite r as β −1 , α as s(f(S)Δt) we get:

$$P(N = n) = \frac{\Gamma(n + \beta^{-1})}{\Gamma(N + 1)\,\Gamma(\beta^{-1})} \left(1 + \alpha\right)^{-\beta^{-1}} \left(\frac{\alpha}{\alpha + 1}\right)^{n}$$

$$= \frac{\Gamma(n+\beta^{-1})}{\Gamma(N+1)\,\Gamma(\beta^{-1})} \left(1+s(f(S)\Delta t)\right)^{-\beta^{-1}} \left(\frac{s(f(S)\Delta t)}{s(f(S)\Delta t)+1}\right)^{n},$$

where

$$N \sim \text{Neg-Bin}(\beta, sf(S)\Delta t).$$

Then by Piegorsch (1990) we have ˆ (sf(S)Δt) = ¯n for $\nabla_{f(S)\Delta t\,1} = 0$ (Piegorsch, 1990). The decoding accuracy is affected by the scale parameter s.

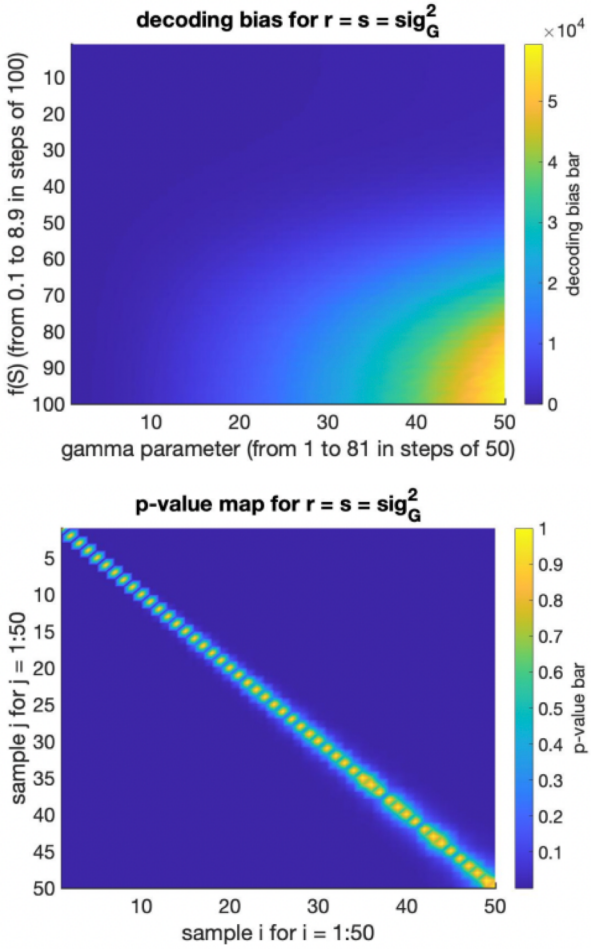

Figure 4: Decoding Metrics for $[r, s] = [\sigma_G^2, \sigma_G^2]$.

With the same procedure and the Kolmogorov-Smirnov test described above, we can conclude that we reject the null hypothesis that 50 samples of decoding bias comes from the same distribution, i. e. the gain signal does affect the decoding accuracy for the f(S)Δt given the assumption that [r, s] = [σ ² G, σ² G], as opposed to [r, s] = [$\frac{1}{\sigma\ 2\ G}$, σ² G] where the decoding accuracy is not affected by the gamma parameters.

Conclusion

This project has shown that a poisson-gamma encoding model from Goris et al (2014) creates excess variability in the population responses, which is reflected in the growth of Fano Factor with varying gamma values and stimulus values. A further decoding of population response through both mathematical derivations and numerical simulations has shown that the decoding accuracy of population responses is not affected by the gain signal represented as convergent inputs to the visual cortex along with the sensory inputs, given the assumptions that [r, s] = [$\frac{1}{\sigma\ 2\ G}$, σ² G]. The decoding accuracy is affected when we assume that [r, s] = [σ ² G, σ² G]. The stability or instability of the decoding process given the gain signal might convey important functional roles of the convergent inputs during visual processings. From a Bayesian perspective, the conjugate prior of the poisson distribution (poisson population) is the gamma distribution (gain signal), highlighting the possibility for bayesian inference (Fink, 1997). Further research should evaluate the optimality or suboptimality of the gain signal as it is transmitted to the visual pathway along with the sensory stimuli.

References

Anscombe, F.J. (1950). Sampling Theory of The Negative Binomial and Logarithmic Series Distributions. Biometrika.

Fink, D. (1997). A Compendium of Conjugate Priors. JOHN D. COOK Consulting.

Gelman. A., Carlin, J.B., Stern, H. S., Dunson, D. B., Vehari A, Rubin, D.B. (2013). Bayesian Data Analysis. CRC Press.

Goris, L.T, Movshon, J.A, Simoncelli, E.P. (2014). Partitioning Neural Variability. Nature Neuroscience. Piegorsch, W.W. (1990). Maximum Likelihood Estimation For The Negative Binomial Dispersion Parameter. Biometrics.

Taouali, W., Benvenuti, G., Wallisch, P., Chavane, C., Perrinet, L.U. (2016). Testing The Odds Of Inherent vs. Observed Overdispersion In Neural Spike Counts. Journal of Neurophysiology.

Neurobiological Relationship Between Addictive, Affective, and Anxiety Disorders

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Addictive, affective, and anxiety disorders are quite common, and they frequently coexist. Thus, approximately half of the individuals who experience a mental illness will also experience a substance use disorder, and vice versa (Kelly et al., 2013). It has been hypothesized that one disorder fosters another. For example, underlying biological tendencies towards sensitization of certain neuronal pathways promote drug dependencies or mood disorders (Quello et al., 2005). However, there is a paucity of research on understanding the complexity of the relationship between these disorders. In that context, the field of biological psychiatry is currently in a stage of rapid expansion, uncovering new information through studies that are demonstrating the evolving relationship between these disorders. This is exciting because such discoveries can pave the way for new pharmacological treatments that can target shared pathways implicated in these disorders. The complexity of the relationship between these disorders has limited the amount of research available due to confounding factors of the respective conditions that make allocating specific substrates and/or drugs to specific abnormalities in neurochemistry difficult (Ross et al., 2012), but due to the nature of this rapidly evolving field of study, new information is constantly emerging around the world.

The aim of this paper is to provide a synthesis of literature on the nature of the relationship between addiction, affective, and anxiety disorders. This paper covers a wide range of topics within this field, including specific neural substrates, clinical diagnoses, and pharmacological treatments of addiction, affective disorders, and anxiety disorders. To accomplish that, this paper has been divided into seven main sections. The *first section* is the introduction, which provides background to the field, proposes the main research questions, and why they are important. The *second section* is dedicated to addiction disorders, specifically the main executive control neuronal network that is involved in substance abuse disorders. It also highlights the psychosocial factors that contribute to addiction, and discusses the current pharmacological treatments of addiction, including what is lacking within the scope of the prevention of relapse in addiction disorder patients. The *third section* focuses on the main drugs of abuse that impact in the shared neural correlates with some affective and anxiety disorders. The *fourth section* is committed to Major Depressive Disorder (MDD), encompassing a clinical overview of the disorder, the pathophysiology of depression, and the substances that share the same neural substrates. The *fifth section* discusses Bipolar I and II disorders (BD I and BD II), how they are clinically, neuroanatomically, and biochemically similar and different, along with the drugs of abuse whose effects are analogous. The *sixth section* covers Generalized Anxiety Disorder (GAD), incorporating clinical diagnostic criteria, pathophysiology, and the shared neural substrates between withdrawal states of certain drugs of abuse and anxiety disorders. The *seventh* and final section summarizes the presented findings and determines the answer to the main question of the paper: what is the nature of the relationship between addictive, affective, and anxiety disorders?

I. Introduction

Comorbidity of Disorders

There is a nuanced comorbidity between substance abuse and affective as well as anxiety disorders. It is often the case that patients with mood and anxiety disorders self-medicate with certain drugs of abuse to alleviate negative symptoms of their disorders. The strong motivation for self-sedation in individuals with manic disorders, including BD I and II, and anxiety disorders, like generalized anxiety disorder (GAD), drive those afflicted mostly towards abuse of depressants, like alcohol and barbiturates as well as opiates like morphine and heroin (Quello et al., 2005) in order to relieve perceived stress and hysteria. MDD also has a very high association with alcohol abuse, with a prevalence of 20.5% and in comparison to non-addicts, alcohol dependent individuals are 3.7 times more likely to develop and eventually be diagnosed with a major depressive disorder (nami.org). There is also overwhelmingly positive and significant evidence to support the fact that in at least 75% of cases, anxiety disorders predate the development of substance abuse disorders (Kushner et al., 2008). *The National Alliiance on Mental Illness* (NAMI) has concluded that patients with mental disorders are more prone to substance abuse, in 37% of cases related to alcohol and to drugs of abuse, including cocaine, amphetamines, cannabis, and opiates in 53% of cases (nami.org). The high degree of comorbid associations is also indicative of the shared involvement of specific brain regions that will be further explored in this paper. Circuits in the brain that mediate reward and executive functioning are implicated in addiction, affective, and anxiety disorders, and share multiple different types of neurotransmitter systems, which demonstrates the complexity of the relationship between them.

Main Research Questions

The nature of the relationship between addictive, affective, and anxiety disorders is a point of interest in the field of biological psychiatry because of the ramifications of its unknown directionality. If neural substrate abnormalities in addiction make those affected vulnerable to affective disorders and anxiety, should pharmacological addiction treatment be modified to preventatively target neuronal pathways compromised in affective and anxiety disorders? Similarly, if neural substrate abnormalities in affective disorders and anxiety predispose those individuals to the development of addiction, should prophylactic approaches normally utilized for affective and anxiety disorders be prescribed to treat addiction?

In this context it is critical to note that environmental and genetic factors play a large role in the development of any neurological disorder. For example, a genetic predisposition to cocaine addiction via heritable epigenetic modulation of gene expression (Pierce et al., 2018) will not make such an individual addicted to cocaine without cocaine availability and/or a malfunctioning executive control neural network.

If this relationship is in fact causal, it poses several interesting scenarios: 1) Does addiction always make an individual vulnerable to affective and anxiety disorders, or do affective and anxiety disorders predispose the individual to an increased risk of the development of addiction to any drug of abuse that substantially reduces the pathways necessary for resilience against such a development? 2) Is the nature of the relationship bidirectional, meaning a combination of both contribute to the acquisition of an addictive, affective, and/or anxiety disorder? This paper will address all of these inquiries in the context of specific affective and anxiety disorders and the specific drugs of abuse that correspond respectively due to shared neural circuitry abnormalities.

II. Addiction

Neural Substrates

Addiction is a chronic and relapsing disorder characterized by compulsive drug seeking, loss of control and continuation of abuse

despite harmful consequences (Carr, 2021). Addictive disorders implicate the neural circuitry systems for reward, motivation, and executive control where maladaptive neurochemical alterations of pathways ultimately leads to dependence. The general finding across all addictive behaviors and disorders is the impaired descending connections from the prefrontal cortex (Carr, 2021). Cortico-striato-pallido-thalamo-cortical loops are central to motivated behaviors (Potenza et al., 2011); these loops provide connectivity to other parts of the brain that are critical to executive functioning, emotion, motivation, and reward. These include the amygdala which provides mood information, the hippocampus which provides contextual memory information, and the insula which provides information on introspective processing (Potenza et al., 2011).

Many drugs of abuse increase dopamine transmission in the nucleus accumbens within the limbic forebrain, thus mimicking the effect of natural rewards like food, albeit more potently. The drugs gradually replace natural rewards as the dominant motivator for behavior, resulting in a devaluation of natural rewards. Chronic activation of dopamine and glutamate receptors via substance abuse leads to a "rewiring" of the nucleus accumbens that dedicates circuits to drug seeking and taking at the expense of homeostatic regulation (Carr, 2021). During withdrawal, vulnerability to relapse increases as cravings are no longer substantiated, and stress induced cravings become the significant adversary to relapse prevention. Intact executive control, in order to recruit prefrontal cortex function, is necessary to exert inhibitory control over impulses and cravings but is impaired in addicts. Whether this abnormality is a pre-existing condition, making individuals more vulnerable to becoming an addict, or whether the chronic use of the drug results in the individual acquiring degraded executive functioning remains unanswered.

Hypoconnectivity within these regions is associated with an increased risk of addiction, which may weaken goal directed decision making (Ersche et al., 2020). These additive neurochemical adaptations escalate as dependent drug use becomes chronic; ultimately resulting in the development of addiction. In contrast, a strong resilience to addiction was associated with hyperconnectivity in these regions as well as the lateral prefrontal cortex, medial caudate nucleus, the supplementary motor area, the superior medial prefrontal cortex, and the putamen (Ersche et al. 2020), all of which are involved in top-down inhibitory control as well as the regulation of habits. Compromised executive and regulatory function is the biological basis of addiction in any context, including addiction to drugs of abuse. This suggests a predisposing vulnerability to the causation of addiction, specifically corresponding with impaired goal directed actions and regulatory behavior.

Psychosocial factors

There are many types of factors that contribute to the development of addiction, but some of the most notable, due to the recent influx of information, are epigenetic factors, specifically the psychosocial factors. Heritability estimates for addictions range between 0.4 with hallucinogens to 0.7 with cocaine (Ducci et al., 2012), but environmental confounders and exposure interactions contribute significantly to the expression of these inherited genes. Environmental and social factors can lead to methylation of DNA, making transcription factor binding more difficult, or acetylation, which makes those genes easier to access (Carr, 2021). Genes involved in vulnerability to addictions include those that are substance specific and those that act on common pathways in addiction (Ducci et al., 2012). Psychosocial factors can have an impact on multiple different neuronal pathways, many of which are also implicated in addiction.

Social situations can be both reinforcing and undermining environments for individuals, especially adolescents. There is current debate as to how adolescent risk-taking and orientation towards short term goals should be perceived.

Drug abuse is sustained despite known harm in the pursuit of socialization (Gopiram et al., 2014), which is an adaptation because maturation, independence, and connection with new peers is evolutionarily critical for succession to adult life and is facilitated by such practices (Cousijn et al., 2017). The onset of puberty marks a shift in brain plasticity and a surge of social attunement, corresponding to "the need to adapt to and harmonize with the social environment" (Cousijn et al., 2017), which also increases the socially reinforcing value of substance use as adolescence progresses. In the adolescent brain, rewarding social outcomes are akin to substance use because of their similar neural correlates and the relative lack of connectivity between the prefrontal cortex and reward systems (Kalivas et al., 2005). The association strengthens between substance use and rewarding social outcomes as puberty progresses, which can ultimately lead to escalation of use, especially in abnormal neural circuitry. With transition to adulthood in healthy individuals, this neurochemical adaptation to risk-taking and reward-seeking behavior evolves to a prioritization of long term goals and improved emotional control (Cousijn et al., 2017) and subsequent social devaluation of substance use. Findings indicated that those who are resilient to social defeats, stresses, and are not socially isolated have an acquired hyperconnectivity between the corticostriatal pathways that are otherwise implicated in addiction disorders (Ersche et al., 2020). This is evidence for the hypothesis that epigenetic modifications can have a tremendous impact on executive functioning, reward, and motivation behavior. Similarly, lower positions in social hierarchies are also correlated with increased vulnerability to addiction (Morgan et al., 2002). These are both avenues for addiction treatment.

Treatments

The treatment of addiction disorders is nuanced and complicated, and can be divided into three main stages: initial detoxification, recovery, and relapse prevention which is crucial (Potenza et al., 2011). In initial detoxification, the goal is to achieve maintained abstinence and reduce immediate withdrawal symptoms. The recovery phase is dedicated to the development of sustained motivation and the avoidance of relapse, by learning strategies for craving tolerance and forming new patterns of behavior that replace drug induced reinforcement with an alternative. The relapse prevention phase occurs after a long period of abstinence from the drug of abuse and long term strategies for maintaining abstinence (Potenza et al., 2011). The biggest issue for recovery from a substance abuse disorder is the debilitating period of withdrawal, compounded by the extremely high risk of relapse due to the hypoconnectivity in regions dedicated to executive functioning (Um et al., 2019), as mentioned previously. Pharmacological treatments often involve choosing a replacement substance that has a similar mode of action on the implicated biological substrate, while having a slower and more sustained effect. As a result, sustained motivation is required and often difficult to achieve, resulting in relapse more often than not. The target of pharmacological treatment is dependent on the type of addiction the individual needs to recover from, but generally targets motivational neurocircuitry and multiple neurotransmitter systems. The most effective pharmacological treatments are those which target specific neural substrates of the drug the afflicted individual is addicted to. This of course requires a comprehensive understanding of the precise neurochemical mechanisms and pathways each drug of abuse implicates and how.

III. Drugs of Abuse

Many different types of substances are addictive, but the most pervasive and threatening are drugs of abuse. These include cocaine, amphetamines, nicotine, cannabis, opiates/opioids, and alcohol because of their neural correlates with substrates implicated in affective, addictive, and anxiety disorders.

Cocaine

Cocaine is a psychostimulant drug of abuse with a sharp onset and offset that blocks the presynaptic dopamine transporter (DAT), thus increasing the concentration of dopamine, specifically in the nucleus accumbens shell (Volkow et al., 1992). This neural circuitry in the ventral striatum and basal ganglia is a critical part of reward circuitry, implicated both in addicts and some psychological disorders. In addition to this ramification of its use, chronic cocaine abuse also generates dissociated grey matter reduction in the dorsal prefrontal cortex (DLPFC) and the anterior cingulate cortex (ACC), which in turn affects executive control and emotional regulation, respectively (Connolly et al., 2013). In a comparative study with neurotypical individuals, a reduction in prefrontal cortex cerebral blood volume and heightened vasoconstriction was found in those addicted to cocaine (Congwu et al., 2020) as well.

Amphetamines

Amphetamines are synthetic stimulants that increase dopamine release in the brain, resulting in an excess of dopamine across the nucleus accumbens shell and extracellularly (Cadet et al., 2014). Since both amphetamines and cocaine are within the class of psychostimulants, they share some of the neural mechanisms and neurotransmitter receptors that they target, but differ in their involvement in external regions outside this reward pathway. Amphetamine also decreases the vesicular monoamine transporter (VMAT) immunoreactivity and blocks long-term synaptic depression in the ventral tegmental area (Jones et al., 2000). Methamphetamine, a subclass of amphetamines, inhibits DAT-mediated dopamine clearance more efficiently than amphetamines, resulting in more extracellular dopamine (Goodwin et al., 2009), accompanied by a greater and far more dangerous level of addiction. Methamphetamine also increases white matter hyperintensities (Bae et al., 2006) as well as the size of lateral ventricles (Jeong et al., 2013), and it also upregulates brain hypermetabolism (Volkow et al., 2001).

Nicotine

Nicotine is within the class of stimulant drugs, but also acts as an anxiolytic, causing a sedative effect. As a stimulant, nicotine excites dopaminergic neurons in the ventral tegmental area, which facilitates increased proliferation of dopamine within this area, thus implicating reward cognition in the same way in which it is affected in addiction (Russell et al., 2019). As an anxiolytic, it decreases the serotonergic 5-HT1A formation and release of dopamine in the hippocampus in the medial temporal lobe (Breslau, 1991). The consequences of these effects include instability of emotions, since the hippocampus also resides in the medial temporal lobe with the amygdala, which controls emotional memory recall and regulation.

Cannabis

Cannabis is a depressant, stimulant, and hallucinogen with two main receptors, CB1 and CB2, within the endocannabinoid system. The psychoactive component of cannabis Δ-9-tetrahydrocannabinol (THC) mimics the naturally produced cannabinoid neurotransmitter anandamide, and once it attaches to CB1 receptors specifically, it overwhelms the system and acts as an agonist for glutamate and antagonist for Gamma-aminobutyric acid (GABA) release (Lucatch et al., 2018). These receptors are located in the cerebellum, hippocampus, caudate nucleus, post medial ventral tegmental area, nucleus accumbens, anterior cingulate cortex, and amygdala, thus resulting in euphoria, heightened and diminished anxiety, paranoia, sedation, etc. (Lucatch et al., 2018). In addition to these implications, cannabis also downregulates brain derived neurotrophic factor (BDNF) thus decreasing neurogenesis (Crippa et al., 2009).

Opiates/Opioids

Opiates are organically produced narcotics that bind to endogenous opioid μ receptors in the brain. Opioids, the synthetic counterpart to opiates, also have a high affinity for and bind to μ receptors in the brain regions including the post medial ventral tegmental area, ultimately resulting in a reduction of inhibitory synaptic transmission mediated by GABAergic interneurons (Kosten et al., 2002). Without exposure to either, the brain normally produces opiate-like chemicals that bind to μ receptors, convert adenosine triphosphate (ATP) into cyclic adenosine monophosphate (cAMP) which then triggers the release of many type of neurotransmitters, including noradrenaline (NA) that maintains normal levels of alertness. When an opioid drug binds to the receptors instead, the enzyme that converts ATP into cAMP is inhibited and less cAMP, and consequently less NA is produced, resulting in fatigue. Overtime with chronic exposure the neuron tries to make up for the inhibitory impact of the opioid and creates excess of cAMP and NA (Kosten et al., 2002). After periods of abstinence, since the receptor is no longer blocked by the opioid, the excess NA results in clinical symptoms of withdrawal, including anxiety, which is evidence for similar neural correlates between each.

Alcohol

Alcohol is an incredibly addictive substance, which blocks certain neurochemical signals from each other, thus hindering the communication between critical regions and pathways of the brain. An increase in the consumption of alcohol releases excess dopamine from the 5-HT1A pathways in the nucleus accumbens, a critical aspect of the reward and motivation pathway that is similarly afflicted in some affective disorders (Suh et al., 2018). There are neurochemical adaptations to chronic alcohol exposure in corticostriatal motivation pathways, including lower hippocampal volume (Agartz, 1999). The system most severely altered by alcohol use disorder (AUD) is the hypothalamic–pituitary–adrenal (HPA) axis, resulting in abnormality within the central stress response system and the release of the stress hormone cortisol (Suh et al., 2018).

IV. Major Depressive Disorder

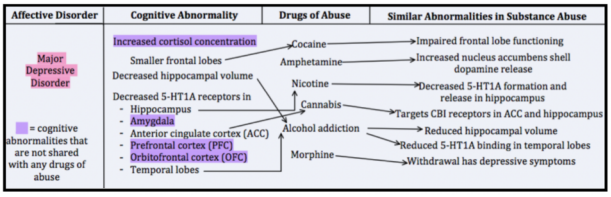

Figure 1: Main shared neural substrates between Major Depressive Disorder (MDD) and certain drugs of abuse

Clinical Overview

Major depressive disorder (MDD) is a very common affective disorder, affecting about 7.1% of adults in the US with a lifetime risk of 10% (NIH. 2021). It is characterized by depressed mood and/or loss of interest associated with cognitive and somatic disturbances, which cause significant functional impairment. Clinical diagnoses define a depressive episode, which is characteristic of MDD, and involves a "period of at least two weeks when a person experiences a depressed mood or loss of interest or pleasure in daily activities, and has a majority of specified symptoms, such as problems with sleep, eating, energy, concentration, or self-worth" (NIH, 2021). MDD impacts the dynamic connectivity among neural substrates that regulate mood and stress responses, resulting in psychological affects of sadness and distress.

Pathophysiology

Major depressive disorder is attributed to structural as well and neurochemical abnormalities. Smaller frontal lobe volumes, increased ventricle to brain ratios, and smaller hippocampal volume are all associated with the disorder (Carr, 2021). The dynamic connectivity

among neuroanatomical structures related to mood and stress response are affected as well - the neural pathways between the limbic structures of the amygdala, hippocampus, and nucleus accumbens and the paralimbic cortical areas that include the subgenual anterior cingulate cortex (area 25) and the ventro medial prefrontal cortex are all afflicted (Maletic et al., 2007). Disrupted connectivity between these regions results in an impaired feedback regulation loop between them, creating a hypoactive executive functioning network and hyperactive limbic system. This hyperactivity stimulates the hypothalamus, leading to neuroendocrine dysregulation that is ultimately manifested in depression symptoms (Maletic et al., 2007). Additionally, damage to the hippocampus leads to an exacerbated endocrine response. As a result of decreased entry of glucose and decreasing reuptake of glutamate in the compromised hippocampal neurons, there is a high influx of Ca^{2+} ions, which if left untreated can become neurotoxic (Carr, 2021). The impaired receptors control the major negative feedback loop of the HPA stress axis, and their impairment increases the concentration of cortisol, which is the main stress hormone (Matsuo et al., 2019) in patients with MDD.

Cocaine and Depression

A significant cognitive abnormality in major depressive disorder is structurally smaller frontal lobes with reduced circuitry, a finding which is also detected in the brain imaging scans of cocaine addicts (Volkow et al., 1992). In that study, cocaine abusers had significantly lower ($P< 0.05$) metabolic activity in 16 of the 21 left frontal regions and 8 of the 21 right frontal regions, including the right dorso medial and lateral prefrontal cortex, and the left dorso medial, lateral, and motor prefrontal cortex as well as the right anterior parietal cortex. (Volkow et al., 1992). These deficits in the neurochemical pathways persisted after 3–4 months of detoxification and correlated to both the amount and duration of cocaine use, suggesting sustained effects of cocaine abuse on the brain (Volkow et al., 1992). Depressed individuals and cocaine addicts share similarities in their clinical presentations as well, including anhedonia and anergia (NIH, 2021), a fact that substantiates the claim that depression can be a precursor to cocaine abuse, and vice versa.

Amphetamines and Depression

Amphetamine addiction also increases nucleus accumbens shell dopamine release, and in chronic exposure conditions, leads to an overcompensation of dopamine release that has severe effects in withdrawal states (Goodwin et al., 2009). While this general finding is not specific to depression episodes in individuals with MDD, it is characteristic of milder forms of depression as well as more severe depressive states. In a basic model of addiction, the drug of abuse is the preferred binding molecule despite the presence of natural neurotransmitters, in this case dopamine, but amphetamine differs in the mechanism of its proliferation in the brain. Amphetamine has a similar structure to the dopamine transporter, so it is reuptaken and released in the terminator button of these serotonergic neurons (Carr, 2021). However once it infiltrates the post synaptic vesicle, it causes a reverse transport, releasing dopamine back into the extracellular space (Goodwin et al., 2009). In withdrawal states with prolonged abstinence from amphetamines, depressive symptoms including acute sadness and generally low mood are observed, indicating a correlational relationship between the two; although it is not yet known which predisposes the individual to the other.

Nicotine and Depression

Decreased hippocampal 5-HT1A serotonin binding is a symptom of nicotine dependence and addiction, as chronic nicotine treatment produces a selective decrease in the concentration of 5-HT in the hippocampus in rat studies, providing evidence for a neuroadaptation

to nicotine that is also present in MDD (Naomi et al., 1991). Evidence from a synthesis of literature suggests that "depression sensitizes patients to the adverse effects of stressful stimuli, and that this can be relieved by drugs that stimulate dopamine release in the forebrain" (Balfour et al., 2000). This mechanism contributes to the increased craving to smoke during the withdrawal states, because of conditioning that correlates smoking with stress relief. As a result of chronic nicotine exposure, the serotonergic pathway is altered to release less serotonin in the hippocampus, an area critical to emotional regulation that is severely afflicted in depression. But to smokers and depressed individuals, nicotine use still retains antidepressant properties while actually producing depressogenic 5-HT formation and release (Balfour et al., 2000), and are thus relatively protected from these consequences until nicotine abstinence is put into effect.

Cannabis and Depression

Cannabis, or specifically THC, targets CB1 receptors in the anterior cingulate cortex and the hippocampus, which influences dopamine transmission and produces a high via an upregulation of GABA neurotransmitters and can ultimately lead to long term reduction and/or inhibition of neurotransmitters associated with dopamine (Lucatch et al., 2018) that is also characteristic of major depressive disorder. While cannabis is often self-administered in recreational contexts, long term exposure changes the prioritization of natural neurotransmitters in the brain, specifically in the postero medial ventral tegmental area (Gobbi et al., 2019). The amplitude and duration of cannabinoids is greater than naturally produced dopamine, and overtime this repeated teaching signal results in overlearning within the CB1 receptors (Carr, 2021). The resulting reduction of dopamine in the brain makes an individual addicted to cannabis susceptible to the development of depression.

Alcohol and Depression

Decreased hippocampal volume and decreased binding of 5-HT1A serotonergic receptors are also a notable feature of MDD as well as alcohol addiction (Agartz, 1999). In chronic alcoholism, the reduction in hippocampal volume is proportional to the reduction of brain volume (Agartz, 1999), indicating that hippocampal atrophy is a substantial outcome of alcohol abuse. This finding is correlated with general decreased neuronal density in depressed individuals in the hippocampus, thus implicating a key region and neural circuitry system involved in learning and memory that explained documented cognitive deficits that characterize major depressive disorder. The shared neural substrate abnormalities between the two substantiates the claim that alcohol abuse can be a predisposition for the development of MDD, and depression can make an individual more vulnerable to self medicate and become addicted to alcohol.

Morphine and Depression

Additionally morphine, which does not share neural correlates with depression, does have symptomatic similarities in its withdrawal (Nalepa et al., 2007), but there is little difference in it's withdrawal state symptoms of acute sadness in comparison with other drugs of abuse. As a result, it's interactions with systems implicated in depression do not support the hypothesis that morphine addiction predisposes an individual to the development of a depressive disorder, or that a depressive disorder makes an individual vulnerable to the development of morphine or any other opiate addiction.

V. Bipolar I and II Disorders

Clinical Overview

Bipolar I (BD I) is a combination of manic episodes and symptoms, accompanied by depressive episodes, and features of each can occur in mixed states. Manic episodes are characteristic of BD I, which is prevalent in about

1% of the population with early onset in late adolescence (NIH. 2021). Bipolar II (BD II) is a pattern of depressive and hypomanic episodes, no manic episodes, and usually begins as a depressive episode (Langan et al., 2009). BD II has a 15% comorbidity rating with depression, is much more common than BD I, and has a later onset of symptoms (NIH, 2021), often because it is clinically misdiagnosed as depressive episodes, due to a relative lack of rule out criteria between the two disorders.

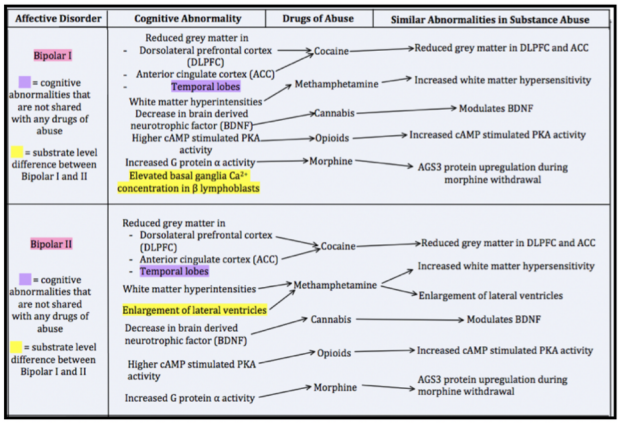

Figure 2: Main shared neural substrates between Bipolar I and II (BD I and BD II) and certain drugs of abuse

Pathophysiology

Bipolar I and II differ in level of severity clinically and neurochemically in two instances - basal ganglia calcification and ventricle size. Elevated basal ganglia calcium concentration in beta lymphoblasts is found in BD I patients, resulting in altered dopaminergic transmission in this region that yields positive psychotic symptoms (Emamghoreishi et al., 1997). A proposed mechanism from this case study discusses the premise of basal ganglia neuronal degeneration, which disrupts frontosubortical circuit pathways to generate the positive symptoms. The patient had "idiopathic basal ganglia calcification and psychosis with drug induced neurological abnormalities" (Johnson et al., 2013), resulting in a decrease in dopaminergic transmission; a possible process for disruption of circuits that cause mania in BD I afflicted individuals. Abnormal dopamine signaling in the basal ganglia is associated with development of certain substance abuse disorders (Ersche et al., 2020), as evidence gathered indicated that addiction develops through atypical engagement for striatal cortices from ventral to dorsal spheres, thus implicating habit formation through associative learning.

In Bipolar II disorder, there are no calcification points but there is neurochemical data on the enlargement of lateral ventricles in these patients (Lai et al., 2017). The subcortical volumetric abnormality is associated with negative symptoms of bipolar disorder but is not present in BD II patients with psychosis (Pearlson et al., 1984), which suggests that it is a morphological feature that could be developed through external means, like drugs of abuse. Other than these differences, BD I and II share similar neural correlates including reduced grey matter in the DLPFC, ACC, temporal lobes, white matter hypersensitivity, decreased BDNF, higher cAMP stimulated PKA activity, and increased G protein α activity.

Cocaine and Bipolar Disorders

A reduction in grey matter in the dorsolateral prefrontal cortex (DLPFC) and the anterior cingulate cortex (ACC) is a co-occuring abnormality in both bipolar disorders and chronic cocaine addiction. A study assessing the grey matter volume variation in different lengths of abstinence from cocaine abuse in addicts found that longer periods of abuse significantly increased the amount of grey matter reduction in these regions (Connolly et al., 2013), forming a proportional relationship between severe cocaine addiction and loss of grey matter. The DLPFC and the ACC are neural substrates also implicated in BD, substantiating the claim that cocaine addiction could make an individual more vulnerable to development of BD, and vice versa.

Cannabis and Bipolar Disorders

A decrease in brain derived neurotrophic

factor (BDNF) is also present in bipolar disorders and has evidence of being facilitated by cannabis in addicted individuals (Crippa et al., 2009). Tetrahydrocannabinol (THC) induces BDNF mRNA transcription via stimulation of the CB1 receptors, activating the microtubule-associated protein kinase or extracellular regulated kinase (MAPK/ERK) pathway (Crippa et al., 2009); this pathway has a critical role in regulating cell fate and exhibiting control over the initiation and termination of neurogenesis. Chronic exposure to cannabis is associated with downregulation and desensitization of CB1 receptors overtime that ultimately lead to the suppression of BDNF release that results in a reduction of neurogenesis. This ramification of cannabis abuse is incredibly dangerous, as a reduction in the process of forming new neurons can inhibit connectivity and result in the heightened proliferation of neurotransmitters transmission to overcompensate for the loss of synaptic networks (Crippa et al., 2009), thus producing psychomimetic effects characteristic of bipolar disorder.

Opioids and Bipolar Disorders

Opioids increase cAMP stimulated PKA activity, a neurochemical abnormality also observed in bipolar disorders. Persistent exposure to opioids in an addicted individual inhibits adenylyl cyclase (AC) activity and reduces the production of cAMP, and therefore, decreases the activity of PKA (Langan et al., 2009). With chronic abuse of opioids, AC activity returns toward basal levels, resulting in increased cAMP levels and PKA activity (Matsuo et al., 2019) also present in bipolar disorder. In a study on a post mortem bipolar disorder patient suicide victim cited no significant differences in PKA subunit mRNA levels in the temporal and frontal cortices of BD patients compared with controls, but there were significantly increased μ receptors density (Gabilondo et al., 1995). These findings infer that the elevated PKA immunolabeling was due to a posttranscriptional mechanism, that can be a result of opioid addiction or another mechanism that alters PKA activity, like a co-occurring bipolar disorder, rather than changes in regulation of gene transcription.

Morphine and Bipolar Disorders

Opioids like morphine have even more specific effects on neural circuitry, including elevation of g protein α activity that is also present in bipolar disorders. It also targets endogenous μ receptors, but morphine abuse specifically promotes the upregulation of AGS_3 protein to compensate after chronic exposure in withdrawal states (Nalepa et al., 2007). This elevation in concentration has been associated with alterations in intracellular signaling, which are reflected in the genes that encode G proteins (Nalepa et al., 2007), and presented as manic as well as hypomanic symptoms characteristic of BD I and II.

Amphetamines and Bipolar Disorders

Methamphetamines particularly are attributed to white matter hyperintensities, a common finding in the brain scans of bipolar disorder patients. Methamphetamine abuse produces microstructural abnormalities in white matter that are significantly correlated with psychiatric symptoms characteristic of bipolar disorder (Bae et al., 2006). Methamphetamine is also correlated with the enlargement of right lateral ventricles in BD II (Jeong et al., 2003), a morphometric abnormality that provides insight into a potential biological substrate predisposition that would make an individual with this consequent structural abnormality more vulnerable to morphine addiction, but there is very little evidence outside of a case study that supports this data.

VI. Generalized Anxiety Disorder

Clinical Overview

Generalized anxiety disorder (GAD) is the uncontrollable and diffused anxiety or worry that is excessive or unrealistic in relation to objective life circumstances, which persist for one month or longer. Approximately 30% of the US population suffer from anxiety disorders (NIH, 2021), it's major components including inappropriate worry and dread, decreased concentration, and an autonomic arousal of the sympathetic nervous system, or "fight or flight" mechanism (Costall et al., 2002). The comorbidity between anxiety and affective disorders is extremely high; approximately 60% of depressed individuals are also anxious (NIH, 2021). The theorized self medication pathway, where drug abuse leads to anxiety, and the substance-induced anxiety pathway, both lead to a "mutual maintenance pattern" of development in which one disorder perpetuates the other (Smith et al., 2008), although it is still unclear the direction in which this relationship. Anxiety disorders are included as a part of this paper studying interactions with drug abuse because the withdrawal symptoms for most drugs often include induced anxiety, which can further develop into GAD. Drug withdrawal induced anxiety is not the same thing as generalized anxiety, but they do share many neural substrate abnormalities.

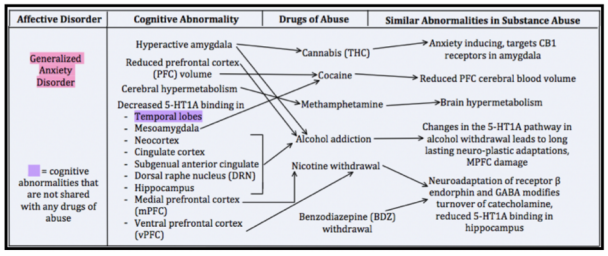

Figure 3: Main shared neural substrates between Generalized Anxiety Disorder (GAD) and certain drugs of abuse

Pathophysiology

There are many different substrates neurochemically implicated in anxiety disorders. The amygdala and the medial prefrontal cortex are at the center of the disorder, processing threat perception and regulating emotions, respectively (Carr, 2021). In anxiety disorder, the amygdala is hyperactive and the mPFC is hypoactive, contributing to a disproportionate fear response to stimuli, a perception of emotionally ambiguous stimuli as negative threats, and an inability to recruit executive functioning to distract the individual from said fear (Duval et al., 2015). As a result, the fear extinction mechanism is implicated so that afflicted individuals can not extinguish a conditioned fear response and continue to be fearful of repeated threats, instead of getting used to them (Suh et al., 2018). Glucocorticoids typically cross the blood brain barrier, exert negative feedback at the hypothalamic-pituitary-adrenal (HPA) axis, consequently reducing the corticotropin-releasing hormone (CRH) and cortisol stress hormone secretion (Suh et al., 2018), but in neurochemically atypical individuals with anxiety disorders this inhibitory mechanism is impaired, resulting in an extensive release of stress hormones. These hormones extend to axonal serotonin projections as well (Carr, 2021), contributing to decreased serotonergic 5-HT1A receptors in the temporal lobes, meso amygdala, neocortex, cingulate cortex, subgenual anterior cingulate cortex, dorsal raphe nucleus (DRN), hippocampus, medial and ventral prefrontal cortices.

Cocaine and Anxiety

Reduced cerebral volume in the prefrontal cortex is an abnormality indicative of anxiety disorders and is also associated with chronic cocaine addiction. Cocaine decreases the amount of cerebral blood volume in the prefrontal cortex, including the mPFC (Du et al., 2020), which is fundamental in addiction because of its involvement in higher-order executive function like self-control, salience attribution and awareness. Less blood flow in this region, as a result, means less executive functioning, and an increased vulnerability to the development of addiction because of the shared neuronal substrate pathway prerequisite making it easier for

addiction to develop.

Alcohol and Anxiety

Alcohol addiction and withdrawal ultimately results in a decrease of serotonergic 5-HT1A binding in the cortex, anterior cingulate, dorsal raphe nucleus, and hippocampus, which are all important substrates that contribute to the range of anxiety disorders (Cadet et al., 2014). The reduction in serotonergic binding in these regions that control emotional regulation and memory yields unsubstantiated anxiety to emotionally ambiguous stimuli, maintaining a consistent fear state that is detrimental to the afflicted individual. Additionally, alcohol abuse impairs the mPFC functioning as found in a mouse study, resulting in an impaired fear extinction mechanism that does not allow for conditioning towards a perceived threat (Suh et al., 2018). The alcohol adaptation to the corticostriatal motivation pathways directly implicated the HPA axis, leading to long term exposure to an influx of stress hormones like cortisol, increasing and enhancing anxiety (Suh et al., 2018). This unfortunately is a negatively reinforcing practice, as anxious individuals typically self-medicate with alcohol to dispel anxieties, especially in social situations, but this prolonged exposure to alcohol actually makes an already anxious individual more anxious overtime, supporting the claim that alcohol use and anxiety disorders have a shared biological basis.

Methamphetamine and Anxiety

Methamphetamine abuse results in cerebral hypermetabolism, which is also a shared neural substrate in anxiety disorders. In a study done on detoxified methamphetamine abusers, whole brain metabolism was 14% higher in methamphetamine addicts than their neurotypical counterparts (Volkow et al., 2001), especially in the cortex which is a substantially implicated neural substrate in anxiety. Higher metabolism in this region is thus indicative of shared correlates between hypermetabolism in neural circuits and anxiety, and affirms the hypothesis that similar neural correlates in methamphetamine addiction and anxiety can make an individual susceptible to either.

Cannabis and Anxiety

A hyperactive amygdala is the hallmark of anxiety disorders, which is also attributed to alcohol and cannabis withdrawal after severe and chronic exposure. The tetrahydrocannabinol component of cannabis binds to CB1 receptors in the amygdala, leading to neuroplastic adaptations over time (Koob et al., 2009) to compensate for the overwhelming concentration in the endocannabinoid system. Anxiety-related stress induced corticotropin releasing factor causes enduring sensitization of norepinephrine systems in the amygdala and nucleus accumbens (Bhattacharyya et al., 2017), which results in a continued stress response, consistently hyperactivating the amygdala to process fear in emotionally ambiguous situations. This shared implication of neural substrates supports the claim that both can act as predispositions to the development of the other.

Nicotine and Benzodiazepine Withdrawal and Anxiety

Nicotine withdrawal and benzodiazepine withdrawal are also substantial anxiety inducing mechanisms, and as a result share neural substrate abnormality with anxiety disorders. Nicotine binds to acetylcholine receptor subtypes that have unique expression patterns in the central nervous system (Russell et al., 2019), they are widely distributed, and they participate in cholinergic signaling in nearly every neural area including the hippocampus, medial and ventral prefrontal cortex. Additionally, β-endorphins inhibit the release of gamma aminobutyric acid (GABA) (Petraglia et al., 1986), which is the primary inhibitory neurotransmitter of the brain that leads to excess accumulation of dopamine. Withdrawal conditions like those of nicotine and

benzodiazepine results in a net reduction in 5-HT1A binding that is characteristic of anxiety disorders.

VII. Concluding Thoughts

What does all of the presented data mean in regards to the initial question i.e., is substance abuse a risk factor for development of affective and/or anxiety disorders and/or are some anxiety and affective disorders risk factors for developing substance addiction? The above narrative would suggest that both scenarios are plausible. However, while the present data shows varying degrees of association between the two, there is no direct evidence for causality, only mild and strong associations in some cases.

There are several theories that address the nature of the complex comorbidity between substance use disorders and affective and anxiety disorders. One theory proposes that pathological effects of an affective disorder or substance use disorder perpetuate each other, driven primarily by self-medication tactics often employed by afflicted individuals who frequently resort to drugs or alcohol to cope with negative affects (Quello et al., 2005). Conversely, chronic substance abuse can also "unmask" bipolar or other mood disorders by triggering an increase in symptom severity from a subclinical to a clinically significant level (Quello et al., 2005). This could be an explanation for cases in which genetically vulnerable individuals observe exacerbated pathophysiological symptoms after consistent drug use. The kindling hypothesis suggests that the more sensitized neurons become during addiction, the less it takes to disrupt them (Quello et al., 2005). Alcohol and cocaine sensitize neurons, which could contribute to the progression of an individual's drug use from occasional to frequent.

Other theories stipulate that the comorbidity of these disorders may be maintained by the fact that many substance abuse symptoms can mimic those of affective and anxiety disorders, which indicates an underlying similar neural circuitry. Some drug abuse symptoms mirror depression and mania, as severe alcohol and psychostimulant intoxication can produce symptoms of BD II mania, while substance withdrawal usually produces symptoms of anxiety and depression (Ross et al., 2012). Chronic use of stimulants like cocaine and amphetamine can produce symptoms typical of bipolar disorders, like increased energy and paranoia, while withdrawal from these yield anhedonia and depressed mood. Continued abuse of depressants like alcohol, benzodiazepines, and opiates can lead to anhedonia and poor concentration while withdrawal states can result in anxiety and agitation (Kelly et al., 2013). All of these shared symptoms are an indication of mutual maintenance patterns explained throughout this investigation.

The nature of the relationship between addictive, affective, and anxiety disorders is characterized by a complex comorbidity, the extent of which has yet to be fully discovered. Understanding the nature and directionality of this relationship within neural substrates of specific substance abuse and cognitive disorders will be crucial in the pursuit of targeted and more effective treatment methods for addictive, affective, and anxiety disorders.

References

Bae, S. C. (n.d.). (2006). *Increased white matter hyperintensities in male methamphetamine abusers*. Drug and alcohol dependence. https://pubmed.ncbi.nlm.nih.gov/16005161/.

Balfour, D. J., & Ridley, D. L. (2000). The effects of nicotine on neural pathways implicated in depression: a factor in nicotine addiction?. *Pharmacology, biochemistry, and behavior*, *66*(1), 79–85.

Bhattacharyya, S., Egerton, A., Kim, E., Rosso, L., Barros, D. R., Hammers, A., ... McGuire, P. (2017, November 3). *Acute induction of anxiety in humans by delta-9-tetrahydrocannabinol related to amygdalar cannabinoid-1 (CB1) receptors*. Nature News.

Bowers, M. S. (2010, September). *Activators of G-protein signaling 3: a drug addiction molecular gateway*. Behavioural pharmacology. https://www.ncbi.nlm.nih.gov/pmc/articles/

Cadet, J. L., Bisagno, V., & Milroy, C. M. (2014). Neuropathology of Substance Use Disorders. *Acta Neuropathol, 127*, 91-107. doi:10.1007/s00401-013-1221-7

Carr, K., Dr. (Presenter). (2021). Drug Abuse and Addiction. Lecture presented at New York University, New York City, NY, United States.

Carr, K., Dr. (Presenter). (2021). Affective Disorders. Lecture presented at New York University, New York City, NY, United States.

Carr, K., Dr. (Presenter). (2021). Anxiety. Lecture presented at New York University, New York City, NY, United States.

Connolly, C. G., Bell, R. P., Foxe, J. J., & Garavan, H. (2013). *Dissociated grey matter changes with prolonged addiction and extended abstinence in cocaine users*. PloS one.

Costall, B., Kelly, M. E., Naylor, R. J., & Onaivi, E. S. (2002, November 7). *The actions of nicotine and cocaine in a mouse model of anxiety*. Pharmacology Biochemistry and Behavior.

Cousijn, J., Luijten, M., & Ewing, S. W. F. (2017, November 20). *Adolescent resilience to addiction: a social plasticity hypothesis*. The Lancet Child & Adolescent Health.

Crippa, J. A., Zuardi, A. W., Martín-Santos, R., Bhattacharyya, S., Atakan, Z., McGuire, P., & Fusar-Poli, P. (2009). Cannabis and anxiety: a critical review of the evidence. *Human psychopharmacology*, *24*(7), 515–523. https://doi.org/10.1002/hup.1048

Ducci, F., & Goldman, D. (2012). The genetic basis of addictive disorders. *The Psychiatric clinics of North America*, *35*(2), 495–519. https://doi.org/10.1016/j.psc.2012.03.010

Du, C., Volkow, N. D., You, J., Park, K., Allen, C. P., Koob, G. F., & Pan, Y. (2020, July 28). *Chronic cocaine exposure causes reduced blood flow and vasoconstriction in mouse prefrontal cortex in vivo*. Nature News.

Duval, E. R., Javanbakht, A., & Liberzon, I. (2015, January 23). *Neural circuits in anxiety and stress disorders: a focused review*. Therapeutics and clinical risk management.

Emamghoreishi, M., Schlichter, L., Li, P. P., Parikh, S., Sen, J., Kamble, A., & Warsh, J. J. (1997). High intracellular calcium concentrations in transformed lymphoblasts from subjects with bipolar I disorder. *The American journal of psychiatry*, *154*(7), 976–982. https://doi.org/10.1176/ajp.154.7.976

Ersche, K. D., Meng, C., Ziauddeen, H., Stochl, J., Williams, G. B., Bullmore, E. T., & Robbins, T. W. (2020, June 30). *Brain networks underlying vulnerability and resilience to drug addiction*. PNAS.

Gabilondo AM, et al. (1995) Increased density of mu-opioid receptors in the postmortem brain of suicide victims. *Brain Res.* https://pubmed.ncbi.nlm.nih.gov/7552322/

Gobbi, G., Atkin, T., Zytynski, T., Wang, S., Askari, S., Boruff, J., Ware, M., Marmorstein, N., Cipriani, A., Dendukuri, N., & Mayo, N. (2019). Association of Cannabis Use in Adolescence and Risk of Depression, Anxiety, and Suicidality in Young Adulthood: A Systematic Review and Meta-analysis. *JAMA psychiatry*, *76*(4), 426–434. https://doi.org/10.1001/jamapsychiatry.2018.4500

Goodwin, J. S., Larson, G. A., Swant, J., Sen, N., Javitch, J. A., Zahniser, N. R., ... Khoshbouei, H. (2009, January 30). *Amphetamine and methamphetamine differentially affect dopamine transporters in vitro and in vivo*. The Journal of biological chemistry.

Gopiram, P., & Kishore, M. T. (2014). Psychosocial Attributes of Substance Abuse Among Adolescents and Young Adults: A Comparative Study of Users and Non-users. *Indian journal of psychological medicine*, *36*(1), 58–61. https://doi.org/10.4103/0253-7176.127252

Home: NAMI: National Alliance on Mental Illness. NAMI. (n.d.). https://www.nami.org/Home.

Ingrid Agartz, M. D. (1999, April 1). *Hippocampal Volume in Patients With Alcohol Dependence*. Archives of General Psychiatry.

Jeong, H. S., Lee, S., Yoon, S., Jung, J. J., Cho, H. B., Kim, B. N., Ma, J., Ko, E., Im, J. J., Ban, S., Renshaw, P. F., & Lyoo, I. K. (2013). Morphometric abnormalities of the lateral ventricles in methamphetamine-dependent subjects. *Drug and alcohol dependence*, *131*(3), 222–229. https://doi.org/10.1016/j.drugalcdep.2013.05.009

Johnson, J. M., Legesse, B., Camprodon, J. A., Murray, E., Price, B. H., Psychiatry, F. the D. of, ... Andres, D. S. (2013, January 1). *The Clinical Significance of Bilateral Basal Ganglia Calcification Presenting With Mania and Delusions*. The Journal of Neuropsychiatry and Clinical Neurosciences.

Jones, S., Kornblum, J. L., & Kauer, J. A. (2000, August 1). *Amphetamine blocks long-term synaptic depression in the ventral tegmental area*. The Journal of neuroscience : the official journal of the Society for Neuroscience. https://www.ncbi.nlm.nih.gov/pubmed/10908593.

Kalivas, P. W., Volkow, N. D., Edwards, A. C., Blanco, C., Hansen, S., Kowalczyk, W. J., ... Miller, J. M. (2005, August 1). *The Neural Basis of Addiction: A Pathology of Motivation and Choice*. American Journal of Psychiatry.

Kelly TM, Daley DC. Integrated Treatment of Substance Use and Psychiatric Disorders. *Soc Work Public Health.* 2013; 28(0):388-406. doi:10.1080/19371918.2013.774673

Koob G. F. (2009). Brain stress systems in the amygdala and addiction. *Brain research*, *1293*, 61–75. https://doi.org/10.1016/j.brainres.2009.03.038

Kosten, T. R., & George, T. P. (2002, July). *The neurobiology of opioid dependence: implications for treatment*. Science & practice perspectives.

Kushner MG, Krueger R, Frye B, Peterson J. Epidemiological perspectives on co-occurring anxiety disorder and substance use disorder. In: Stewart SH, Conrod PJ, editors. *Anxiety and Substance Use Disorders: The Vicious Cycle of Comorbidity.* New York: Springer; 2008. pp. 3–17

Lai, J., Lu, Q., Huang, T., Hu, S., & Xu, Y. (2017, July 19). *Convulsive syncope related to a small dose of quetiapine in an adolesc: NDT*. Neuropsychiatric Disease and Treatment. Langan, C., &

McDonald, C. (2009). Neurobiological Trait Abnormalities in Bipolar Disorder. *Molecular Psychiatry*, *14*, 833-846.

Lucatch, A. M., Coles, A. S., Hill, K. P., & George, T. P. (2018). Cannabis and Mood Disorders. *Current addiction reports*, *5*(3), 336–345. https://doi.org/10.1007/s40429-018-0214-y

Maletic, V., Robinson, M., Oakes, T., Iyengar, S., Ball, S. G., & Russell, J. (2007, December). *Neurobiology of depression: an integrated view of key findings*. International journal of clinical practice.

Matsuo, K., et al. (2019). Distinctive Neuroanatomical Substrates for Depression in Bipolar Disorder versus Major Depressive Disorder. *Cerebral cortex (New York, N.Y. : 1991)*, *29*(1), 202–214. https://doi.org/10.1093/cercor/bhx319

Morgan, D., Grant, K. A., Gage, H. D., Mach, R. H., Kaplan, J. R., Prioleau, O., Nader, S. H., Buchheimer, N., Ehrenkaufer, R. L., & Nader, M. A. (2002). Social dominance in monkeys: dopamine D2 receptors and cocaine self-administration. *Nature neuroscience*, *5*(2), 169–174. https://doi.org/10.1038/nn798

Nalepa, I., Zelek-Molik, A., Bielawski, A., Roman, A., & Vetulani, J. (n.d.). *Does the presence of morphine counteract adaptive changes in expression of G-protein alpha subunits mRNA induced by chronic morphine treatment?* Pharmacological reports : PR. https://pubmed.ncbi.nlm.nih.gov/17377204/.

Naomi Breslau, P. D. (1991, December 1). *Nicotine Dependence, Major Depression, and Anxiety in Young Adults*. Archives of General Psychiatry.

Pearlson, G. D., Garbacz, D. J., Tompkins, R. H., Ahn, H. S., Gutterman, D. F., Veroff, A. E., & DePaulo, J. R. (1984). Clinical correlates of lateral ventricular enlargement in bipolar affective disorder. *The American journal of psychiatry*, *141*(2), 253–256. https://doi.org/10.1176/ajp.141.2.253

Petraglia , F. (n.d.). *gamma-Aminobutyric acid inhibits beta-endorphin secretion from the anterior pituitary but not the neuro intermediate lobe in the rat*. Endocrinology. https://pubmed.ncbi.nlm.nih.gov/2934244/.

Pierce, R. C., Fant, B., Swinford-Jackson, S. E., Heller, E. A., Berrettini, W. H., & Wimmer, M. E. (2018, February 5). *Environmental, genetic and epigenetic contributions to cocaine addiction*. Nature News.

Potenza, M. N., Sofuoglu, M., Carroll, K. M., & Rounsaville, B. J. (2011, February 23). *Neuroscience of Behavioral and Pharmacological Treatments for Addictions*. Neuron.

Quello, S. B., Brady, K. T., & Sonne, S. C. (2005). Mood disorders and substance use disorder: a complex comorbidity. *Science & practice perspectives*, *3*(1), 13–21. https://doi.org/10.1151/spp053113

Ross, S., Dr., & Peselow, E., Dr. (2012). Co-Occurring Psychotic and Addictive Disorders: Neurobiology and Diagnosis. *Clinical Neuropharmacology*, *35*(5), 235-243.)

Russell, T. I., & Robinson, M. (2019). Effects of nicotine exposure and anxiety on motivation for reward and gambling-like cues under reward uncertainty. *Behavioral neuroscience*, *133*(4), 361–377. https://doi.org/10.1037/bne0000311

Smith, J. P., & Book, S. W. (2008). Anxiety and Substance Use Disorders: A Review. *The Psychiatric times*, *25*(10), 19–23.

Suh, J., & Ressler, K. J. (2018). Common Biological Mechanisms of Alcohol Use Disorder and Post-Traumatic Stress Disorder. *Alcohol research : current reviews*, *39*(2), 131– 145. Um, M., Whitt, Z. T., Revilla, R., Hunton, T., & Cyders, M. A. (2019). Shared Neural Correlates Underlying Addictive Disorders and Negative Urgency. *Brain sciences*, *9*(2), 36. https://doi.org/10.3390/brainsci9020036

U.S. Department of Health and Human Services. (n.d.). *Major Depression*. National Institute of Mental Health.

Volkow, N. D., Hitzemann, R., Wang, G. J., Fowler, J. S., Wolf, A. P., Dewey, S. L., & Handlesman, L. (1992). Long-term frontal brain metabolic changes in cocaine abusers. *Synapse (New York, N.Y.)*, *11*(3), 184–190. https://doi.org/10.1002/syn.890110303

Volkow, N. D., Chang, L., Wang, G.-J., Fowler, J. S., Franceschi, D., Sedler, M. J., ... Jernigan, T. L. (2001, March 1). *Higher Cortical and Lower Subcortical Metabolism in Detoxified Methamphetamine Abusers*. American Journal of Psychiatry.

Neurobiological Abnormalities in and Genetic Predispositions to Psychopathy

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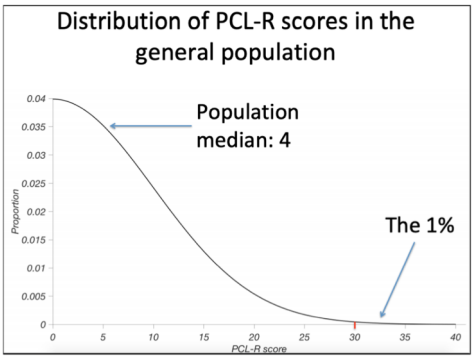
Introduction

Psychopathy is a complex topic that many scientists seek to understand, from both a psycho-biological and socio-historical perspective. When looking at this subject from a psychological perspective, scientists define psychopathy as an extreme form of antisocial behavior (Tiihonen, 2019). There are numerous studies looking into the neurobiology and genetic composition of psychopaths, and these have all, for the most part, yielded similar results. This research paper will examine whether abnormalities in genetics and neurobiology are determinants for psychopathy. The first section of this paper will focus on the neurobiological aspects, specifically looking at neurotransmitters, hormone imbalances, matter composition, and underdevelopment of certain areas of the brain. The second section will focus on genetics, specifically whether abnormal gene expression and heredity have profound implications on psychopathy.

`Before looking at the neurology and genetics behind psychopathy, it is essential to understand how it is measured and identified. From a psychological perspective, the standard central features of the condition include the following: lack of emotional affect, no empathy or guilt, reckless behavior, indifference to the infliction of suffering on others and lack of morality, etc. (Glenn, 2008). The most common way to diagnose an individual with psychopathy is through clinical interviews paired with scales or checklists, the Hare Psychopathy Checklist (PCL-R) being one of the most commonly used (Wallisch, 2020). The PCL-R is composed of 20 items and is on a 3-point scale. This particular measure is based on an ordinal scale and is then interpreted as a number over 30 (out of 40) establishing an individual as highly psychopathic, and individuals over 20 as having substantial psychopathic traits and tendencies (Wallisch, 2020). While some may assume that individuals with psychopathy compose an extremely small percentage of the population, it is actually far more prevalent. The epidemiological reality is that psychopathy is a spectrum. Approximately 1% of the general population is diagnosed with psychopathy, and approximately 10-30% of incarcerated criminals are (Glenn, 2008).



Graph 1: Data from Pascal Wallisch, 2020: PCL-R Score Distribution

Hare Psychopathy Checklist (PCL-R)	
Factor 1	**Facet 1: Interpersonal** 1. Glibness & superficial charm 2. Grandiose sense of self-worth 4. Pathological lying 5. conning/manipulation
	Facet 2: Affective 6. Lack of remorse or guilt 7. Shallow affect 8. callousness/lack of empathy 16. Failure to accept responsibility for own actions
Factor 2	**Facet 3: Lifestyle** 3. Need for stimulation/proneness to boredom 9. Parasitic lifestyle 13. Lack of realistic, long-term goals 14. Impulsivity 15. Irresponsibility
	Facet 4: Antisocial 10. Poor behavioral controls 12. Early behavioral problems 18. Juvenile delinquency 19. Revocation of conditional release 20. Criminal versatility

Table 1: Pascal Wallisch, 2020: Hare Psychopathy Checklist (PCL-R) Self-Made Replication

Part 1: Neurobiological Abnormalities

Part 1.A: Neurotransmitters & Interaction with Hormones

Having outlined the diagnostic criteria and scaling of psychopathy, this paper will now focus on the neurobiological abnormalities that have been preliminarily identified as contributing factors to its development. This paper will divide this discussion into parts, focusing on aspects that have both individual and cumulative influence. Firstly, many papers discuss the role of neurotransmitters and the endocrine system (chemical messenger system). Various studies have shown that psychopathy is associated with an increased ratio of dopamine metabolite HVA and serotonin metabolite 5-HIAA (Glenn, 2008). This increase could suggest an impairment in the regulation of dopamine activity, which in turn affects aggression and impulse control. As with most other manifestations in an organism, an abnormality in one part of the body will most likely not remain isolated, affecting other bodily components and processes. Neurotransmitters, for instance, have a functional relationship with hormones, specifically cortisol and testosterone. For instance, serotonin neurotransmission connects to the hypothalamic-pituitary-adrenal (HPA) axis, so more activity in serotonin receptors in the hypothalamus will augment the production of cortisol in the adrenal cortex. It has also been hypothesized that serotonin may affect testosterone levels (Glenn, 2008). In neurotypical cases, this doesn't have much of an impact, but in cases where there are any abnormalities, an increase in testosterone levels can increase violence and aggression (Glenn, 2008). In a previous instance, the research mentioned that an increase in serotonin, leading to an imbalance in levels of cortisol, has an impact on aggression. Conversely, a decrease in serotonin combined with augmented production of testosterone can have the same result (Glenn, 2008). Additionally, raised levels of testosterone have been linked to dominance-seeking behaviors (Glenn, 2008), which in conjunction with an increase in aggression can lead to violent acts. Such hypothesized neurobiological abnormalities could be proven in the fact that a large percentage of incarcerated individuals (who have been found to have said abnormalities) score highly on the PCL-R checklist and thus are considered to have psychopathy (Wallisch, 2020).

Part 1.B: Hormones

It is important to understand the function of the hormones in question due to the fact that they function against each other to maintain a homeostatic relationship. The role of cortisol is to gather the body's resources and provide energy when the organism is having a stress response, and it has been proposed that it is involved in fear response, sensitivity to punishment, and withdrawal (Glenn, 2008). Testosterone, on the other hand, is a product of the hypothalamic-pituitary-gonadal (HPG) axis and relates to reward versus punishment processing and fear reduction (Gao, 2009). Having understood their origins and functions, analyzing how they work together and against each other is important for understanding why an imbalance would cause a major disruption in behavioral patterns. Cortisol ultimately suppresses HPG axis activity and diminishes testosterone production, inhibiting its effects. Conversely, testosterone suppresses HPA axis activity, which therefore decreases cortisol production. Taking this information into consideration, it becomes clear that if there were to be an increase or decrease that was caused either by natural dysregulation or by a dysfunction in the serotonin receptors affecting the hypothalamus, this would cause not only one, but both of these hormones to have detrimental effects when it comes to phenotypical attributions (such as aggression, poor decision-making, and poor valuation). Looking at these theories in a real-life application, experiments have found that lower levels of cortisol are linked to young adult males whose history of violence has a negative correlation with psychopathy, although further

investigation needs to take place given the small sample size of this research (Glenn, 2008). It has therefore been hypothesized that men scoring high in psychopathy have less cortisol reactivity in social stress testing than males who score lower on the scale. In regard to testosterone levels, research has found that they are positively correlated to scores in facets 3 and 4 of the PCL-R, yet these results need to be looked over again to ensure that there are no confounds with comorbid substance abuse and psychosis (Glenn, 2008). Additionally, girls with conduct disorders and adolescent boys with externalizing behaviors, as well as criminals have shown to have high levels of testosterone and have been diagnosed with varying severities of antisocial behaviors and disorders (Gao, 2009).

Part 1.C: A Brief Note on Testosterone

Looking at testosterone specifically, a common trend when examining research on psychopathy happens to be that most participants in these studies are males. When it comes to analyzing patterns of behavior, brain abnormalities and genetic predispositions, finding a group of individuals who have been deemed psychopaths based on the PCL-R scale (done so by professionals in the field) is hard to come by. Thus, most of the people composing this sample come from incarcerated individuals (majority male). The reason for this has to do with "successful" and "unsuccessful" psychopaths (Gao, 2009). The former refers to individuals with highly psychopathic traits who are able to more or less blend in with the rest of society, while the latter refers to individuals who are apprehended and then charged with crimes as a result of their externalized aggression. The main consequence of psychopathy that leads to arrests and convictions is violence, so most psychopaths who participate in these studies have been charged with assaults or murders, and on average, males are the ones who predominantly commit these crimes (Wallisch, 2020). That being said, there are other studies in this field that involve and incorporate female participants who have also demonstrated higher levels of testosterone in their systems versus the average female, so although testosterone is usually associated with men (and then tested in mostly males) in regard to psychopathy, there has been a fair amount of research that delves into the hormonal components for women as well.

Part 1.D: Neuroimaging

Moving beyond neurotransmitters and hormones, one of the most researched areas in the neuroscience of psychopathy are abnormalities in brain structures and problems with connectivity (Glenn, 2008). Through brain imaging studies, there have been several findings that suggest psychopaths have impaired prefrontal cortexes. In one study, there was a significant reduction in prefrontal gray matter volume in patients diagnosed with an antisocial personality disorder, who scored highly on the PCL-R (Gao, 2009). In another, it was found that there is a substantial prefrontal grey matter reduction in psychopathic criminals' vis-a-vis neurotypical control participants. Based on investigations such as these, it has been suggested that underdevelopment or reduced volume of the prefrontal grey matter results in impaired moral judgment, faulty decision-making, and problems with emotional regulation (Gao, 2009). There have also been a plethora of other studies focusing on different regions of the brain, such as the hippocampus and the amygdala. Psychopaths have shown volume reductions in the bilateral amygdala, and some other individuals who score highly on the PCL-R show reduced posterior hippocampal volumes (Gao, 2009). Deficits in these regions are thought to be associated with emotional deficits such as superficial charm, pathological lying, lack of regret or remorse, and shallow affect (all of which are traits seen in some form or another on the PCL-R checklist). One study of particular note looking at moral decision-making demonstrated that psychopaths have less amygdala activity during contemplation of moral dilemmas. Those who scored particularly high on

the interpersonal facet of the PCL-R showed reduced functioning in the amygdala, medial prefrontal cortex, angular gyrus, and the posterior cingulate, all of which are critical in moral decision-making (Gao, 2009).

Part 2: Abnormal Gene Expression and Heredity

This section will speak about abnormal gene expression in the neurobiological roots of psychopathy, predominantly using a source from a study conducted by Tiihonen et. al, in Finland. It will also focus on a study carried out by Blonigen et. al., in the United States when looking at the role of heredity in psychopathy.

Part 2.A: Abnormal Gene Expression

Using Tiihonen's *Neurobiological Roots of Psychopathy*, this paper will now discuss irregularities in the genes of psychopaths in comparison to two other groups, one of non-offending substance abusers and the other of healthy individuals with no criminal record or history of substance abuse as a control measure. The participants' information is seen in Table 2, taken directly from the research paper. The experiment set out to confirm whether or not severe antisocial and criminal behavior has a substantial genetic component and accomplished this task by generating and characterizing iPSC lines from each participant of each group, differentiating between cortical neurons expressing markers of glutamatergic and GABAergic neurons and to astrocytes (Tiihonen, 2019). They found that "the expression of *ZNF132* [(associated with developmental and malignant disorders, expressed in the cerebellum which has been shown to be highly correlated with severe antisocial behaviors as well)] in neurons and *RPL10P9* in both neurons and astrocytes is markedly abnormal among habitually violent offenders" (Tiihonen, 2019). Beyond this, changes in protein levels demonstrated a sensitivity to insulin and glucose metabolism which has been found as a predictor for violent crimes. Finally, they found that their data suggested that dysfunction of the opioid system contributes to the behavioral manifestations of psychopathy (Tiihonen, 2019).

	Age	Diagnosis	Number of committed homicides	Number of violent crimes	PCL-R score
Subject 1	30	Antisocial personality disorder, ADHD, alcohol dependence, benzodiazepine abuse, multiple sclerosis, asthma	2	19	37.0
Subject 2	42	Antisocial personality disorder, alcohol dependence	3	4	Not available
Subject 3	49	Antisocial personality disorder, alcohol dependence	2	11	30.0
Subject 4	43	Antisocial personality disorder, alcohol dependence, polysubstance dependence	2	7	33.7
Subject 5	30	Antisocial personality disorder, alcohol dependence, opioid dependence, cannabis dependence, benzodiazepine dependence, amphetamine dependence	3	8	36.0
Subject 6	47	Antisocial personality disorder, borderline personality disorder, paranoid personality disorder, alcohol dependence, polysubstance dependence, amphetamine dependence, hepatitis C	2	9	37.0
Subject 7	38	Alcohol dependence	0	0	2
Subject 8	25	Alcohol dependence	0	0	3
Subject 9	31	Alcohol dependence, cannabis dependence, bulimia	0	0	11
Subject 10	44	None	0	0	3
Subject 11	28	None	0	0	2
Subject 12	28	None	0	0	1
Subject 13	47	None	0	0	3
Subject 14	26	None	0	0	2
Subject 15	51	None	0	0	1

All individuals were males. Subjects 1–6 are violent offenders, 7–9 are individuals with substance abuse but without criminal behavior, and 10–15 are healthy controls. The biological fathers of Subject 1, Subject 3, and Subject 5 had prison convictions due to violent and nonviolent crimes. None of the biological mothers had been convicted into prison

PCL-R psychopathy checklist revised

Table 2: Table from Tiihonen et. al, 2019: Experiment demonstrating each participant, their diagnostic condition, their crimes or lack thereof and their PCL-R scores determined by clinical psychiatrists

Part 2.B: Heredity

Now looking at the question of heredity, using Blonigen's *Psychopathic Personality Traits: Heritability and Genetic Overlap with Internalizing and Externalizing Psychopathology*, it has been hypothesized that there is some genetic influence in determining the extreme antisocial behaviors that are indicators of psychopathy. This study involved a moderate sample size of 626 pairs of adolescent (17 years of age) male and female twins whose traits were qualified and quantified using the Multidimensional Personality Questionnaire (MPQ). The experimenters examined the features such as antisocial behavior and delinquency against environmental and genetic structures that could possibly contribute to their manifestations (Blonigen, 2005). They categorized psychopathy into two groups, making a distinction between primary and secondary psychopathy which they determined could differ in both the genetic disposition and environmental influences (Blonigen, 2005). Cross-referencing PCL-R checklist items with the results from the MPQ, the experimenters looked at monozygotic and dizygotic twins to estimate what portion of

Table 3: Table from Blonigen et. al, 2005: Experiment demonstrating heritability estimates in male, female and combined twins for fearless dominance, impulsive antisociality, and internal/external symptoms

Heritability estimates from best-fitting univariate twin models for psychopathic personality traits, internalizing, and externalizing

	Men		Women		Combined	
	Heritability	(95% CI)	Heritability	(95% CI)	Heritability	(95% CI)
Fearless Dominance	0.46	(0.32–0.57)	0.45	(0.34–0.54)	0.45	(0.37–0.53)
Impulsive Antisociality	0.51	(0.39–0.62)	0.48	(0.37–0.57)	0.49	(0.41–0.56)
Internalizing	0.49	(0.35–0.60)	0.31	(0.18–0.43)	0.36	(0.27–0.45)
Externalizing	0.76	(0.70–0.81)	0.68	(0.60–0.74)	0.73	(0.68–0.77)

n=1252 individuals from 626 twin pairs, some with missing data. CI, confidence interval. The heritability for all variables are due to additive genetic effects. Combined refers to models in which the unstandardized parameters were equated across the genders.

Table 4: Data from Blonigen et. al, 2005: Experiment demonstrating genetic correlations for male, female and combined twins between psychopathic personality traits and internalizing/externalizing behaviors

Genetic correlations between psychopathic personality traits, internalizing, and externalizing

	Internalizing						Externalizing					
	Men		Women		Combined		Men		Women		Combined	
	r_g	(95% CI)	r_g	(95% CI)	r_g	(95% CI)	r_g	(95% CI)	r_g	(95% CI)	r_g	(95% CI)
Fearless Dominance	**−0.40**	(−0.64 to −0.16)	**−0.39**	(−0.63 to −0.15)	**−0.40**	(−0.57 to −0.22)	***0.56***	(0.17 to 0.56)	0.01	(−0.16 to 0.19)	**0.16**	(0.04 to 0.29)
Impulsive Antisociality	−0.03	(−0.27 to 0.20)	***0.38***	(0.14 to 0.64)	**0.20**	(0.03 to 0.37)	**0.45**	(0.28 to 0.60)	**0.52**	(0.37 to 0.65)	**0.49**	(0.38 to 0.59)

r_g, Genetic correlation; CI, confidence interval.

Correlations in bold are significant (confidence intervals do not include zero). Correlations in bold and italicized are significant and significantly different for men and women. Combined refers to models in which the unstandardized parameters were equated across the genders.

phenotypic covariance between the traits came about as a result of genetic and or environmental factors. They found that for both males and females, twin correlations for fearless dominance, impulsive antisociality, both on internalized and externalized levels, were generally consistent with an additive model of inheritance where monozygotic correlations were twice that of dizygotic correlations (Blonigen, 2005). Breaking this down further, males demonstrated greater average levels of psychopathic traits and externalizing symptoms than women for both categories (fearless dominance and impulsive antisociality), whereas women exhibited higher average levels of internalized symptoms (Blonigen, 2005). They estimated that approximately half of the various traits in question were due to genetic effects, stating that heritability was moderate for internalized symptoms and strong for externalized symptoms (Blonigen, 2005). These findings match other studies that have been conducted, firstly when it comes to men's apparent external manifestations versus women's internal manifestations, and secondly when it comes to determining that genetics play a significant role in psychopathy, of which heritability accounting for approximately half of the total variance in men and women (Blonigen, 2005). Looking at these findings, it seems as if both factors they were exploring have an impact on psychopathy: genetics, which is what was predominantly in question, and environment, which is difficult to prove but many scientists (especially psychologists) believe to be a key determinant.

Conclusion

Looking at this research, a few problems present themselves in understanding psychopathy. While, as previously established, it is determined on a scale, there is also a question in where one should draw the line to determine whether an individual in question is a danger to themselves and to society. For instance, what is the distinction between 25 and 30 on the PCL-R scale? These figures are somewhat arbitrary and need further evaluation to come up with a feasible way of monitoring and treating psychopathy in the future. Additionally, there is far more research conducted on the neurobiology of psychopathy in comparison to that of genetics. The reason for this is most likely the fact that heritability is extremely difficult to decipher when it comes to psychological phenomena. This obstacle is also found in other areas of research like psychotic disorders such as bipolar disorder and schizophrenia. Additionally, distinguishing between genetic effects and environmental effects on psychopathy is difficult, namely because the two are often overlapping. If an individual is raised in a violent household without structure

and positive redirection of malicious or dangerous behavior throughout their childhood, they will inevitably be more likely to solidify their psychopathic traits. Thus, while the genetic component may be a significant contributing factor, it is difficult to say whether it outweighs or has the same weight as epigenetics. Much of the genetic components of psychological disorders and traits are unknown as of yet, so these areas need further investigation and evaluation.

Ultimately, when looking at the roles of genetics and neurobiology of psychopathy, it is clear that there is a significant need for further investigation in the field, a fact that most scientists who have written on the matter agree with. The implications of understanding psychopathy are numerous, but among the most relevant, would be our ability to help individuals who suffer from it and to ensure the safety of communities with reduced crime rates and convictions should psychopaths have a form of treatment and rehabilitation. The question of whether people are violent by nature or as a result of an unconducive environment has and will certainly continue to be a debate for sociologists as much as it is for biologists, psychologists, and neuroscientists. However, if or when there is more concrete evidence showing that psychopathy can be a result of direct heritability rather than solely abnormalities in gene expression and neurological components like underdevelopment or maldevelopment of brain structures as well as destabilized increases and decreases of hormones and neurotransmitters, there could be a larger problem of pondering whether or not there is actually a treatment for such a condition.

References

Blonigen, D. M., Hicks, B. M., Krueger, R. F., Patrick, C. J., & Iacono, W. G. (2005). Psychopathic personality traits: heritability and genetic overlap with internalizing and externalizing psychopathology. *Psychological medicine*, *35*(5), 637–648. https://doi.org/10.1017/s0033291704004180

Gao, Y., Glenn, A. L., Schug, R. A., Yang, Y., & Raine, A. (2009). The neurobiology of psychopathy: a neurodevelopmental perspective. *Canadian journal of psychiatry. Revue canadienne de psychiatrie*, *54*(12), 813–823. https://doi.org/10.1177/070674370905401204

Glenn, A. L., & Raine, A. (2008). The neurobiology of psychopathy. *The Psychiatric clinics of North America*, *31*(3), 463–vii. https://doi.org/10.1016/j.psc.2008.03.004

Tiihonen, J., Koskuvi, M., Lähteenvuo, M. *et al.* Neurobiological roots of psychopathy. *Mol Psychiatry* (2019). https://doi.org/10.1038/s41380-019-0488-z

Wallisch, P. (Presenter). (2020, February). *Cognitive Neuroscience - Psychopathy*. Lecture presented at New York University, New York City, NY, United States.

Figures: Graphs and Tables

Blonigen, D. M. (n.d.). Twin Correlations for Psychopathic Personality Traits, Internalizing, and Externalizing [Table]. In *Psychopathic Personality Traits: Heritability and Genetic Overlap with Internalizing and Externalizing Psychopathology* (p. 13).

Blonigen, D. M. (n.d.). Genetic Correlations between Psychopathic Personality Traits, Internalizing, and Externalizing [Table]. In *Psychopathic Personality Traits: Heritability and Genetic Overlap with Internalizing and Externalizing Psychopathology* (p. 16).

Tiihonen, J., Koskuvi, M., Lähteenvuo, M., Virtanen, P., Ojansuu, I., Vaurio, O., Gao, Y., Hyötyläinen, I., Puttonen, K. A., Repo-Tiihonen, E., Paunio, T., Rautiainen, M.-R., Tyni, S., Koistinaho, J., & Lehtonen, S. (n.d.). Clinical and Sociodemographic Characteristics of Study Subjects [Table]. In *Neurobiological Roots of Psychopathy*.

Wallisch, P. (2020). *Distribution of PCL-R Scores in the General Population* [Graph].

Wallisch, P., & Gordon, S. (2020). *Hare Psychopathy Checklist (PCL-R)* [Table].

Free Energy Equation and Its Integration with Brain Revival

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Introduction

There has been a universal question of the god equation, the theory of everything, that no one has ever figured out. The Theory of Everything is defined as "equations capable of describing all phenomena that have been observed, or that will ever be observed" (Laughline and Pines, 2000, pg. 28). "Perception Web of Bias," presumably to be known as the God equation (at least what I had figured out), hypothetically speaking, is the key to immortality in humanity. Along with the Free energy equation formulated from this poetic phrase of god equation, I believe it is the solution to all problems that we are facing on the brink of extinction such as climate change.

EB = *free energy*
E = *energy*
B = *any arbitrary numbers of constant*

Free energy is an illusion. With this idea, we might have a resolution in reviving brain death only if it works in experimental settings. Therefore, the equation can be modified into following:

$EB^{*}C = E_2$
E_2 = *Energy of life after death*
E = *Energy of Life*
B = *Blood pressure*
C = *Any constant of energy that is added onto the energy equation to sustain life after* reviving the brain with EB.
Precisely the equation will be $(E_0)^{*}C = C$

C will be, as a result, an energy required to sustain life, hence, replication of heartbeat energy.

Therefore, brain revival can be possible if one briefly and intentionally makes cardiac arrest in life support devices in patients who are brain dead and supply the appropriate manipulation in life support to make the patient beat in heart after revival in his or her brain.

As the majority of this paper will be based on my thought experiments, it will be heavily emphasized on what I have been simulating in mind rather than actual experiments (due to the conditions of a pandemic).

Free energy equation in physics--an illusion of reality:

EB = *Free energy*

E as Energy and *B* as any arbitrary number of constant. All reality is an illusion; therefore, the theory that argues that our reality is in the matrix has its reasonability and power among the academia with strong persuasions and assurances among the scientists.

In the modern era, all energy equations are equated based on killing the fuel. However, if you tip this stereotype onto the other side, (hence, by creating an equation that does not kill the fuel but also uses the fuel at the same time to create clean energy) we can conclude that free energy is merely an illusion that still uses fuel. The only difference between free energy and modern energy equations is based on whether you use the fuel as dead matter or living matter to use the fuel to kill it or use the existence of life as a fuel.

Let's consider city planning as an example. Hypothetically speaking, if the city mayor renovates a road by converting the vibrations and heat on the road that a car emits, we can convert

that energy into electricity. Such mathematical components will be represented as Energy. If you put the numbers of cars as a constant, "*B*," the road will keep converting energy into electricity as long as there are numbers of cars existing on roads; hence, clean energy does not kill a fuel in creating energy (while we just use the existence of cars on roads). Once we change this power generator on roads into an electric car charger hub for all roads in the city, the city will have roads as charging ports for electric cars to charge while the drivers are driving. Once we attach a GPS based on the electricity from power generators on roads, we will even allow autopiloting cars as main public transportation with a self-sustaining road system with free energy that never dies out as long as there are drivers on it. And as more and more drivers are on roads, the traffic will create electrical energy exponential growth.

If such a method works in the actual experiment to create a self-sustaining road system for electric cars, we will soon substitute all cars that are run by fossil fuels and combat climate change. In other words, the free energy equation is an equation for clean energy.

Brain revival by applying free energy equation:

Brain revival after brain death requires sophisticated science with precision. However, with this equation I came up with, it will be just enough to create a life in brain death by tapping into this simple equation of (EB)*C=free energy=E_2.

If you set *E* as energy for life to sustain before death and *B* as blood pressure, we can hypothetically state that *B* is 0 assuming the subject is defined as dead. Therefore,

$$E0=1$$

If we multiply by any constant to fuel the life in the brain to continue to sustain life and consciousness (in the mathematical variable, *C*) we can revive a dead brain on top of the layer of E0 as CPR for the brain by cutting the blood pressure in patients who are declared as brain dead with life supports on him or her for a brief amount of time. As Bhattacharyya et al. (2020, pg. 1) mention, necrosignaling is a signal when a bacteria alerts its swarm when it is killed by outside forces or predator bacterias to have its swarm survive from threats. If hypothetically, the brain is ever revived in brain cells, it will prove that brain cells have bacterias and neurons that scream when blood pressure is cut off from patients who are on life support with an inactive brain (or dead brain).

Intentional Cardiac Arrest to Revive the Dead Brain:

$$(EB)=E0=1$$

E = Energy for life to sustain

B = blood pressure = 0

As Bhattacharyya, Walker and Harshey (2020, pg.1) mention, If microbes in brains supposedly exist and have an immune system of necrosignaling, it will prove that the brain is conscious through microbes and neurons interacting together. If this experiment of causing cardiac arrest intentionally in the heart succeeds in reviving the brain after the brain dies by an accident but still functions as life with life support systems, it will hypothetically indicate that microbes in brains do exist in brains in sleep mode to signal the brain with necrosignaling to revive the circuits in brains which will bring the dead brain to life (E0)=1 and *E* as the positive arbitrary number that defines the energy of life supported by life support). After a brief revival in the brain, manipulating life supports to bring the heartbeat again after theoretical intentional cardiac arrest would help the body to function again. If microbes do not exist, otherwise, it will still be an indicator that brain cells still do respond to zero blood pressure function-wise with sparks of conscious life within the brain.

Once we add additional energy of constant *C* by multiplying the E0 we will have a free energy of life stretched into prolonging life. On the contrary, if one develops *C* as in 1/D or (E0)/D, we will be able to make the free energy trapped in a battery format that can be applied to new types

of medicine that are efficient in powering the medications or vaccinations in battery format of free energy.

For example, If one hypothetically states E as an energy of life after death and B as fuel for life along with D as an energy of an artificial DNA of so-called XNAs as Talyor et al. studied to replicate new enzymes (Taylor et al., 2014, pg. 427) which will be theoretically engineered to kill diseases as future applications, we will be able to formulate our body as a battery in life to revive recharge whenever we reach brain death into life every time we reach death.

Therefore, as Balbus (2016, pg. 11662) defines the stress tensor or pseudotensor as "the stress energy of the gravitational radiation," the constant C or 1/D is based on the same mechanisms of stretching the reality to balance and cancel the metaphoric vector of EB, only if we were to visualize only the mechanism of how stress tensor cancels reality. As a result the brain revival is applied by the free energy equation to trap the free energy in battery format inside our biology to charge a life after death.

Conclusion

Life after brain death--is it alive or not?

Zeman (2001) states that consciousness is "(i) the waking state; (ii) experience; and (iii) the possession of any mental state" as a definition. However, modern science has yet to discover much about where consciousness comes from and where it is located in the brain. If the theoretical approach of microbes and neurons interacting to create consciousness is ever legitimate after proving that brain cells do respond in sparking life from death, we will realize the true nature of self-consciousness and its origin. And, particularly, if one functions normally with memory and emotions after a revival in one's brain, the definition of death will be rewritten.

The Theory of Everything:

The Theory of Everything or god equation can therefore be defined as "perception bias." Universe expands. Mathematics expands. Thus, the God equation cannot be defined with mathematics we have today but as a poetic expression of Perception Bias. If the universe is zero in summation in physics, it can be concluded that the universe exists because consciousness exists to perceive reality. And, most of all, humans are not the only self-aware species; even bats have their perceptions in perceiving the world in wavelengths and sound while spiders perceive the world based on vibrations. Humans are merely primates that perceive the world based on light wavelengths in perception bias. Therefore, our universe is a web of perception biases of all life and alien species in alien planets. If we trap all of these perceptions into one unified theoretical mathematics, we will have the true God equation. The free energy is one example of formulating direction in this Perception Bias of tipping the definition of death into reverse to pursue immortality. If The Theory of Everything is defined as the equation that describes every phenomenon, (Laughline and Pines, 2000, pg. 28), Perception Bias is the Theory of Everything and (EB)*C or (EB)/D will be the toolkits of the God equation to solve problems in science to even defy death.

The basic logic of how I came up with this equation is based on E as the set reality and B as the alternative reality on the top layer. As a result, exponents can be never-ending in (((EB)C)D)E ... if we were supposed to express only the realities in layers with linear directions to solve problems in all types of energy.

Conflict of Interest: None.

References

Adam Zeman, (2001), Consciousness, *Brain, 124(7)*, 1263-1289. https://doi.org/10.1093/brain/124.7.1263

Alexander I. Taylor, Vitor B. Pinheiro, Matthew J. Smola, Alexey S. Morgunov, Sew PeakChew, Christopher Cozens, Kevin M. Weeks, Piet Herdewijn & Philipp

Balbus, Steven A. Simplified derivation of the gravitational wave stress tensor from the linearized Einstein field equations. *National Academy of Sciences. 113(42), 11662-11666.*

Holliger. (2014). Catalysts from synthetic genetic polymers. Nature, *518*, 427-430.

Laughlin, R. B. and Pines, David (2000), The Theory of Everything, *National Academy of Sciences*, *97(1)*, 28-31,

Souvik Bhattacharyya, David M. Walker & Rasika M. Harshey. (2020). Dead cells release a 'necrosignal' that activates antibiotic survival pathways in bacterial swarms. Nature Communications, 4157.

General Innocuous Statements May Be Preventing Us From Leaving Social Stereotypes In The Past

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Acknowledgements

I would like to thank the entire psychology department at NYU, but especially Pascal Wallisch who first taught me the QDAFI method, which allowed me to fully understand research articles. I would also like to thank my TA for that course, and author of the original research article, Kelsey Moty.

Abstract

Social stereotypes have long existed in our society. Much research has been done in the field of psychology analyzing why this continues to be the case today and how it is perpetuated and the effects of it. This literature review looks at how much of the literature on this topic intersects and aims to define one of the underlying causes. Language and speech play a big role in day to day life, and it is thus no coincidence that they impact the shaping of our social understanding.

The research article by Moty et al., 2021 focused on what children infer from generic claims and whether their inferences reinforce social stereotypes and their beliefs about unmentioned groups. Previous research has shown that certain statements about groups generally convey what people who make up that group are like. The authors wanted to focus on how children draw inferences from generic claims that may lead to beliefs about a group that is not mentioned. For example, "boys are good at sports" may lead children to believe that girls, the unmentioned group in this case, are not. In the first and second study the authors hypothesized that the participants would assume that the group mentioned in the claim would be good at the activity, and the unmentioned group would not be. In the third study, the authors hypothesized that if a knowledgeable person made a claim that one group is good at something, the participants would infer the unmentioned group is not good at that, and that the older the participants the more they would make this inference when conveyed by a knowledgeable person.

The design of the study was as follows. In study 1, the participants completed a novel category inference task, which introduced the groups and the two generic claims associated with each, and a context dependent pragmatic ability task. Study 2 was a replication of the first, but the authors removed the generic claims from the introduction. In study 3, the child participants completed the same tasks as in study 1, but the participants first completed a warm up task. Meanwhile the adult participants complete just the warm up and the novel category inference task. Additionally, half the participants learned from a knowledgeable person while the other half learned from an unknowledgeable person.

The authors found that in all three studies, child participants made inferences about groups not mentioned in the generic statements when conveyed by a knowledgeable speaker. The children started making these inferences around four and a half years old and as they get older the inferences grow stronger. The article suggests that children draw inferences from the person's intended meaning, and that social stereotypes may

very well be unintentionally conveyed to children by their parents starting at a very young age and continuing to be reinforced. These generic claims may be shaping children's social understanding. The authors note that more research needs to be done on the underlying mechanisms as to why this occurs.

Interestingly, there have been many articles similar to Moty et al., 2021, which relate to social stereotypes emerging in childhood. Bian et al., 2017 found that as girls grow up they are more likely to categorize boys as "really, really, smart" than they are girls. Moreover, Furnham et al., 2002 found that parents rated their son's IQ as being much higher than their daughter's, and generally boys self rated their intelligence higher than girls did. This in turn later leads to the findings that women are underrepresented in fields where brilliance is believed to be a requirement to success (Meyer et al., 2015). This is an issue that impacts the whole of society and that is being perpetuated by society. The way adults speak to their children really matters, and it has a much bigger impact than most believe.

From Moty et al., 2021 article, the question was to investigate what the underlying mechanism at play is. This is also the question in many of the research articles cited. Why does this happen? Part of the answer as to why women and girls think of themselves, and others also think of them, as being less intelligent than men and boys is probably due in part to the finding from Moty et al., 2021. The messages given to children are often generic statements that although may seem innocuous, determine their social understanding. More often than not we have all heard statements like "Sports are for boys", thus implying that sports are not for girls, and that "Boys are good at math", implying that girls are not. However, inferences are not just made about unmentioned groups. When trying to include both groups in a sentence such as "Girls are as good as boys at math," the position of the word in the sentence mattered, whether it is in the position of being the complement or subject. In this sentence the inference drawn by the adult participants was that boys are actually better at math than girls.

Even as adults, messages continue to reinforce the social understanding that has likely already been present since childhood. There is a silver lining that all these research articles teach us: to be aware of how we say things. One key takeaway is to not use generics and instead of saying "Girls are good at drawing" perhaps say "that girl is good at drawing" or "Sally is good at drawing." This is all in order to stop perpetuating social stereotypes and give children a chance to form their own social understanding of the world, and believe in themselves and in their own intelligence. More research is still needed, especially as to why inferences are drawn about unmentioned groups and why exactly the sentence structure is important.

References

Bian, L., Leslie, S. J., & Cimpian, A. (2017). Gender stereotypes about intellectual ability emerge early and influence children's interests. Science (New York, N.Y.), 355(6323), 389–391. https://doi.org/10.1126/science.aah6524

Chestnut, E.K. and Markman, E.M. (2018), "Girls Are as Good as Boys at Math" Implies That Boys Are Probably Better: A Study of Expressions of Gender Equality. Cogn Sci, 42: 2229-2249. https://doi.org/10.1111/cogs.12637

Furnham, A., Reeves, E., & Budhani, S. (2002). Parents think their sons are brighter than their daughters: sex differences in parental self-estimations and estimations of their children's multiple intelligences. The Journal of genetic psychology, 163(1), 24–39. https://doi.org/10.1080/00221320209597966

Meyer, M., Cimpian, A., & Leslie, S. J. (2015). Women are underrepresented in fields where success is believed to require brilliance. Frontiers in psychology, 6, 235. https://doi.org/10.3389/fpsyg.2015.00235

Moty, K., & Rhodes, M. (2021). The Unintended Consequences of the Things We Say: What Generic Statements Communicate to Children About Unmentioned Categories. Psychological science, 32(2), 189–203. https://doi.org/10.1177/0956797620953132