Testing the Effect of the Social Networking Services Environment on the Schema of Depressed Humans

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I. Abstract

With the rise of depression, various explanations as to why the disorder endures have risen. The central issue within depression's permanence in society proves to be its evolutionary mismatch in which our current environment differs from ancient settings but the same genetic factors are in play. In this way, the biological pathway itself has not changed, but the external stressors that elicit depressive reactions have and in turn, induce depressed symptoms and behaviors. Moreover, individualized mismatch disorder is also common since early, developmental environments in synthesis with one's genetics can impact one's future response to environmental stressors and potential depressive responses. Thus, research conducted in the following experiment sought to observe how modern environments, particularly characterized by social networking stressors, negatively impact neurological networks and inherently, depression.

II. Introduction and Literature Review

Depression, a neuropsychiatric mental health illness (Wingert & Kantrowitz, 2002), proves to permeate society at an expeditious and seemingly uncontrollable rate. Depressive disorders are best characterized in disease-state terms including episodes, remission, recovery, relapse, and recurrence (Frank et al., 1991). Due to this codification, identification and treatment for depression most often follow the *Diagnostic and Statistical Manual of Mental Disorders*, the standard diagnostic system, to check for symptoms (American Psychiatric Association, 2000). Moreover, the diagnosis and prevalence of depression in local communities and global populations has greatly increased in our current society. According to Ferrari et al. (2013), depression afflicts approximately 4.7 percent of the global population with high levels of comorbidity, including anxiety, and the capacity to damage social relationships. This

proliferation of major depressive disorder only increases in industrialized societies such as the United States, Europe, and Japan. In the United States alone, 25 million citizens qualified for a depressive disorder (Keller, 1994) which often results in monetary expenditure and lifestyle renovation. Antonouccio et al. (1997) explicate that the annual financial cost of depressive disorders based on health care costs, treatment prices, and job absence is over 40 billion dollars. Similarly, approximately 18.4 million Europeans battle with depression, indicating a lifetime prevalence of 20% in that population; further, the World Health Organization demonstrates that by 2020, depression will serve as the highest-ranking cause of disease in Europe (Wittchen & Jacobi, 2005). Lastly, as of 2011, the Japanese Ministry deemed mental illness as one of the "Five Major Diseases" receiving special focus in national medical policies. With nearly 10 to 15 percent of its population suffering from depression, Japan expects depression will be the second-ranked disease to cause a decrease in overall life expectancy (Nabeshima & Kim, 2013). Evidently, depression continues to pose a significant threat to society, and its ubiquity is rising in various environments.

Depression proves an evolutionary paradox as it persists in different environments despite selective pressures to eliminate its existence in the gene pool. In more primitive, ancestral environments, host immune responses to pathogens and predators provided humans with certain survival advantages (Miller & Raison, 2016). Communication between cerebral neurocircuits and inflammatory pathways incited behavioral responses, like avoidance and alarm, that advantaged early humans. In turn, humans developed a genomic bias towards inflammation because depressive response and symptoms promoted host survival and reproduction, particularly in highly pathogenic environments of human development (Raison & Miller, 2013).

In these early contexts, infectious causes killed 50 percent of humans before adulthood (Gurven & Kaplan, 2007), and subsequently, the body formulated defense mechanisms to counter external, threatening stimuli. The stressor triggers inflammatory pathways within peripheral blood mononuclear cells, such as the actuation of the transcription factor nuclear factor –κB (NF-κB), which prompts pronounced rises in circulating levels of pro-inflammatory cytokines, including interleukin-6 (IL-6) (Pace et al., 2006; Bierhaus et al., 2003). In this sense, the body actually constructs an immune response against a threat to its self-esteem, not against the pathogen directly. Since microbial interactions catalyze evolution, strong selective pressures favored genetic alleles and organisms that maximize host defense, stress perception, and wound healing. According to Miller & Raison (2016), these duplicated alleles for depressive responses in the modern climate possess pro-inflammatory or anti-pathogen protective effects such as chronic cytokine exposure that translates into pathogen-reducing social behaviors. For example, social avoidance and anhedonic tendencies of depression originally allowed individuals to administer energy resources towards infection-fighting and wound healing. Similarly, the hypervigilance of anxiety disorders, normally comorbid with depression, enabled protection and defense from attacks and diseases (Slavich & Irwin, 2014). This overlap between pro-inflammatory cytokines and the sickness syndrome of depression serves as the basis as to why depression and its modern manifestations have endured.

From the above, it is apparent that depression acts as an evolutionary mismatch disorder as its once advantageous traits now prove maladaptive due to environmental changes (Miller & Raison, 2016), both evolutionarily and within one's own lifespan. As Andrews and Thomson (2009) explain, new neuropsychiatric studies regard depression as an adaptation rather than as a

disorder. The main piece of supporting evidence for this theory is the maintenance of the 5HT1A receptor in the human brain. In an analysis of the receptor in rodents, whose 5HT1A receptor is 99 percent similar to humans' (Hartig et al., 1992), rodents that did not possess this receptor exhibited fewer depressive symptoms in response to stress than those with this neurological receptor. Hence, the receptor proves to be involved in the promotion of depression, and thus, its presence in modern humans alludes to the idea that the receptor was favored in natural selection. Furthermore, according to Sheeber et al. (2001), increasingly depressed individuals often exhibit increased intelligence and critical thinking in academia and social dilemmas. One reason to suspect that depressed individuals demonstrate enhanced problem-solving ability comes from their prolonged, uninterrupted ruminations with minimal neuronal damage. In this way, depressed humans focus on information processing and cost-benefit analysis, prioritized over daily activity, which explains symptoms such as loss of appetite (Andrews and Thomson, 2009). Hence, new behavioral treatment therapies frequently aim to solve the root problems that induce depression in coordination with medical treatment.

Similarly, in current society, the relationship between depression, genetics, and environment proves to be crucial. Faced with the psychological challenges of the modern world, humans have demonstrated ancestral immunological behaviors that constitute a decided liability, such as high rates of inflammation-related disorders like depression. Environmental risk factors for depression development, including psychosocial stress, obesity, processed-food diet, and early life adversity, are invariably pro-inflammatory (Raison et al., 2010), and consequently, interactions between inflammation and the brain exacerbate depressive tendencies and symptoms (Miller & Raison, 2016). In highly technological, volatile modern times, chronic stresses such as

social nonconformity, bullying, addictive drug abuse, and family loss enable humans to become gradually more vulnerable to depression and other mood disorders. According to Bayer et al. (1999), a two "hit" hypothesis helps correlate genetics and environmental depressive factors between developmental stages and adult stages in humans. The first "hit" is composed of environmental factors, including obstetric complications, undernourished birthing, winter birth, birth/growth in a large city, influenza infection during the midgestation period, or relationships with family members, as well as genetic mutations, such as ABCB1, 5-HTT LPR, DISC1, neuritin, dysbindin, and proline dehydrogenase (PRODH). The second "hit" encompasses purely environmental factors, like mental stress, social nonconformity, withdrawal and other forms of diminished communication, bullying, bereavement of a close relative, which then expose genetic mutations for depression. Children who experience the first "hit" prove vulnerable to psychiatric depression, and when they receive the second "hit" in puberty, they may develop depression.The researchers elucidate that to avoid depressive exposure and promote resilience, people should aim to create enriched environments fostered by communal assistance and support, active exercise, and stress resistance procural (Bayer et al., 1999).

With regards to the mechanism of depression in modern times, according to Beck (2008), when cognitive structures are activated by a depressive event, the schemas disrupt the information processing system, similar to its adaptive advantage, but, in turn, they direct attentional resources towards negative stimuli and distort experiences into a negative interpretation. These schemas are regulated by the 5-HTTLPR (serotonin transporter) gene; individuals with variant short copies of this gene, which are less transcriptionally effective as the long-form, experience higher levels of depression and suicidality after stress-inducing events

(Caspi et al., 2003). In this way, stressful environments prey upon those with depressive genetic mutations that now prove maladaptive. For example, a loss in childhood or early adolescence would sensitize an individual to an analogous loss at a later time and lead to depression (Sethi, 1964). Hence, as Gluckman et al. (2007) proposed, diseases characterized by developmental risk factors such as depression reflect a mismatch between early, developmental environments and later, matured environments, not just an evolutionary mismatch. Moreover, once in a depressive state, the fully activated mode, a network of cognitive, affective, motivational, behavioral, and physiological schemas to account for depressive behaviors, becomes autonomous and ineffectively reactive to external stimuli. Thus, positive events lack the capacity to reduce negative moods (Beck, 2008). Another example of mismatch disorder is evident in a study of Oomen et al. (2010) in which rats deprived of maternal care at postnatal day 3 saw reduced hippocampal neurogenesis and impaired spatial cognition under mild stress condition. In contrast, these rats under high-stress levels exhibited contextual learning and long-term potentiation in the dentate gyrus. In this example, mismatch disorder proves to be a fine line since a match between developmental and adult environments can promote successful adaptation and survival, while a subtle mismatch can result in disease like depression (Schmidt, 2011). The match facet of the mismatch hypothesis demonstrates that early life environments shape coping strategies and stress resiliency to best face similar environments later (Belsky and Pluess, 2009; Ricon et al., 2012; Schmidt, 2011).

One of the primary changes from ancient to modern environments is the advent of social media networks. Although not always negative, social networking service (SNS) use increases the exposure of individuals to negative social interactions such as cyberbullying which often

results in negatively impacted mood and mental health including depleted self-esteem and satisfaction (Valkenburg et al., 2006). Moreover, the SNS environment enables one to ruminate on negative events while the entire social network exacerbates corresponding shortcomings. According to Steers et al. (2014)'s results from a retrospective study, a positive correlation exists between time spent on Facebook and depression. More so, passive users of Facebook often exhibited higher levels of anxiety through rumination, a mediator (Seabrook et al., 2016). In this way, Facebook exacerbates depressive symptoms through sentiments like envy (Tandoc et al., 2015). However, Facebook and other SNSs only serves as exacerbators rather than sources of depression. For example, Facebook frequency did not predict social anxiety in the entire population but rather only correlated with anxiety for a high anxiety group. (McCord et al., 2014). Similarly, people with depression perceived negative interactions on SNSs, exhibited through disconnection and depressive symptoms, at a greater magnitude than those without (Davila et al., 2012). Therefore, a perceptual bias is prevalent as a participant's interpretation of the quality of interactions varies depending on depressive levels regardless of true communication content exchanged (Seabrook et al., 2016). In summary, negative interactions, SNS addiction or problematic use, and continual social comparison often correlate to higher levels of depression exacerbated by cognitive response styles such as rumination.

III. Research Hypothesis

The elimination of the currently unique environmental mismatch, social networking service use, even for a brief period of time, will alter schema outlooks in depressed people to be more positive and can help normalize and treat depressive states.

IV. Material and Methods

As the most ubiquitous environmental change between ancient and modern society, social media networks' effect on neural schema would be observed in this experiment. More specifically, the experiment would analyze the absence of social media platforms from former users among various age groups and different mental states (i.e. depressive or normal). As schemas comprise a person's perception and are believed to be formulated throughout one's development, four main age groups would be studied: 6-18 year olds, 18-34 year olds, 34-50 year olds, and 50-65 year olds. Each age range would consist of a control group free of social media use for the prior two weeks. In turn, each age group would also possess experimental groups that used social media for the prior two weeks for at least 5 hours per week. Within the experimental groups, classifications would be arranged to further organize frequency of SNS use; for example, users of 5-10 hours per week would be arranged together likewise with users of 10-15 hours per week, 15-20 hours per week, and 20-25 hours per week. Moreover, each age and frequency range would possess two varying categories: a group of testers with depressive symptoms and/or medical depression and a healthy group without. Lastly, each individualized group would possess a roughly equal amount of men and women to eliminate genetic gender differences. Hence, the "ultimate" control group would be 34-50 year olds of each gender who did not use social media during the initial two weeks and did not exhibit any depressive tendencies. Optimally, the sample size would be greater than 500 participants to eliminate abnormalities and exceptions.

After the initial two week period of social media usage or deprivation, all participants would lack access to any social networking services including Facebook, Instagram, text

messaging, Snapchat, and email for the following two weeks. Throughout these latter two weeks, experiment administrators would evaluate schema outlooks of individuals every 24 hours via the Early Maladaptive Schema test that evaluates 18 specific schemas under five broader schema domains: (I) Disconnection and Rejection, (II) Impaired Autonomy and Performance (III) Impaired Limits, (IV) Other-Directedness, (V) Over vigilance and Inhibition. As similar to its use in Darvishi et al. (2012), these schema qualifications would be determined using the Young Schema Questionnaire Short Form, a 75 item self report measure of early maladaptive schemas. This measure allows participants to rate items in terms of how they feel about their lives on a 6-point Likert scale. After each day, the results would be recorded and at the end of the trial would also be plotted on a bar graph (X axis: five major schema domains, Y axis: number of participants who suffered from the corresponding domain, two bars for each domain: those with depression and those without) to observe and display the development and change of schema in depressed people when exposed to social networking services and when independent of any social media.

V. Conclusion and Justification

Visibly, depression serves as one of the largest threats to our modern society, not only as the number of affected people rise but also in the convulided quality of treatment plans. This research enables physicians and psychologists to coordinate treatment and provide behavioral models for depressive patients to follow in addition to standard medicinal treatment. By tracking schema's reactive response, change, and restructure due to social media, healthy social media usage may also be better regulated within households to adjust for age ranges and mental health states. Moreover, this frame of research allows society to determine to what extent are social

networking services beneficial in contrast with technology as a whole within our current, ever-industrializing society. Ultimately, this study provides profound insight into modern society's contextual mismatch with our evolutionary ancestors and has the capacity to change individual lives and health care policy in regards to depressive treatment and social networking service regulation.

VI. Bibliography

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