

SFU Ψ UJP

SIMON FRASER UNIVERSITY
UNDERGRADUATE
JOURNAL OF PSYCHOLOGY



VOLUME III
2016

SFU ψ UJP

SIMON FRASER UNIVERSITY
UNDERGRADUATE
JOURNAL OF **PSYCHOLOGY**



VOLUME III
2016

ISSN 2368-6340 (Print)
ISSN 2368-6359 (Online)

JOURNAL STAFF

MANAGING EDITORIAL STAFF

Emma Carlson

Dana Cochrane

Chelsey Lee

ASSOCIATE EDITORS

Sebastian Bagole	Laila Namur
Emily Hirst	Sarah Power
Negina Khalil	Samara Wessel
Sean Maxey	

REVIEWERS

Hilary Aime	Lynnaea Northey
Adam Baker	Bertrand Sager
Adam Blanchard	Andrea Smit
Richard Hohn	Maxine Wagner
Elisabeth Kreykenbohm	Matthew Wakefield

FACULTY ADVISORS

Dr. Tim Racine Dr. Neil Watson

The views and ideas contained within this publication reflect only those of the contributing authors, not the SFU-UJP or the Department of Psychology at Simon Fraser University.

All articles in this publication are licensed under a Creative Commons Attribution - 4.0 International License. For all other content: © 2016 Simon Fraser University Undergraduate Journal of Psychology.

For more information: visit members.psyc.sfu.ca/ujp, or email ujp@sfu.ca

Journal layout design by Filip Kosel, Erin Fuller, and Ryan W. Carlson.

CONTENTS

- iii JOURNAL STAFF
- v LETTER FROM THE EDITORS
Emma Carlson, Dana Cochrane, Chelsey Lee

REVIEW ARTICLES

- 1 MENTAL HEALTH AND WELL-BEING AMONG CANADIAN UNIVERSITY STUDENTS: REVIEW AND RECOMMENDATIONS
Ravina Gill
- 10 A NEED FOR LEGAL RECLASSIFICATION: THERAPEUTIC AND MEDICAL BENEFITS USING LYSERGIC ACID DIETHYLAMIDE
Cynthia McDowell
- 17 THE FEELING OF BEING “THERE”: PRESENCE AND THE ROLE OF VIRTUAL REALITY AS A RESEARCH TOOL
Alec McLeod
- 25 AGAINST THE GRAIN: DIET AND ALZHEIMER’S DISEASE
Nancy Yang

LETTER FROM THE EDITORS

It is with pleasure and excitement that we present Volume III of the Simon Fraser University Undergraduate Journal of Psychology. Our goal this year was to extend and honour the effort put forth by those who contributed to the first and second editions. Sustainability of this journal and dedication to the SFU community are cornerstones that we hope to carry forward with this edition.

This year, we received particularly outstanding submissions, which demonstrate the caliber of the work being produced by Undergraduate Psychology students this year. We are so thankful that there is an opportunity to highlight their determination and perseverance towards research excellence, and their work serves as the foundation of the Journal. Without them, this would not be possible. While we can only print a small portion of the articles we receive, our Journal team extends its admiration to all those who submitted. Each body of work offers something unique, and we feel honoured to experience these perspectives in Psychology.

We would like to extend our gratitude to the editors and reviewers for all their work and effort on this issue; they have volunteered hours of their time to review and revise each article submitted, and have worked tirelessly to help develop their fellow students as authors and researchers. In addition, this edition would not have been possible without the guidance of Dr. Tim Racine and Dr. Neil Watson, as well as the generous support of the SFU Department of Psychology and the Psychology Student Union.

Finally, we extend our utmost thanks to you as our reader. It is our hope that this publication inspires your inner researcher, and we look forward to seeing great things from you in the future.

- The Managing Editorial Staff
Emma Carlson, Dana Cochrane & Chelsey Lee

Mental Health and Well-being among Canadian University Students: Review and Recommendations

Ravina Gill

SIMON FRASER UNIVERSITY

Mental health and well-being among Canadian university students has been declining over the past few decades (Durand-Bush, McNeill, Harding, & Dobransky 2015; Mahmoud, Staten, Hall, & Lennie, 2012; Markoulakis & Kirsh, 2013; Nunes et al., 2014). A number of studies demonstrate a positive association between academic success and optimal mental health (Durand-Bush et al., 2015; Markoulakis & Kirsh, 2013; Nunes et al., 2014). Research shows there is a greater prevalence of psychological distress in university students compared to young adults in the general population (Durand-Bush et al., 2015). This literature review examines a few explanations for this decline in mental health and some potential stressors that may have contributed to this problem. The literature reveals the following factors as the most prevalent in the decline of Canadian students' mental health: academic pressure, financial stress, and increased competition in higher education. Finally, the paper suggests recommendations for future research and strategies to be employed by higher educational institutions to increase psychological well-being. This paper is modeled using Keyes's dual factor model of mental health.

Keywords: Canadian undergraduate students, mental health, well-being, academic

There is a growing concern regarding the overall mental health and well-being of young adults in Canada (Markoulakis & Kirsh, 2013). Mental health and well-being are essential components to an individual's overall health (Keyes, 2005; Markoulakis & Kirsh, 2013; Statistics Canada, 2010). According to the World Health Organization (2014), the well-being of an individual includes their ability to manage common life stressors and work effectively while being able to contribute to their community. In general, increased psychological well-being is correlated with success across various life domains such as work, relationships, and overall health (Keyes & Grzywacz, 2005; Lyubomirsky, King, & Diener, 2005). Research within positive psychology, as

applied to educational settings, suggests a similar relationship between well-being and academic achievement (Durand-Bush, McNeill, Harding, & Dobransky, 2015; El Ansari & Stock, 2010). Many young adults will experience various significant life changes in conjunction to completing their education (Nunes et al., 2014). These changes may include forming relationships, gaining financial responsibilities, balancing education and employment commitments and so forth. It is not a shock that mental health concerns arise during early adulthood given these important changes (Nunes et al., 2014). Over the past decade, there has been a growing awareness in the rise of mental health concerns among university students (Durand-Bush et al.,

Copyright: © 2016 Gill. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

2015). The purpose of this paper is to review the decrease in mental health among the young adult population across Canadian universities, address the potential reasons for this declining trend, and provide strategies and recommendations for future research in order to increase psychological well-being and academic success.

Model of Mental Health

Mental health is not simply the absence of mental illness (Keyes, 2005). Keyes, a psychologist who has done significant work within positive psychology, developed a model for mental health that is widely used across Canada (Keyes, 2005; Peters, Roberts, & Dengate, 2011; Windhorst & Williams, 2015). Keyes's (2005) dual factor model recognizes that mental health and mental illness exists as two separate but co-occurring continuums, which provides a complete representation of an individual's mental state. This paradigm defines mental health as a state of subjective well-being in which an individual is able to positively function in their life; in contrast, mental illness refers to an individual who meets the criteria for a certain mental condition (Keyes, 2005; Peter et al., 2011). The model illustrates that individuals without a mental illness are not necessarily mentally healthy (Peter et al., 2011). Consequently, it is possible for someone to have a mental illness but be mentally healthy and vice versa. Therefore, an individual can have a mental illness but because they are able to optimally function within their life, they show positive mental health (Keyes, 2005). Similarly, an individual may have lower levels of mental health as shown by symptoms of distress, but not have a mental illness (Keyes, 2005). For instance, a Canadian study using the dual continua model found that students with low levels of well-being displayed symptoms

of depression, but some others with a mental illness showed high levels of well-being (Peter et al., 2011). This study operationally defines levels of well-being using six dimensions of positive psychological functioning, which include self-acceptance, interpersonal relationships, autonomy, personal growth, managing one's environment, and a purpose in life (Peter et al., 2011). Therefore, the dual factor model allows for the conceptualization of mental health as emotional, psychological, and social well-being (Keyes, 2005). The application of this model to the mental health of the student population is necessary in order to understand the mental health difficulties of young adults in the context of post-secondary education.

Decline in Psychological Well-being

In recent years, the demographic of the current generation of university students has changed tremendously, as have their mental health needs. The university student population is considerably diverse with a large number of international students and a broad age range of students with many over the age of 25 (Burns, Lee, & Brown, 2011; Kitzrow, 2009; Mahmoud, Staten, Hall, & Lennie, 2012; Soet & Sevig, 2006; Statistics Canada, 2010). The age of onset for many mental illnesses is between 18 to 24 years old (Kessler et al., 2005). This is often the age of the typical university student (Eisenberg, Gollust, Golberstein, & Hefner, 2007; Kessler et al., 2005). During the last few decades, reports show mental health problems are increasing among young adults within university (Eisenberg et al., 2007; Kitzrow, 2009). A greater proportion of students are experiencing severe levels of psychological distress, compared to traditional issues of adjustment to university seen in students during the 1980's (Eisenberg et al., 2007; Kitzrow, 2009). The most

common mental health problems found in young adults include anxiety, mood, and substance use disorders (Nunes et al., 2014). Correspondingly, there is an increase in the number of university students dealing with depression and anxiety (Nunes et al., 2014). According to the American College Health Association, a great number of students felt overwhelmed (87%), excessive anxiety (55%), and depressed with decreased functioning (33%) at some point within the past year (Durand-Bush et al., 2015). This survey data, collected from approximately 16,000 Ontario university students, indicated a significant proportion of students were severely distressed (Durand-Bush et al., 2015). These findings are consistent with results from a health survey conducted by the Centre for Addiction and Mental Health, which report 30 percent of Canadian undergraduates are highly distressed, as measured by the twelve item General Health Questionnaire (Durand-Bush et al., 2015; Nunes et al., 2014). This questionnaire is a well established measure used to test mental health functioning, which has been used in many studies involving university students (Durand-Bush et al., 2015). Studies show constant, unresolved stress can lead to psychological distress, which may result in severe mental health concerns (Durand-Bush et al., 2015; Nunes et al., 2014). The levels of distress among students are twice as high compared to their age-matched peers not in university (Durand-Bush et al., 2015). Furthermore, elevated levels of distress are associated with decreases in physical and mental health and poorer academic success (Park, Edmondson, & Lee, 2012). This data illustrates the serious mental health difficulties among undergraduate students within Canada over the last decade.

Health survey data from students across Canadian post-secondary institutions reveal that emotional distress is

associated with academic performance (Durand-Bush et al., 2015; Nunes et al., 2014; Park et al., 2012). Research shows a positive relationship between poor mental health and academic attrition because students who display higher levels of distress are more likely to perform worse academically or withdraw from university (Durand-Bush et al., 2015; Nunes et al., 2014). A survey of over 6,000 university students across Canada showed symptoms of distress, such as feeling over-worked, worried, unhappy, depressed, and exhausted, in approximately 30 percent of the sample (Nunes et al., 2014). In addition, students with mental health problems experience physical, psychological, and social difficulties as well as poorer academic outcomes (Markoulakis & Kirsh, 2013). These associations highlight the extensive mental health difficulties experienced by students, which can impact their success within the university community and beyond.

Stressors Impacting Mental Health

The previously mentioned research shows many students are under great psychological distress, but this research fails to address the potential stressors impacting mental health. The pressures faced by students during their post-secondary education are complex and may include, but are not limited to, academic demands, increased competition in higher education, and financial burdens (Eisenberg et al., 2007; Kruisselbrink, 2013; Markoulakis & Kirsh, 2013). Studies indicate students encounter these various life stressors, but often do not perceive them as related to their mental health difficulties (Durand-Bush et al., 2015; Markoulakis & Kirsh, 2013). Many of these identified stressors are also seen in the general student population, but the severity of the concerns experienced by students with mental

health problems is greater (Markoulakis & Kirsh, 2013).

Students who show signs of depression and anxiety often state that academic stress is one of the main factors contributing to their mental health problems (Nunes et al., 2014; Markoulakis & Kirsh, 2013). Students with untreated mental health concerns earn significantly lower grades, compared to their peers (Markoulakis & Kirsh, 2013). Some researchers suggest mental distress may be contributing to students' difficulties with concentration, memory, stress, and organizational ability (Markoulakis & Kirsh, 2013). It is important to note that concentration, memory, stress, and organization are essential components for academic success. If these components are impaired in students due to mental health troubles, it can have detrimental effects on their academic success (Markoulakis & Kirsh, 2013).

Students feel a great pressure to achieve high grades within university. High grade expectations may be due to the competitive nature within post-secondary institutions (Markoulakis & Kirsh, 2013; Nunes et al., 2014). In today's job market, there is a greater requirement for higher education compared to thirty years ago (Kitzrow, 2009; Markoulakis & Kirsh, 2013; Nunes et al., 2014). Students feel they must contend with other students in order to ensure academic success (Kitzrow, 2009). Research shows 75 percent of young Canadians participate in higher education within at least four years after graduating high school (Kitzrow, 2009). Thus, greater accessibility to post-secondary education has allowed for a more diverse student population (Kitzrow, 2009). Currently, very little research has been conducted on the mental health needs of this diverse student population. The effort to meet such high expectations within university and inability to cope with stress, are likely contributing to the decline

in mental health in the undergraduate student population (Markoulakis & Kirsh, 2013; Peter et al., 2011).

Another common stressor for students is financial stress. In Canada, tuition fees have been steadily rising with students experiencing greater student loans and debt after graduation (Merani et al., 2010). A study conducted by Merani and colleagues (2010) show a positive correlation between the amount of tuition fees paid and the reported stress. Research shows students with large amounts of financial burden tend to have reduced academic performance and are more likely to experience mental distress compared to their peers (Eisenberg et al., 2007; Merani et al., 2010; Ross, Cleland, & Macleod, 2006). Another study shows students from lower socioeconomic backgrounds are more likely to experience symptoms of anxiety and depression (Eisenberg et al., 2007). This finding is similar to the socioeconomic differences in mental health within the general population (Eisenberg et al., 2007). However, further research examining socioeconomic status and mental health among university students is needed (Markoulakis & Kirsh, 2013). This may imply some students are performing poorly and experiencing poorer mental health because of debt and financial burdens (Eisenberg et al., 2007). The most common instances of financial stress include paying for tuition and living expenses while balancing work and academic commitments (Markoulakis & Kirsh, 2013). Additionally, students experiencing mental health problems often take longer to finish their degrees compared to the average university student (Markoulakis & Kirsh, 2013). It is possible that a combination of academic stress and financial burdens may prolong degree completion rates. The stress caused from high tuition costs and academic pressures both appear to be common factors impacting mental

health in students.

University students come in contact with many academic, social and financial stressors that may negatively impact their mental health (Mahmoud et al., 2012). Additional research examining the potential factors involved in student distress is required. Very few studies have examined the demands associated within university life from the viewpoint of students experiencing mental distress (Markoulakis & Kirsh, 2013). This perspective is essential to determine the best support services for students. The complex interaction of factors that create negative outcomes for students under mental distress need to be more clearly recognized in order to provide solutions for students to overcome these barriers.

Recommendations & Challenges

At the institutional level there is a critical need to address this rise in mental health problems by implementing strategies to increase the well-being of students. In general, it appears that students' experiences of stress are hindering their academic performance (Durand-Bush et al., 2015). Since consistent stress can translate into severe mental health concerns, students would benefit from strategies that alleviate these difficulties. Some studies suggest students do not have sufficient coping skills to deal with university life (Durand-Bush et al., 2015; Hofer, Busch, & Kartner, 2011; Mahmoud et al., 2012). Researchers suggest the use of self-regulating skills in planning, controlling, and evaluating thoughts to achieve success in a dynamic university environment can lead to higher levels of well-being (Hofer et al., 2011). Research indicates that utilizing these self-regulating skills significantly predicts the students' levels of stress, well-being, and mental health (Park et al., 2012). Compared to previous

decades, studies show more students are seeking out counselling services (Burns et al., 2011; Kitzrow, 2009; Markoulakis & Kirsh, 2013). However, other reports indicate students are less likely to obtain help (Martin, 2010; Peter et al., 2011). It appears many students avoid seeking help on campus for their mental health conditions because they are worried about stigmatization (Martin, 2010). Students worry about how others may perceive them if they utilize mental health services (Markoulakis & Kirsh, 2013). Reports show students fear being rejected within university, their community, and from future employment opportunities, if they disclose their mental health problems (Markoulakis & Kirsh, 2013; Martin, 2010). Moreover, researchers notice students who decide to seek help have already experienced academic impairment (Martin, 2010). A potential solution to this timing issue is to increase awareness about mental health to reduce the stigma around seeking help. University programs aimed at spreading acceptance of mental health is one possible way to increase awareness and reduce stigmatization (Martin, 2010). Such a prevention strategy may help students understand the concept of mental wellness, so should they experience distress they will seek help prior to academic impairment.

Traditionally, therapeutic approaches such as counselling services have been widely used to treat mental illnesses. Many challenges have occurred with the increase in serious mental health problems among students, including the increased need for more counselling services provided by institutions (Martin, 2010). However, these conventional practices neglect factors that promote well-being with the focus being on mental illness. In line with Keyes's (2005) model, it is important to recognize that mental health is on a continuum in which an individual may not be at

optimal mental health. Therefore, new strategies must be developed and implemented in addition to providing student support services to ensure optimal mental health among the student population. Future research examining programs that will provide students with essential coping skills and strategies to relieve stress may act as alternatives to counselling (Martin, 2010). For instance, offering free and easily accessible educational courses on stress management, basic health, and finances during the first year of university life may be beneficial for students. Another alternative to counselling may include meditation based programs to reduce symptoms of distress (Burns et al., 2011). Studies show meditation significantly alleviates symptoms of anxiety, depression, and stress (Burns et al., 2011; Kitzrow, 2009). Meditation programs can be a cost-effective approach to reduce mental distress among university students. Meditation can occur in a group setting or alone, which allows the university to reach a broader student population and ensure positive mental health and well-being among all students (Burns et al., 2011). It is important for institutions to be open to such alternative resources in order to ensure academic success and mental wellness of their students.

The physical environment of an institution can significantly impact mental well-being of the student population. Many environmental factors can influence student mental health such as natural environments, access to campus support services, and social inclusivity (Windhorst & Williams, 2015; Van den Berg et al., 2007). One study illustrated that natural environments can help promote positive mental health (Windhorst & Williams, 2015). The findings of this study suggest students prefer areas that are familiar, had aspects of nature such as trees, and are separated from various stressors such as auditory

and visual stimulation created by busy environments (Windhorst & Williams, 2015). The study reveals natural environments promote relaxation and reflection among students (Windhorst & Williams, 2015). These findings are consistent with research conducted by environmental psychologists who found that cross culturally, people prefer natural environments because they promote relaxation and provide social interaction (Van den Berg et al., 2007; Windhorst & Williams, 2015; Plane and Klodawsky, 2013). Since natural settings impact overall well-being, learning environments may benefit from incorporating such elements into their institutions. Since very few studies have investigated the relationship between natural environments in an academic institution and student mental health, further research is required.

The next step for higher educational institutions is to provide services and education that is geared towards creating successful and resilient citizens in the classroom and beyond. Future research could explore cost-effective alternatives to therapy such as changing the learning atmosphere (Windhorst & Williams, 2015). Offering alternative resources that educate students on mental health early in their university career may help impact student well-being by giving them the appropriate coping skills and information (Durand-Bush et al., 2015). Further research examining student mental health and well-being should focus on methods to reduce such stressors and promote resilience to produce functional young adults within society.

Conclusion

It is clear that mental health can dramatically impact the well-being of students within university. Post-secondary institutions across Canada are dealing with significant challenges regarding the changing mental health

needs of the current generation of university students. Mental health is a vital component of university life that can greatly impact learning and academic success. As previously discussed, mental health struggles are strongly associated with poorer academic outcomes (Markoulakis & Kirsh, 2013). It is difficult to determine all the stressors that influence mental health because of the complex interactions between each factor. Moreover, studies fail to address the differences in the current demographic of university students such as culture, which may differentially impact the mental health needs of students (Mahmoud et al., 2012). However, current research suggests some of the key contributors to the strain on mental health may include academic and financial pressures. Research examining the factors impacting student mental health, needs to be further investigated to provide effective solutions for students to overcome these obstacles. Although there has been increased awareness of mental health problems arising among university students, more must be done across all levels of the higher education system. Research shows it is equally as important to promote mental health to increase subjective well-being as it is to provide mental illness prevention strategies because promotion of positive mental health should be directed to all individuals (Peter et al., 2011). Active engagement in Canadian post-secondary institutions is required to develop policies and services to address student mental health. Thus, strategies to improve mental health must be treated as an institutional priority within higher education to ensure optimal well-being among all students.

References

- Burns, J. L., Lee, R. M., & Brown, L. J. (2011). The effect of meditation on self-reported measures of stress, anxiety, depression, and perfectionism in a college population. *Journal of College Student Psychotherapy*, 25(2), 132-144. doi:10.1080/87568225.2011.556947
- Dale, M. (2010) Trends in the Age Composition of College and University Students and Graduates. Statistics Canada. Retrieved on March 1st 2016, from <http://www.statcan.gc.ca/pub/81-004-x/2010005/article/11386-eng.htm>
- Durand-Bush, N., McNeill, K., Harding, M., & Dobransky, J. (2015). Investigating stress, psychological well-being, mental health functioning, and self-regulation capacity among university undergraduate students: Is this population optimally functioning. *Canadian Journal of Counselling and Psychotherapy*, 49(3), 253-274. Retrieved from <http://search.proquest.com/>
- Eisenberg, D., Gollust, S. E., Golberstein, E., & Hefner, J. L. (2007). Prevalence and correlates of depression, anxiety, and suicidality among university students. *American Journal of Orthopsychiatry*, 77(4), 534-542. doi:10.1037/0002-9432.77.4.534
- El Ansari, W., & Stock, C. (2010). Is the health and wellbeing of university students associated with their academic performance? *International Journal of Environmental Research and Public Health*, 7(2), 509-527. doi:10.3390/ijerph7020509
- Hofer, J., Busch, H., & Kärtner, J. (2011). Self-regulation and well-being: The influence of identity and motives. *European Journal of Personality*, 25(3), 211-224. doi:10.1002/per.789
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorder on the national co-morbidity survey replications. *Archives of General Psychiatry*, 62, 593-602. doi:10.1001/archpsyc.62.6.593
- Keyes, C. L. M. (2005). Mental illness and/or mental health? Investigating axioms of the complete state model of health. *Journal of Consulting and Clinical Psychology*, 73, 539-548. doi:10.1037/0022-006X.73.3.539
- Keyes, C. L. M., & Grzywacz, J. G. (2005). Health as a complete state: The added value in work performance and healthcare costs. *Journal of Occupational and Environ-*

- mental Medicine, 47, 523-532. Retrieved from <http://www.ovid.com/site/catalog/journals/index.jsp>
- Kitzrow, M. A. (2009). The mental health needs of today's college students: Challenges and recommendations. *Journal of Student Affairs Research and Practice*, 46(4): 646-660. doi:10.2202/1949-6605.5037
- Kruisselbrink, A.F. (2013). A suffering generation: Six factors contributing to the mental health crisis in North American higher education. *College Quarterly*, 16(1), 1-17. Retrieved from <http://collegequarterly.ca/index.html>
- Lyubomirsky, S., King, L., & Diener, E. (2005). The benefits of frequent positive affect: Does happiness lead to success? *Psychological Bulletin*, 131(6), 803-855. doi:10.1037/0033-2909.131.6.803
- Mahmoud, J. S. R., Staten, R. T., Hall, L. A., & Lennie, T. A. (2012). The relationship among young adult college students' depression, anxiety, stress, demographics, life satisfaction, and coping styles. *Issues in Mental Health Nursing*, 33(3), 149-156. doi:10.3109/01612840.2011.632708
- Markoulakis, R., & Kirsh, B. (2013). Difficulties for university students with mental health problems: A critical interpretive synthesis. *Journal of the Association for the Study of Higher Education*, 37(1), 77-100. doi:10.1353/rhe.2013.0073
- Martin, J. (2010). Stigma and student mental health in higher education. *Higher Education Research and Development*, 29, 259-274. doi:10.1080/07294360903470969
- Merani, S., Abdulla, S., Kwong, J. C., Rosella, L., Streiner, D. L., Johnson, I. L., & Dhalla, I. A. (2010). Increasing tuition fees in a country with two different models of medical education. *Medical Education*, 44(6), 577-586. doi:10.1111/j.1365-2923.2010.03630.x
- Nunes, M., Walker, J. R., Syed, T., De Jong, M., Stewart, D. W., Provencher, M. D., & Furer, P. (2014). A national survey of student extended health insurance programs in post-secondary institutions in Canada: Limited support for students with mental health problems. *Canadian Psychology*, 55(2), 101-109. doi:10.1037/a0036476
- Park, C. L., Edmondson, D., & Lee, J. (2012). Development of self-regulation abilities as predictors of psychological adjustment across the first year of college. *Journal of Adult Development*, 19(1), 40-49. doi:10.1007/s10804-011-9133-z
- Peter, T., Roberts, L. W., & Dengate, J. (2011). Flourishing in life: An empirical test of the dual continua model of mental health and mental illness among Canadian university students. *International Journal of Mental Health Promotion*, 13(1), 13-22. doi:10.1080/14623730.2011.9715646
- Plane, J., & Klodawsky, F. (2013). Neighbourhood amenities and health: Examining the significance of a local park. *Social Science and Medicine*, 99, 1-8. doi:10.1016/j.socscimed.2013.10.008
- Ross, S., Cleland, J., & Macleod, M. J. (2006). Stress, debt and undergraduate medical student performance. *Medical Education*, 40(6), 584-589. doi:10.1111/j.1365-2929.2006.02448.x
- Soet, J., & Sevig, T. (2006). Mental health issues facing a diverse sample of college students: Results from the College Student Mental Health Survey. *National Association of Student Personnel Administrators Education Journal*, 43(3), 410-431. doi:10.2202/1949-6605.1676
- Windhorst, E., & Williams, A. (2015). It's like a different world: Natural places, post-secondary students, and mental health. *Health and Place*, 34, 241-250. doi:10.1016/j.healthplace.2015.06.002
- World Health Organization. (2014). Mental health evidence and research. Retrieved on March 5th, 2016, from http://www.who.int/mental_health/evidence/en/
- Van den Berg, A. E., Hartig, T., & Staats, H. (2007). Preference for nature in urbanized societies: Stress, restoration, and the pursuit of sustainability. *Journal of Social Issues*, 63(1), 79-96. doi:10.1111/j.1540-4560.2007.00497.x

A Need for Legal Reclassification: Therapeutic and Medical Benefits Using Lysergic Acid Diethylamide

Cynthia McDowell

SIMON FRASER UNIVERSITY

Depression, addiction, and other mental health disorders present a serious challenge socially, personally, and economically, creating an urgent need for novel treatments. In the present research, the therapeutic potential of lysergic acid diethylamide (LSD) is examined in the context of alcohol and smoking addictions, Autism Spectrum Disorder, and other mental health conditions such as depression and anxiety. This includes an examination of the psychedelic substance as a way to explore mental states and reorient emotion-based cognitions. Using the psychedelic experience as medicine, LSD can be used to treat a specific psychiatric illness, targeting the source of the issue and allowing patients to gain insight into their own disorders and behaviour, all with relative safety. It is concluded that LSD should be legally reclassified in such a way that allows further exploration of its utility as a treatment for clinical disorders, as well as its potential to provide insight into the psychology of these conditions.

Keywords: lysergic acid diethylamide, mental health, therapy, treatment, legal

Lysergic acid diethylamide (LSD) is a potent hallucinogenic drug that evokes profound psychological and somatic effects (Frankel & Cunningham, 2002). It was first synthesized in 1943 by Swiss scientist Albert Hofmann, who soon after discovered the drug's psychedelic properties (Gasser, Kirchner & Passie, 2015). In 1949, it was brought to the attention of multiple medical centers in the United States when it was discovered that LSD could be used as a means of temporarily mimicking mental illness and thus producing a model for psychosis (Osmond & Smythies, 1952). Research into its treatment applications goes back as far as the late 1950s, when Dr. Humphry Osmond and Abram Hoffer gave doses of LSD to severe alcoholics and found that the substance produced a vivid aware-

ness of personality problems (Mangini, 1998). The researchers speculated that it was able to induce experiences inspiring an alcoholic to dismantle habitual patterns through self-reflection and create a change in self-concept. Similar studies were also conducted, most notably by Ditman and Whittlesley (1959) and O'Reilly and Reich (1962), further inspiring extensive research on LSD and resulting in the publication of thousands of scientific papers and a proliferation of prescriptions to patients for its use in treating psychiatric disorders and promoting personality improvement.

The healing qualities of LSD were believed to be a medical breakthrough, and researchers began exploring the use of psychedelics for a variety of other purposes such as facilitating psychother-

Copyright: © 2016 McDowell. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

apy, enhancing creativity, and studying schizophrenia. It was seen as a promising and idealistic therapeutic method until the research came to a halt in 1967 due to inconsistent empirical data, government interventions, and societal disapproval. Increasing concerns resulted in psychedelics being legally classified as Schedule One controlled substances in the United States, which suggests that LSD has a high potential for abuse and has no accepted medical use. This shut down avenues of research on unanswered questions and hindered future attempts to explore the benefits of the substance (Mangini, 1998). However the medical use of LSD must not be viewed as a treatment for psychiatric disorders generally, but rather as a way to target specific disorders. The goal is not to find a miracle drug that will universally aid all medical issues as was postulated 40 years ago; this only leads to a surge in inconsistent research and contradictory results, all of which will spark controversy and create doubt concerning its healing properties. Expecting unrealistic results acts to discredit moderate or short-term effects, and poorly described trials result in the impression that studies were not well designed thus leading to the dismissal of valid results (Krebs & Johansen, 2012). The medical field needs something above and beyond a quick fix daily medication that temporarily alters neurochemical relations. Psychiatry must start addressing the source of mental illnesses and LSD may have the potential to do that. In light of its therapeutic benefits and low potential for abuse, LSD should be legally reclassified to a Schedule Four substance which suggests a low potential for dependence and accepted medical use in treatment. This would allow researchers to investigate its possible treatment applications and re-open avenues for research concerning its effects on addictions and mental health disorders such as Autism Spectrum Disorder

(ASD), anxiety, and depression.

LSD has promising implications in the mental health field and may be able to treat a multitude of disorders. LSD is a serotonergic agonist that down regulates 5-HT_{2A}, a system which has many implications regarding depressive and anxiety disorders (Buchborn, Schröder, Höllt, & Grecksch, 2014). Similar to antidepressants, LSD drug treatment has the potential to reverse depressive responses and feelings by rebalancing the hippocampal 5-HT₂ and 5-HT_{1A} signaling; in fact, rebalancing serotonin postsynaptic receptor signaling is a common property that many antidepressants share (Buchborn et al., 2014). Furthermore, serotonin released from the median dorsal raphe forebrain primarily binds to postsynaptic 5-HT_{1A} receptors in the hippocampus and limbic system (Baumeister, Barnes, & Giaroli, 2014). This pathway is involved in tolerance and resilience to chronic stressors, allowing one to adapt to and cope with stressful situations. Targeting and stimulating this pathway using LSD has the potential to diminish the severity of, and treat, pervasive disorders such as generalized anxiety disorder, as well as prevent them from occurring in high-risk subjects. A recent study showed psychological improvements over a twelve-month period in anxiety patients given three months of LSD-assisted psychotherapy (Gasser et al., 2015). Other than an initial sense of loss of control, none of the patients reported lasting adverse effects. Furthermore, patient reactions to the LSD treatment involved a positive facilitation of emotions, confrontation with previously unknown anxieties, as well as a restructuring of emotions, situational understandings, and world views (Gasser et al., 2015). This restructuring of emotions and recognition of mood states is key in helping patients suffering from severe depression and anxiety, but may also extend further and have beneficial implications for other mental health

disorders such as Post Traumatic Stress Disorder (PTSD). LSD can alleviate emotional distress, facilitate cognitions such as memory, and reorient the negative biases that prime an individual's mindset (Buchborn et al., 2014). Examining the precise nature of LSD binding effects and the rebalancing of serotonin in specific neural pathways may have drastic therapeutic benefits for mental health disorders and further research is needed.

Another application for which LSD has potential is the psychotherapeutic treatment of alcoholism. LSD is well-known for providing insight into one's own behaviour by allowing the subject to explore new sensory experiences and by giving the individual a deeper meaning and motivation in their lives (Gasser et al., 2015). Such introspection requires patients to observe insights into their problems and the hardships that have led them to rely on alcohol. LSD's profound effects on the mind allows the patient to address deep underlying problems, confront their memories, and meaningfully re-label their past experiences (Sessa, 2014). In randomized controlled trials conducted by Krebs and Johansen (2012), results showed that patients in alcoholic treatment programs given a single dose of LSD displayed a decrease in alcohol misuse. LSD treatment approximately doubled the success rates during the first follow-up. There was a beneficial effect in short-term and medium-term patients after follow-ups, however there was not a statistically significant effect for long-term follow-ups (Krebs & Johansen, 2012). However, this is expected, as it is uncommon for any drug to continue its treatment effects months after a single dose; follow-up admissions of the drug may be necessary to prevent longer-term relapse prevention. This, in combination with other therapies such as cognitive-behavioural therapy and psychosocial interventions, can further decrease a patient's risk of relapse. Krebs and Johansen

(2012) also found that it was not uncommon for patients to become much more self-accepting, optimistic, and feel as if they were given a new positive energy after a period of illness or sadness. Thus there is reason to believe that the legal reclassification of LSD to a Schedule Four controlled substance can serve as an adequate solution to alcohol dependence, allowing patients treated with LSD to obtain the confidence and courage to conquer current addictions and face future problems.

There is also evidence to indicate that LSD could be helpful in the treatment of tobacco addiction. Advancing the scientific study of LSD for this purpose may hold the same promise found in its application to alcoholism. Five million mortalities world-wide are smoking related, which highlights an urgent need for effective treatments (Johnson, Garcia-Romeu, & Cosimano, 2014). Using psilocybin, a naturally occurring psychedelic produced from mushrooms, Johnson et al. (2014) observed smoking cessation rates of 80% throughout the following 10 weeks after psilocybin treatment. The majority of participants also associated their cessation experience with drastic behavioural changes. Participants labeled three key mechanisms by which psilocybin aided them in quitting smoking, including their changing orientation toward the future with long-term benefits outweighing immediate desire, changing priorities and values, and a strengthening of their own belief in their ability to quit (Johnson et al., 2014). As noted previously, these are the same goals of LSD treatment, so it is entirely possible that treatment with LSD, a substance functionally similar to psilocybin, may also result in the cessation of smoking. Other drug addictions of this nature may also benefit from LSD administration. For example, after only a single dose of LSD in trials for heroin addiction, results showed a significantly lower rate

of relapse in the LSD group compared to the control group at three, six, nine, and twelve months post treatment (Savage & McCabe, 1973, as cited in Krebs & Johansen, 2012). A possible concern that may be raised in the context of using LSD to treat addictions is the use of one psychoactive drug to replace the dependence of another drug, therefore creating an entirely new addiction. However, LSD is a 5-HT_{2A} agonist which is associated with fast elimination and prompt tolerance as a result of receptor downregulation, thus it is not found to produce compulsive drug-seeking and addiction (Nichols, 2014; Johnson et al., 2014). Due to this tolerance, it has been argued that dependence and frequent use are much less likely compared to other drugs (Fantegrossi, Woods, & Winger, 2004). Replacing a harmful and dangerous drug with a different controlled, non-addictive substance can serve as a way to prevent or alleviate serious drug problems and in turn save lives. The evidence regarding LSD is compelling enough to pursue this method of treatment and dated societal biases and misguided beliefs should not impede potential breakthroughs in the treatment of addiction.

In addition to its therapeutic benefits, LSD is known to heighten creativity which has many potential applications, especially to the clinical field (Sessa, 2008). Creative enhancement involves an increase in consciousness, altering perceptions, changing an individual's emotions or self-concept, and the elimination of self-imposed restrictions, all of which are involved in the psychological experience induced by LSD in humans (Baumeister et al., 2014). The medical field is poised to benefit greatly from these enhancements in creativity, specifically in the case of Autism Spectrum Disorder (ASD; Sessa, 2008). Creative innovation is largely dependent on greater cooperation and communication between brain regions that may not be usually strongly

connected (Heilman, Nadeau, & Beversdorf, 2003; Beaty et al., 2014). As patients with autism usually have deficits in the social domain, such as difficulty seeing the connectivity between people and objects (Lai, Lombardo, & Baron-Cohen, 2014), it is postulated that LSD will allow patients to find new meaning and associations between objects and people. Allowing this enhanced communication between brain areas in such patients may allow for a better understanding of ASD and the brain in general. Furthermore, Klintwall et al. (2011) postulated that altered sensory modalities contribute to autistic symptoms, and a mechanism that targets specific sensory input has potential to change the patient's behaviour. Thus, creative enhancement by the psychedelic experience is one area that may have potential to advance not only the study of ASD, but also the study of neuroscience more generally by furthering the research on both the brain and the mind. Despite this, the credibility and medical benefit of LSD's potential for creative enhancement is often dismissed due to social stigma and perceived negative image of psychedelics. Therefore, in order to preserve the credibility of research on LSD as a legitimate treatment for clinical problems, legalization should allow research to be carried out in a controlled manner while maintaining restrictions on its public access.

Although many would argue that LSD is a harmful and dangerous drug, it is not nearly as harmful as many other freely available substances (Nutt et al., 2007). Alcohol, by comparison, is said to cause more harm than any other drug and results in 5% of global disability rates and 4% of total global mortality rates (Rehm, Mathers, & Popova, 2009). Following this, some may then argue that alcohol is more dangerous simply because it is more prevalent, and the increased use and availability of LSD would cause similar harmful results. However un-

like alcohol, a widely accepted yet fatal substance, it is believed that LSD has no critical dose acting directly on the body that would cause fatality. It is also important to note that LSD would not become widely available, and instead be limited to the scientific and medical community. In addition, benzodiazepines, which are currently used to treat anxiety, are commonly prescribed yet can result in severe sedation, impaired cognitive and psychomotor skills, as well as severe withdrawal leading to insomnia, muscle spasms, tension, and even death (Lader, 2014; Neale & Smith, 2007). While no drug or medical intervention is completely without risk, LSD is, statistically, very safe (Sessa, 2014), and perhaps even safer than some psychoactive substances currently used to treat mental illness. In light of this, it is clear that the current legal classifications of LSD are not based on scientific evidence and are in need of re-evaluation.

Additional concerns regarding the usage of LSD center around possible side effects such as hallucinogen persisting perception disorder (HPPD). HPPD is a condition characterized by re-experiencing one or more of the perceptual symptoms causing distress and impairment in significant areas of functioning (Lerner et al., 2003). Other studies have also suggested that the prolonged use of LSD induces anxious mood, tension, paranoia, and suicide attempts (Cohen, 1960). However, concerns of this nature are based on misleading studies conducted from the 1960s to the 1980s intended to deter use of the substance. Most claims positing the harms of psychedelics should be evaluated with caution, as they are based on case reports or theoretical assumptions (Johansen & Krebs, 2015). In a recent population study of 130,000 adults in the United States, Johansen and Krebs (2015) failed to find a link between psychedelic drugs (such as LSD, psilocybin, and mescaline) and mental health problems. Specifically,

there were no significant associations between lifetime use of psychedelics and mental health issues, psychological distress, anxiety, depression, or suicidal thoughts. Psychedelics are not known to harm the brain or other body organs, result in addiction and compulsion, disrupt psychosocial functioning, or cause severe adverse effects (Halberstadt, 2011). It can then be hypothesized that in a controlled, monitored environment, the possible negative outcomes of repeated LSD usage can be avoided. However, due to legal restraints, the research community is limited in its ability to investigate and dispute concerns regarding the postulated side effects. Such restrictions on this research reflect misguided and outdated beliefs about the dangers of LSD.

Depression, addiction, and other clinical disorders present a serious challenge to society and the misinformation on both illegal drugs, and mental illness, is overwhelming. LSD offers a novel alternative that can generate lasting benefits while avoiding harsh side effects, such as sedation and dysphoria, that accompany many medications used today. Physical and mental health barriers can potentially be overcome with the legalized medical and therapeutic use of LSD, allowing patients to improve their emotional state, confidence and trust in given situations, and the ability to cope by enabling access to the thoughts and feelings that are usually excluded from consciousness. However, due to the historical methodological problems and the illegality of hallucinogens, evidence for LSD's therapeutic benefits are still largely unevaluated and its known potential is highly limited. The rationale behind the Schedule One classification of LSD appears to be misguided and illogical in light of the evidence for its potential benefits and relative safety (Baumeister et al., 2014). From a medical and public health perspective, it is difficult to understand the justification for the dismissal of

the medical use of LSD. A major impact on the quality of life for those living with mental health issues and addictions is likely within reach.

References

- Baumeister, D., Barnes, G., Giaroli, G., & Tracy, D. (2014). Classical hallucinogens as antidepressants? A review of pharmacodynamics and putative clinical roles. *Therapeutic Advances in Psychopharmacology*, 4(4), 156-169. doi:10.1177/2045125314527985
- Beaty, R. E., Benedek, M., Wilkins, R. W., Jauk, E., Fink, A., Silvia, P. J., . . . Neubauer, A. C. (2014). Creativity and the default network: A functional connectivity analysis of the creative brain at rest. *Neuropsychologia*, 64, 92-98. doi:10.1016/j.neuropsychologia.2014.09.019
- Buchborn, T., Schröder, H., Höllt, V., & Grecksch, G. (2014). Repeated lysergic acid diethylamide in an animal model of depression: Normalisation of learning behaviour and hippocampal serotonin 5-HT₂ signalling. *Journal of Psychopharmacology*, 28(6), 545-552. doi:10.1177/0269881114531666
- Cohen, S. (1960). Lysergic acid diethylamide: Side effects and complications. *The Journal of Nervous and Mental Disease*, 130(1), 30-40. doi:10.1097/00005053-196001000-00005
- Ditman, K.S. & Whittlesley, J.R.B. (1959). Comparison of the LSD-25 experience and delirium tremens. *AMA Archives of General Psychiatry* 1, 63-73.
- Fantegrossi, W. E., Woods, J. H., & Winger, G. (2004). Transient reinforcing effects of phenylisopropylamine and indolealkylamine hallucinogens in rhesus monkeys. *Behavioural Pharmacology*, 15(2), 149-157. doi:10.1097/00008877-200403000-00007
- Frankel, P. S., & Cunningham, K. A. (2002). The hallucinogen d-lysergic acid diethylamide (LSD) induces the immediate-early gene c-fos in rat forebrain. *Brain Research*, 958(2), 251-260. doi:10.1016/S0006-8993(02)
- Gasser, P., Kirchner, K., & Passie, T. (2015). LSD-assisted psychotherapy for anxiety associated with a life-threatening disease: A qualitative study of acute and sustained subjective effects. *Journal of Psychopharmacology*, 29(1), 57-68. doi:10.1177/0269881114555249
- Halberstadt, A. L., & Geyer, M. A. (2011). Multiple receptors contribute to the behavioral effects of indoleamine hallucinogens. *Neuropharmacology*, 61(3), 364-381. doi:10.1016/j.neuropharm.2011.01.017
- Heilman, KM, Nadeau, SE, Beversdorf, DO (2003) Creative innovation: possible brain mechanisms. *Neurocase*, 9(5), 369-379. doi:10.1076/neur.9.5.369.16553
- Johansen, P., & Krebs T. S. (2015) Psychedelics not linked to mental health problems or suicidal behaviour: a population study. *Journal of Psychopharmacology*, 29(3) 270-279. doi:10.1177/0269881114568039
- Johnson, M. W., Garcia-Romeu, A., Cosimano, M. P., & Griffiths, R. R. (2014). Pilot study of the 5-HT_{2A}R agonist psilocybin in the treatment of tobacco addiction. *Journal of Psychopharmacology*, 28(11), 983-992. doi:10.1177/0269881114548296
- Klintwall, L., Holm, A., Eriksson, M., Carlsson, L. H., Olsson, M. B., Hedvall, Å., . . . Fernell, E. (2011). Sensory abnormalities in autism. *Research in Developmental Disabilities*, 32(2), 795-800. doi:10.1016/j.ridd.2010.10.021
- Krebs, T. S., & Johansen, P. (2012). Lysergic acid diethylamide (LSD) for alcoholism: Meta-analysis of randomized controlled trials. *Journal of Psychopharmacology*, 26(7), 994-1002. doi:10.1177/0269881112439253
- Lader, M. (2014). Benzodiazepine harm: How can it be reduced? *British Journal of Clinical Pharmacology*, 77(2), 295-301. doi:10.1111/j.1365-2125.2012.04418.x
- Lai, M., Lombardo, M. V., & Baron-Cohen, S. (2014). Autism. *Lancet* (London, England), 383(9920), 896-910. doi:10.1016/S0140-6736(13)61539-1
- Lerner, A. G., Gelkopf, M., Skladman, I., Rudinski, D., Nachshon, H., & Bleich, A. (2003). Clonazepam treatment of lysergic acid diethylamide-induced hallucinogen persisting perception disorder with anxiety features. *International Clinical Psychopharmacology*; 18(2), 101-105. doi:10.1097/00004850-200303000-00007

- Mangini, M. (1998). Treatment of alcoholism using psychedelic drugs: A review of the program of research. *Journal of Psychoactive Drugs*, 30(4), 381. doi:10.1080/02791072.1998.10399714
- Neale, G., & Smith, A. J. (2007). Self-harm and suicide associated with benzodiazepine usage. *British Journal of General Practice*, 57(538), 407-408.
- Nichols, D. (2004). Hallucinogens. *Pharmacological Therapy*, 101, 131-18. doi:10.1016/j.pharmthera.2003.11.002
- Nutt, D., King, L. A., Saulsbury, W., Blakemore, C. (2007). Developing a rational scale for assessing the risks of drugs of potential misuse. *Lancet*, 369(9566), 1047-1053. doi:10.1016/S0140-6736(07)60464-4
- O'Reilly, P.O. & Reich, G. (1962). Lysergic acid and the alcoholic. *Diseases of the Nervous System*, 23(6), 331-34.
- Osmond, H. & Smythies, J. (1952). Schizophrenia: A new approach. *Journal of Mental Science*, 98(411), 309-315. doi:10.1192/bjp.98.411.309
- Rehm J, Mathers C, Popova S, et al. (2009). Global burden of disease and injury and economic cost attributable to alcohol use and alcohol-use disorders. *Lancet*, 373(9682), 2223-2233. doi:10.1016/S0140-6736(09)60746-7
- Sessa, B., (2008). Is it time to revisit the role of psychedelic drugs in enhancing human creativity? *Journal of Psychopharmacology*, 22(8): 821-827. doi:10.1177/0269881108091597
- Sessa, B. (2014). Why psychiatry needs psychedelics and psychedelics need psychiatry. *Journal of Psychoactive Drugs*, 46(1), 57-62. doi:10.1080/02791072.2014.877322

The Feeling of Being 'There': Presence and the Role of Virtual Reality as a Research Tool

Alec McLeod

SIMON FRASER UNIVERSITY

This paper provides evidence to suggest that Virtual Reality (VR) technology offers psychology a more ecologically and internally valid research tool than traditional means of research. As generalizations about certain areas of psychological research, such as in neuropsychology, are benefited by this brand of ecological validity, VR has the potential to radically change how research is performed. This paper offers presence, an individual's feeling of being in a virtual environment, as the metric that dictates the realness of experience in VR. A focus on maximizing presence, then, should allow for the most ecologically valid research. Means of increasing an individual's presence are elaborated, and current implementations and future applications of VR for psychology are discussed.

Keywords: virtual reality, presence, ecological validity, internal validity

Since its inception, both in science fiction and in reality, Virtual Reality (VR) technology has been envisioned as an entertainment product; however, in recent years, science has slowly adopted the technology as an experimental and clinical tool. While it has been applied in the treatment of phobias (Klinger et al., 2005), eating disorders (Riva, 2011), and posttraumatic stress disorder (Rothbaum, Hodges, Ready, Graap, & Alarcon, 2001), many areas of psychology have yet to implement VR technology, often preferring traditional paper-and-pen assessment with computerized scoring (Parsons, 2011). Fortunately, many see VR as a viable new tool, offering greater ecological validity without compromising a researcher's experimental control (Loomis, Blascovich, & Beall, 1999; Campbell et al., 2009; Parsons, 2015). Crucial to VR is a measurement of an individual's 'presence' in the Virtual Environment (VE): Presence is the feeling of being

'there', and reflects to what degree an individual feels as though they are actually occupying a real environment. Essentially, then, an increase to presence equates to greater overall effectiveness of VR (Sanchez-Vives & Slater, 2005). Consequently, presence is an imperative factor in determining the ecological validity of VR, and its current and future role as a research tool in psychology.

VR is the simulation of an artificial, or virtual, environment through the aid of specialized hardware. Sitting in front of a desktop display that generates three-dimensional images can be considered VR, though non-immersive, as there is still a peripheral awareness of the direct environment. Conversely, the type of VR discussed here is immersive VR (Slater & Wilbur, 1997), which often uses head-mounted displays (HMDs) paired with head-tracking to create a more realistic experience. This type of VR setup necessitates other hardware,

Copyright: © 2016 McLeod. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

including audio equipment and motion based input devices, as well as computers sufficiently powerful to render a VE. A common drawback is cost (Bowman & McMahan, 2007), but with more off-the-shelf VR products reaching the market and the hands of researchers (Rand, Kizony, & Weiss, 2008), there is potential for the hardware to become less costly and more available in the near future (Desai, Desai, Ajmera, & Mehta, 2014). It is important to note that the level of immersion only objectively reflects the nature of the hardware used, and the experience available to the individual (Slater & Wilbur, 1997). Presence, on the other hand, measures the experience itself.

Presence is the feeling of physically being in a virtual environment rather than where your body is actually situated. Though insufficient psychological research has been performed on the phenomenon itself, presence is arguably the defining metric through which the effectiveness of VR can be assessed (Slater & Wilbur, 1997). Presence is typically assessed subjectively via self-reporting or through questionnaire, though there has been some evidence to suggest that it might be measurable objectively through changes in galvanic skin response (GSR; Lo Priore, Castelnuovo, Liccione, & Liccione, 2003). Presence can also be suggested through activation of the sympathetic nervous system as a response to threats in VR, as in one study, where GSR increased when participants observed their virtual bodies being stabbed (Hägner et al., 2008). In another study, participants suffering from acrophobia (the fear of heights) showed anxiety in VR (Hodges et al., 1995); while presence was not measured, if the participants felt anxiety, logically, they must have felt a degree of presence in the VE. Typically, though, as in the above studies, behavior in VR that is nearly identical to real-world behav-

ior is sufficient to surmise a level of an individual's presence.

While a feeling of presence in VR is imperative, implementation of realistic and presence-inducing VR can be problematic. A typical VR experience is provided to individuals via HMD, with visual information being the primary, and often only source of stimulus; however, it has been recently argued that visual cues are insufficient for creating a cognitive spatial map of the virtual environment (Aghajan et al., 2015). Instead, cues from other modalities, such as through audition or haptic feedback, have been suggested as necessary for the brain to be passably fooled by VR (Ravassard et al., 2013). In fact, in rats, place cells of the hippocampus — a region of the brain involved in memory and station navigation — were found to be much less active if rats were unable to use proprioceptive cues, particularly vestibular cues, to assess position in a virtual environment (Aghajan et al., 2015). Strong feelings of presence can still be achieved during fMRI scans, where the head is fixed, and loud noises are occurring (Hoffman, Richards, Coda, Richards, & Sharar, 2003). Even so, feelings of presence appear to be the greatest when individuals have the ability to move freely and without restriction (Slater & Steed, 2000). Similarly, vestibular motion cues seem to be essential in convincing individuals of the occurrence of real motion, as well as decreasing the sickness sometimes associated with VR use (Harris, Jenkin, & Zikovitz, 1999). As a whole, it is apparent that presence in VR is strongest when movement is both visually and non-visually experienced just as it would be in the real world. Ergo, realistic perceptions of motion in VR are paramount to a realistic and thus presence-inducing experience.

Presence is best maximized by understanding and employing features that increase it. Because of the increased visual detail, some might assume that photo-

realism increases presence, but this does not appear to be the case (Zimmons & Panter, 2003). Likewise, the illusion of depth does not have any tremendous effect on an individual's feeling of presence (Baños et al., 2008). Instead, factors such as bodily representation (Slater & Usoh, 1994) and increased participant agency (Steuer, 1993) have a greater tie to perceived presence. When participants are led to believe that their VE is identical to the real world room they're occupying, they report increased feelings of presence in the VE (Bouchard et al., 2012). Similarly, being persuaded that virtual people encountered in VR exist for real also increases the presence of the participant (Nowak & Biocca, 2003). Taken together, realistic stimuli and persuasion of realism are sufficient to induce perceived realism in VR. Ultimately, then, a perception that VR is occurring believably offers the maximal potential feelings of presence.

Crafting a research environment that is realistic and believable creates potential for realistic participant experience and response; increased realism, then should increase ecological validity, and thus the overall validity of research. Ecological validity is the degree to which the methods and results of experimentation can be generalized to the real-world setting that is being researched. Opinions on the need for greater ecological validity in research are mixed, where some argue that failings or inaccuracies in methodology may not strongly impact results (Diamond, 1997) and that research validity ultimately depends on the kinds of claims being made about the results (Bornstein, 1999). A different pattern emerges from experimentation in neuropsychology, an area of psychology examining the relationship between the brain and behavior. For instance, Chaytor and Schmitter-Edgecombe (2004) analyzed the ecological validity of a sizeable number of neuropsychol-

ogy experiments, finding that many of the tests — such as the Wisconsin Card Sorting Test and the Trail Making Test — were insufficiently related to outcome measures, reducing the potential generalizability of the results. This is particularly unfortunate, as in neuroscience, the activity of particular brain regions can be largely affected by the ecological validity of the measures used (Campbell et al., 2009). Clinically, too, a neuropsychologist's informed judgment of the scope of an impairment can be largely dependent on the ecological validity of the pertaining research (Kieffaber, Marcoulides, White, & Harrington, 2007), though this is not necessarily the case for neuropsychology generally. Fortunately, many assessment tools in neuropsychology are already designed with ecological validity in mind (Robertson, Ward, Ridgeway, & Nimmo-Smith, 1996; Lamberts, Evans, & Spikman, 2010). Thus, greater ecological validity allows for greater overall validity of neuropsychological research and allows for greater generalizability and breadth of potential claims. And, just as with perception of presence in VR, making an individual's experience believable and maximally realistic allows for more veridical behavior and valid measurement.

The creation or recreation of a large ecologically valid experimental environment, while beneficial, can be costly and difficult. However, VR also has the capacity to facilitate and ease internally valid research design. For instance, VR allows for greater and more precise control over participant's environment and stimuli, resulting in greater consistency and computational precision in assessing results (Parsons, 2015). A pivotal advantage of VR over traditional means of research is the ease of control over adjustments to particular variables. For example, a study researching nicotine craving used VR to easily present participants with varying environments

containing either craving-inducing or neutral cues (Bordnick et al., 2004). VR allowed for identical environments to be rendered and presented to each participant, as well as allowing for social interaction to be fulfilled by virtual avatars. This supplanted any need to employ or instruct additional confederates as part of experimentation, and allowed for each participant to receive the same social interaction stimuli. Crucially, the application of VR facilitates experimental design, allowing for easily replicable experimental manipulations, and offers unmatched consistency in terms of experimental execution.

Implementing VR as a valid research tool doesn't necessarily impact results when compared to traditional paper-and-pen means. In the above nicotine study (Bordnick et al., 2004), VR experimentation results match nicotine craving research previously performed through traditional means (Sayette, Martin, Wertz, Shiffman, & Perrott, 2001). In another study, VR was used to measure attention in boys diagnosed with attention deficit-hyperactivity disorder (ADHD) in a virtual classroom (Parsons, Bowerly, Buckwalter, & Rizzo, 2007). As expected, ADHD-diagnosed boys were found to be more distracted than normal boys, as evident in their task-performance and bodily movement; also, the measured results mirrored results from traditional ADHD assessments of the same participants. Here, the virtual environment allowed for better control of potential distractors that might impact a traditional assessment (Parsons et al., 2007). The implementation of VR produces essentially the same or equivalent findings, and of equal importance, effectively erases any biases or errors that might occur as a result of the inclusion of human roles in the research process.

On the whole, VR offers greater validity without compromising control (Loomis et al., 1999), facilitates design (Bord-

nick et al., 2004), and eases replication (Blascovich et al., 2002). But, VR also has benefit in that it extends design beyond what is typically possible for traditional research; for example, VR allows for adjustment to the environment between conditions and in real-time, by altering the environment itself, or adjusting the scale of objects or the individual, or even exaggerating the effects of stimuli (Hodges et al., 1995). This has many benefits, including allowing precise control over exposure to phobic stimuli (Klinger et al., 2005). VR removes the necessity for many human roles in the research process; logically, the fewer human roles, be they as confederate, observer, coder, or otherwise, the less likely a human bias or error could negatively affect results, and the more internally valid those results are likely to be. Because of the digital nature of VR, methodology and complex research environments can be easily shared throughout the research community, ultimately offering greater ease of replication. Thus, while current technology used for neuropsychological assessment might be outdated (Parsons, 2011) and potentially negatively affected by human involvement, VR offers an equivalent, and much more valid tool.

VR has the ability to facilitate a new brand of valid research; furthermore, 'solving' presence, i.e., maximizing the presence felt by individuals in the virtual world, will allow for VR to become the foremost tool of psychological research. Though it has potential to generally benefit psychology (Loomis et al., 1999; Sanchez-Vives & Slater, 2005), it has particular benefit to cognitive psychology, especially in perception (Sanchez-Vives & Slater, 2005), social psychology (Blascovich et al., 2002), and neuropsychology. VR also has tremendous clinical applications, both in assessment (Parsons et al., 2007; Parsons, 2011; Parsons, 2015) and treatment (Hodges et al., 1995; Rizzo & Kim, 2005). In fact, one of the most

striking implementations of VR is in the treatment of PTSD, where the precise recreation of traumatic events in a safe environment is paramount to patient amelioration (Rizzo et al., 2015). Due to the significant incidence rates of PTSD in military personnel internationally, as well as in survivors of terrorist attacks and other traumatic events, VR is and will continue to be an important tool in treatment. And, presence is crucial to its effectiveness.

Be that as it may, there is a tremendous lack of information in the scientific literature on presence, particularly on objective means of measurement. By the same token, there has been almost no neuroscientific research on the neural correlates of presence, though some initial evidence has shown brain activation in areas involved in spatial navigation (Baumgartner, Valko, Esslen, & Jäncke, 2006). By admission, there is some difficulty in simultaneously administering VR while imaging the brain through traditional means, though relatively recent advances in near-infrared spectroscopy are allowing for an easier non-invasive method of neuroimaging while in VR (Kober, Wood, & Neuper, 2013). Furthermore, there has been too little research or analysis directly comparing results of VR research against traditional means of research; while there is increasing evidence that VR experimentation produces results on par with previous findings (Bordnick et al., 2004; Parsons et al., 2007), a larger body of evidence will help to promote further trust in VR. Going forward, greater experimentation is recommended as to thoroughly flesh out the understanding of presence, and thus the underpinnings of VR. As it stands, VR technology is advancing quicker than the research community can accommodate. Though the tools of the trade can be understandably expensive, and the environments difficult to produce, they are not theoretical tools for the near future.

Virtual reality is a tangible tool that is available now, primed and ready to join psychology's arsenal.

References

- Aghajan, Z., Acharya, L., Moore, J., Cushman, J., Vuong, C., & Mehta, M. (2015). Impaired spatial selectivity and intact phase precession in two-dimensional virtual reality. *Nature Neuroscience*, 18, 121-128. doi:10.1038/nn.3884
- Baños, R. M., Botella, C., Rubió, I., Quero, S., García-Palacios, A., & Alcañiz, M. (2008). Presence and emotions in virtual environments: The influence of stereoscopy. *CyberPsychology & Behavior*, 11(1), 1-8. doi:10.1089/cpb.2007.9936
- Baumgartner, T., Valko, L., Esslen, M., & Jäncke, L. (2006). Neural correlate of spatial presence in an arousing and noninteractive virtual reality: an EEG and psychophysiology study. *CyberPsychology & Behavior*, 9(1), 30-45. doi:10.1089/cpb.2006.9.30
- Blascovich, J., Loomis, J., Beall, A., Swinth, K., Hoyt, C., & Bailenson, J. (2002). Immersive virtual environment technology as a methodological tool for social psychology. *Psychological Inquiry*, 13(2), 103-124. doi:10.1207/S15327965PLI1302_01
- Bordnick, S., Graap, K., Copp, H., Brooks, J., Ferrer, M., & Logue, B. (2004). Utilizing virtual reality to standardize nicotine craving research: A pilot study. *Addictive Behaviours*, 29, 1889-1894. doi:10.1016/j.addbeh.2004.06.008
- Bornstein, B. (1999). The ecological validity of jury simulations: Is the jury still out?. *Law and Human Behavior*, 23(1), 75-91. doi:10.1023/A:1022326807441
- Bouchard, S., Dumoulin, S., Talbot, J., Ledoux, A., Phillips, J., Monthuy-Blanc, J., Labonté-Chartrand, G., Robillard, G., Cantamesse, M., & Renaud, P. (2012). Manipulating subjective realism and its impact on presence: Preliminary results on feasibility and neuroanatomical correlates. *Interacting with Computers*, 24, 227-236. doi:10.1016/j.

intcom.2012.04.011

- Bowman, D., & McMahan, R. (2007). Virtual Reality: How Much Immersion Is Enough? *Computer*, 40(7), 36-43. doi:10.1109/MC.2007.257
- Campbell, Z., Zakzanis, K., Jovanovski, D., Joordens, S., Mraz, R., & Graham, S. (2009). Utilizing virtual reality to improve the ecological validity of clinical neuropsychology: an fMRI case study elucidating the neural basis of planning by comparing the Tower of London with a three-dimensional navigation task. *Applied Neuropsychology*, 16(4), 295-306. doi:10.1080/09084280903297891
- Chaytor, N., & Schmitter-Edgecombe, M. (2003). The ecological validity of neuropsychological tests: A review of the literature on everyday cognitive skills. *Neuropsychology review*, 13(4), 181-197. doi:10.1023/B:NERV.0000009483.91468.fb
- Desai, P., Desai, P., Ajmera, K., & Mehta, K. (2014). A Review Paper on Oculus Rift - A Virtual Reality Headset. *International Journal of Engineering Trends and Technology*, 13(4), 175-179. Retrieved from <https://arxiv.org/abs/1408.1173>
- Diamond, S. (1997). Illuminations and shadows from jury simulations. *Law and Human Behavior*, 21(5), 561-571. doi:10.1023/A:1024831908377
- Hägni, K., Eng, K., Hepp-Reymond, M., Holper, L., Keisker, B., Siekierka, E., & Kiper, D. (2008). Observing virtual arms that you imagine are yours increases the galvanic skin response to an unexpected threat. *PLoS one*, 3(8), e3082. doi:10.1371/journal.pone.0003082
- Harris, L., Jenkin, M., & Zikovitz, D. (1999). Vestibular cues and virtual environments: choosing the magnitude of the vestibular cue. In: *IEEE Int Conf on Virtual Reality 1*, 229-236. doi:10.1109/VR.1999.756956
- Hodges, L., Kooper, R., Meyer, T., Rothbaum, B., Opdyke, D., De Graaff, J., Williford, J., & North, M. (1995). Virtual environments for treating the fear of heights. *Computer*, 28(7), 27-34. doi:10.1109/2.391038
- Hoffman, H., Richards, T., Coda, B., Richards, A., & Sharar, S. (2003). The Illusion of Presence in Immersive Virtual Reality during an fMRI Brain Scan. *CyberPsychology & Behaviour*, 6(2), 127-131. doi:10.1089/109493103321640310
- Kieffaber, P., Marcoulides, G., White, M., & Harrington, D. (2007). Modeling the ecological validity of neurocognitive assessment in adults with acquired brain injury. *Journal of Clinical Psychology in Medical Settings*, 14(3), 206-218. doi:10.1007/s10880-007-9075-6
- Klinger, E., Bouchard, S., Légeron, P., Roy, S., Lauer, F., Chemin, I., & Nugues, P. (2005). Virtual Reality Therapy Versus Cognitive Behavior Therapy for Social Phobia: A Preliminary Controlled Study. *Cyberpsychology & Behavior*, 8(1), 76-88. doi:10.1089/cpb.2005.8.76
- Kober, S. E., Wood, G., and Neuper, C. (2013). Measuring brain activation during spatial navigation in virtual reality: a combined EEG-NIRS study. In S. Trautman & F. Julien (Eds.), *Virtual Environments: Developments, Applications and Challenges* (pp. 1-24). New York, NY: NOVA Publisher.
- Lamberts, K., Evans, J., & Spikman, J. (2010). A real-life, ecologically valid test of executive functioning: The executive secretarial task. *Journal of Clinical and Experimental Neuropsychology*, 32(1), 56-65. doi:10.1080/13803390902806550
- Lo Priore, C., Castelnuovo, G., Liccione, D., & Liccione, D. (2003). Experience with V-STORE: considerations on presence in virtual environments for effective neuropsychological rehabilitation of executive functions. *CyberPsychology & Behavior*, 6(3), 281-287. doi:10.1089/109493103322011579
- Loomis, J., Blascovich, J., & Beall, A. (1999). Immersive virtual environment technology as a basic research tool in psychology. *Behavior Research Methods, Instruments, & Computers*, 31(4), 557-564. doi:10.3758/BF03200735

- Nowak, K., & Biocca, F. (2003). The Effect of the Agency and Anthropomorphism on Users' Sense of Telepresence, Copresence, and Social Presence in Virtual Environments. *Presence*, 12(5), 481-494. doi:10.1162/105474603322761289
- Parsons, T. (2011). Neuropsychological Assessment Using Virtual Environments: Enhanced Assessment Technology for Improved Ecological Validity. In S. Brahmam & L.C. Jain (Eds.), *Adv. Comput. Intell. Paradigms in Healthcare*, 6, pp. 271-289. doi:10.1007/978-3-642-17824-5_13
- Parsons, T. (2015). Ecological Validity in Virtual Reality-Based Neuropsychological Assessment. *Encyclopedia of Information Science and Technology* 2015, 1006-1015. doi:10.4018/978-1-4666-5888-2.ch095
- Parsons, T., Bowerly, T., Buckwalter, J., & Rizzo, A. (2007). A controlled clinical comparison of attention performance in children with ADHD in a virtual reality classroom compared to standard neuropsychological methods. *Child Neuropsychology*, 13(4), 363-381. doi:10.1080/13825580600943473
- Rand, D., Kizony, R., & Weiss, P. (2008). The Sony PlayStation II EyeToy: low-cost virtual reality for use in rehabilitation. *Journal of Neurologic Physical Therapy*, 32(4), 155-163. doi:10.1097/NPT.0b013e31818ee779
- Ravassard, P., Kees, A., Willers, B., Ho, D., Aharoni, D., Cushman, J., Aghajan, Z., & Mehta, M. (2013). Multisensory Control of Hippocampal Spatiotemporal Selectivity. *Science*, 340, 1342-1346. doi:10.1126/science.1232655
- Riva, G. (2011). The Key to Unlocking the Virtual Body: Virtual Reality in the Treatment of Obesity and Eating Disorders. *Journal of Diabetes Science and Technology*, 5(2), 283-292. doi:10.1177/193229681100500213
- Rizzo, A., Cukor, J., Gerardi, M., Alley, S., Reist, C., Roy, M., Rothbaum, B., & Difede, J. (2015). Virtual Reality Exposure for PTSD Due to Military Combat and Terrorist Attacks. *Journal of Contemporary Psychotherapy*, 45(4), 255-264. doi:10.1007/s10879-015-9306-3
- Rizzo, A., & Kim, G. (2005). A SWOT analysis of the field of virtual reality rehabilitation and therapy. *Presence*, 14(2), 119-146. doi:10.1162/1054746053967094
- Robertson, I., Ward, T., Ridgeway, V., & Nimmo-Smith, I. (1996). The structure of normal human attention: The Test of Everyday Attention. *Journal of the International Neuropsychological Society*, 2(06), 525-534. doi:10.1017/S1355617700001697
- Rothbaum, B., Hodges, L., Ready, D., Graap, K., & Alarcon, R. (2001). Virtual reality exposure therapy for Vietnam veterans with posttraumatic stress disorder. *Journal of Clinical Psychiatry*, 62(8), 617-622. doi:10.4088/JCP.v62n0808
- Sanchez-Vives, M., & Slater, M. (2005). From presence to consciousness through virtual reality. *Nature*, 6, 322-339. doi:doi:10.1038/nrn1651
- Sayette, M., Martin, C., Wertz, J., Shiffman, S., & Perrott, M. (2001). A multi-dimensional analysis of cue-elicited craving in heavy smokers and tobacco chippers. *Addiction*, 96(10), 1419-1432. doi:10.1046/j.1360-0443.2001.961014196.x
- Slater, M., & Steed, A. (2000). A virtual presence counter. *Presence: Teleoperators and Virtual Environment*, 9, 413-434. doi:10.1162/105474600566925
- Slater, M., & Usoh, M. (1994). Representations systems, perceptual position, and presence in immersive virtual environments. *Presence: Teleoperators and Virtual Environments*, 2 (3), 221-233. doi:10.1162/pres.1993.2.3.221
- Slater, M., & Wilbur, S. (1997). A framework for immersive virtual environments (FIVE): Speculations on the role of presence in virtual environments. *Presence: Teleoperators and Virtual Environments*, 6, 603-616. doi:10.1162/pres.1997.6.6.603
- Steuer, J. (1993). Defining Virtual Reality: Dimensions Determining Telepresence. *Journal of Communication*, 42(4), 73-93.

10.1111/j.1460-2466.1992.tb00812.x

Zimmons, P., & Panter, A. (2003). The influence of rendering quality on presence and task performance in a virtual environment. In: *Virtual Reality, 2003. Proceedings.* IEEE, 293-294. doi:10.1109/VR.2003.1191170

Against the Grain: Diet and Alzheimer's Disease

Nancy Yang

SIMON FRASER UNIVERSITY

Alzheimer's disease (AD) is a progressive neurological disorder characterized by neurodegeneration and loss of cognitive functions. Although its etiology is not yet clear, evidence suggests that AD, diabetes, and obesity may share a common pathophysiology of disordered insulin signaling. Currently, there is growing evidence that hyperinsulinemia may play a key role in the development of AD, with some researchers dubbing it as "type 3 diabetes". This paper addresses the recent increasing incidence of AD by adopting an interdisciplinary approach that unites anthropological, biological, and psychological research to create a holistic understanding of how an evolutionary mismatch between our ancestral and current environment contributes to development of lifestyle diseases. Lastly, this paper discusses the theoretical implications of using nutritional therapy to treat cognitive symptoms of AD.

Keywords: Alzheimer's disease, insulin, carbohydrates, fat, diet, evolution

Alzheimer's disease (AD) is a neurodegenerative disease marked by impaired memory and cognitive deficits (Bird, 2014). Described as the pandemic of the 21st century (Jellinger, 2006), statistics indicate that around 24 million people in the world were afflicted with dementia in 2001, with predictions that numbers would rise to 42.3 million in 2020 and 81.1 million by 2040 (Ferri et al, 2005). The increasing prevalence of AD and an aging population has led many western countries to prioritize it as a public health concern (Weiler, 1987).

Worryingly, statistics on other lifestyle diseases such as type 2 diabetes (T2D) and obesity are also rising. According to Statistics Canada (2016), approximately two million Canadians have (T2D) in 2014, an 8.4% increase from 2010. More than 14 million of adult Canadians are also either overweight or obese, an estimated 8% increase from 2010 (Statistics Canada, 2016). AD is highly comorbid

with both diabetes and obesity (Pro-fenno, Porsteinsson, & Faraone, 2010). Patients with T2D who have a history of multiple hypoglycemic episodes are also at an increased risk of developing dementia later in life (Whitmer, Karter, Yaffe, Quesenberry, & Selby, 2009). Metabolic syndrome, a set of risk factors for cardiovascular disease and diabetes, is also associated with late-onset dementia and AD (Frisardi et al, 2010). Currently, metabolic syndrome is diagnosed as having three of the following symptoms: High waist circumference, high plasma triglycerides, high fasting glucose, low high-density lipoprotein cholesterol, and high blood pressure (Alberti, Zimmet, & Shaw, 2005).

Notably, incidence of metabolic syndrome was relatively rare or almost absent in hunter-gather societies that followed a traditional diet (Cordain et al, 2005; Schaeffer, 1971; Trowell & Burkitt, 1980). Historical data indicates that such

Copyright: © 2016 Yang. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

cultures were also free from common maladies such as dental cavities, myopia, gall bladder disease, acne, obesity, and cancer that were prevalent in European societies at the time (Cleave, 1974). Early observations classified such symptoms under the umbrella of “saccharine disease”, noted for its association with a diet rich in refined starches and sugar (Cleave, 1974). Subsequent researchers adopted the term “syndrome X” or “diseases of civilization” to address the cluster of symptoms that were especially prevalent in western civilizations but relatively rare in traditional societies (Burkitt, 1973; Cleave, 1974; Reaven & Laws, 1999). Although the definition of metabolic syndrome has fluctuated over the years, its connection to a westernized diet remains robust. According to Schaeffer (1971), the development of diseases of civilization was directly associated with the westernization of dietary habits.

In light of both old and new research that invites insight into traditional lifestyle of hunter-gatherer societies, this paper proposes that the primary contribution to the increasing prevalence of AD and its sequelae of metabolic abnormalities is an evolutionary mismatch between our ancestral and modern food environment. Currently, research has investigated diabetes, obesity, and AD as separate but related constructs. This paper proposes that a common pathophysiology of dysregulated insulin signaling may unite each disease as different points on a single continuum of hyperinsulinemic disorders. Lastly, this paper discusses the therapeutic implications of using a low-carbohydrate diet to treat AD and its comorbid disorders.

Insulin and Diseases of Civilization

Lifestyle disease such as diabetes, obesity, and AD often occur sequentially and share a common pathophysiology

of disordered insulin signaling. Insulin is a key hormone responsible for the partitioning of nutrients and energy (Ludwig, 2002). The pancreas releases insulin upon ingestion of a meal. There are many factors that determine how much insulin is secreted, one of which being the glycemic load of foods (Ludwig, 2002). The higher the glycemic load, the steeper the blood sugar rises, and more insulin secreted to bring it back down. Regular consumption of high glycemic load carbohydrates have been linked to increased risk for metabolic disorders such as: Cardiovascular disease (Liu et al, 2000), T2D (Krishnan et al, 2007), and obesity (Bell & Sears, 2003).

According to Cordain, Eades and Eades (2003), peripheral insulin resistance is the first stage in the development of T2D. T2D is an endocrine disorder featuring progressive deterioration of carbohydrate metabolism, usually over several years (Weyer, Bogardus, Mott, & Pratley, 1999). Researchers propose that a defect in insulin signaling marks the early stages of T2D (Pimenta et al, 1996), but the lack of longitudinal studies have made it difficult to pinpoint the exact temporal sequence of such deterioration (Weyer, Bogardus, Mott, & Pratley, 1999). Peripheral insulin resistance is the physiological condition in when peripheral tissues such as muscles, adipose tissues, and liver become unresponsive to insulin signaling. One possible cause for peripheral insulin resistance is insulin receptor down-regulation in response to exposure to high concentrations of serum insulin (Khan & Flier, 2000), or hyperinsulinemia. To compensate for receptor down-regulation, the pancreas secretes additional insulin to bring blood sugar back down (Cordain, Eades, & Eades, 2003). Although the organism will maintain normal level of blood sugar as long as the pancreas is able to maintain this extra output of insulin, this state of “com-

pensatory hyperinsulinemia” (Cordain, Eades, & Eades, 2003) can lead to many metabolic abnormalities distributed throughout the body.

One consequence of hyperinsulinemia may be abnormal adipose growth in genetically susceptible individual. Obesity is a metabolic disorder characterized by excessive accumulation of fat tissue. Research indicates that patients with obesity also have elevated levels of basal insulin and exaggerated insulin response to food intake (Grey & Kipnis, 1971). As both obesity and T2D feature abnormal insulin signaling, the two may be sequentially related. In other words, obesity is the disorder in which adipose tissues remain insulin sensitive and grow disproportionately in response to elevated insulin levels. In turn, T2D is the latter stage in which insulin receptors in the peripheral tissues have downregulated to the effects of chronic hyperinsulinemia; the pancreas ceases to maintain the additional output of insulin, leaving blood sugar level pathologically elevated. Notably, both diabetes and obesity are comorbid with Alzheimer’s disease (Profenno, Porsteinsson, & Faraone, 2010). People with abdominal obesity are three times more likely to develop dementia later in life, compared to people with a low waist circumference (Whitmer et al, 2008). As abdominal obesity is a marker for both glucose intolerance and insulin resistance (Després & Lemieux, 2006), this suggests that these three disorders may share a common causal mechanism that is also temporally related.

Chronic hyperinsulinemia may exert effects beyond diabetes and obesity. The brain uses glucose as its primary fuel source. The brain is also an insulin-sensitive organ, with distributed insulin receptors maintaining glucose metabolism and neuronal growth (Frölich et al, 1998). According to Craft and Watson (2004), peripheral insulin resistance may

affect the functioning of the central nervous system. Additional research found that insulin crosses the blood-brain-barrier (Reinhardt & Bondy, 1994) and is involved in cognition, learning, memory (Biessels, Bravenboer, & Gispen, 2004). One connection between hyperinsulinemia and AD lies in the insulin-degrading enzyme (IDE) (Qiu & Folstein, 2006). According to Qiu and Folstein (2006), IDE is an enzyme that works on two substrates: insulin and amyloid- β peptide (A β). As amyloid- β peptide is neurotoxic, the role of IDE is to regulate its levels in the brain’s neuronal and microglial cells (Qiu & Folstein, 2006). Qiu and Folstein hypothesized that hyperinsulinemia may promote pathogenesis of AD by disrupting the activity of IDE, as both insulin and A β competes for IDE. Further research points to that metformin, a common insulin-sensitizing drug used in T2D, improve cognitive symptoms in patients with AD (Alagiakrishnan, Sankaralingam, Ghosh, Mereu, & Senior, 2013).

Lastly, there is evidence showing that risk of cognitive decline does not only increase in patients with T2D, it also increases in patients with pre-diabetes and metabolic syndrome (Luchsinger, Tang, Shea, & Mayeux, 2004). One study on patients with type 1 diabetes found an inverse correlation between the number of hypoglycemic episodes and performance on cognitive tests such as reaction time (Langan, Deary, Hepburn, & Frier, 1991). As the brain depends on a steady supply of glucose transported through the blood-brain-barrier, maintaining glycemic homeostasis may be instrumental in preventing cognitive deficits commonly seen in patients with AD.

Diet and Evolution

Regular consumption of refined grains and sugar is a dietary anomaly for most of human evolutionary history. Indeed,

on an evolutionary time scale, the practice of agriculture and animal husbandry was introduced as recently as 10 000 years ago, an “eyeblick” compared to five to seven million years of hominin evolution (Wills, 2008, p.50). Food processing techniques that enabled the mass-production of sugar were introduced as recently as the Industrial Revolution (Yudkin, 1972). Indeed, evidence indicates that for most of human existence, sustenance was largely limited to low-glycemic foods such as wild plants and animals (Cordain et al, 2005).

In the human ancestral environments, insulin spikes are an adaptive response for appropriating energy storage, and this would have been a self-regulating cycle as most traditional diets featured a limited range of low-carbohydrate foods. Sugar intake would also have been limited to seasonal fruits, its glycemic impact reduced by fiber. Modern food processing techniques have disrupted this self-limiting cycle, making it possible to access sugar year-round in increasing quantities (Yudkin, 1972). To date, sugar and refined starches take up 36% of the calories consumed by the average American (Cordain, Eades, & Eades, 2003). Data indicates that sugar consumption in the U.S. had increased by 64% from 1909 to 1999 (Gerritor & Bente, 2002) – an unprecedented amount that would have been unthinkable to our hunter-gatherer ancestors.

Indeed, contrast to the “short, nasty, brutish” adage that has plagued the popular description of ancestral humans, paleontology research shows that most pre-agricultural societies lived on a diet that was ample in nutrients, such as iron, zinc, magnesium, and folate, and exhibited little to no chronic diseases observed in contemporary Western society (Cordain et al, 2002). Symptoms of metabolic syndrome were rare or entirely absent in native cultures who consumed a traditional low-carb, high-

fat diet (Cleave, 1973; Fouche, 1923; Schaeffer, 1971). Indeed, anthropological research on the nomadic group Masai indicated that they enjoyed good health despite their largely carnivorous diet of high-fat diet of milk, blood, and meat (Orr & Gilks, 1931). The Eskimos, whose diet based on blubber and animal meat, were also free from symptoms of diabetes or coronary heart disease (Cleave, 1974). Incidence of cancer was also extremely rare in the native African populations that consumed a traditional, hunter-gatherer diet (Fouche, 1923). Symptoms of physical deterioration did not emerge until the native population settle in colonized areas and adopted a westernized diet predominant in flour and sugar (Schaeffer, 1971).

In contrast to modern dietary guidelines that recommend a low fat, plant-based diet to prevent age-related cognitive decline (Barnard et al, 2014), Crawford (1992) observed that both fat and protein were imperative for the massive growth in brain size during human evolution. Other research indicated that energy derived from meat and fat was important for maintaining the higher metabolic costs of bigger brains (Leonard, Robertson, Snodgrass, & Kuzawa, 2003; Aillo & Wheeler, 1995). Myelin is the fatty sheath that wraps around nerve cells to speed up neurotransmission. High levels of cholesterol are critical for myelin growth and maintenance (Saher et al, 2005). The most bioavailable sources of dietary cholesterol are in animal foods, such as egg yolks and liver (USDA Food Composition Database, 2016). Zinc, a trace mineral found predominately in animal foods, is also critical for normal neuronal development during gestation (Wallwork, 1986). This suggests that for most of human evolutionary history, an animal-based diet was integral in the evolution and development of complex human nervous systems.

In addition, a comparative anthropo-

logical analysis on the Masai and Kikyu tribe also revealed the former, whose diet was mainly carnivorous, consisted of relatively high intake of protein and calcium, averaged five inches taller than the Kikyu tribe, whose diet was mainly vegetarian, and subsisted on millet, legumes, and roots such as plantains (Orr & Gilks, 1931). Other research on hunter-gatherer diets indicates that despite geographic variation in carbohydrate ratios – ranging from 30% in tropical areas and 15% in high altitudes – they were all markedly lower than the 60% ratio recommended today (Strohl & Han, 2011). In sum, this suggests that the displacement of dietary protein and fat by grains and sugar in the westernized diet may have played a key role in the development of lifestyle diseases commonly observed today.

One common controversy in the relationship of AD and diet is the role of fat intake. When it comes to the association between diet and AD, the lipid-hypothesis has its evidence largely based on rodent models where high-fat diets is used to induce brain inflammation (Morrison et al, 2010; Panthan et al, 2008; Pistell et al, 2010). One limitation of contemporary AD research is the use of rodent models to simulate the effects of high-fat consumption in humans. Although animal models provide valuable insight into how diseases emerge, humans and mice have diverged considerably in their evolutionarily natural diets. Whereas humans have evolved to a mixed diet of protein and fat, rodents are opportunistic omnivores that have evolved to a high-carb diet of grains and cereal (Clark, 1982). As such, the dietary recommendations drawn from such experiments are limited in its application to humans.

Furthermore, research on human participants has shown that a low-carb diet provides an effective strategy for treating metabolic syndrome (Volek &

Feinman, 2005). Because carbohydrates is one of the most insulinogenic macronutrient out of the three (Kopp, 2003), its reduction can help improve insulin sensitivity (Volek & Feinman, 2005). A low-carb diet was also more effective than a low-fat diet in inducing weight loss in patients with obesity (Samaha et al, 2003; Yancy, Olsen, Guyton, Bakst, & Westman, 2004). A low-carb, high fat ketogenic diet also holds promise for treating a variety of neurological disorders, including but not limited to dementia, epilepsy, and Alzheimer's disease (Rho & Stafstrom, 2012). The classic ketogenic diet is a high-fat diet with a 4:1 ratio of fat to carbohydrates, but therapeutic benefits were found with less restrictive forms such as a modified Atkins diet (Dhamija, Eckert, & Wirrell, 2013). The ketogenic diet works by restricting carbohydrates intake, inducing the body to enter a state of ketosis in which ketone bodies become the predominant energy source (Rho & Stafstrom, 2012). A ketogenic diet has found to improve memory performance in patients with AD (Reger et al, 2004). Although it is not yet clear how the ketogenic diet works, some possible mechanisms may involve ketone bodies' modulation of potassium channels (Ma, Berg & Yellen, 2007), and glutamate metabolism (Yudkoff et al, 2004).

Conclusion

The classic adage goes "Nothing in biology makes sense except in light of evolution" (Dobzhansky, 2013, p. 87). In other words, the use of evolutionary theory as a meta-framework can reveal important insights regarding human nutrition and etiology of diseases. The theory of evolution can be the "universal acid" (Dennet, 1995) that erodes the dogmatic barriers between disparate fields of scientific research, explaining ultimate causations that would not be possible otherwise. In light of new

research that has emerged over the last couple of decades, evidence suggests that hyperinsulinemia is the central component that underlies many of the chronic diseases associated with metabolic syndrome. Thus, it may be necessary to revise current nutrition guidelines to promote higher intake of fat in its natural forms, such as butter, cream, and coconut oil, in favour of processed carbohydrate such as grains, cereal, and bread.

Such policy revisions will be difficult to implement for four reasons. First, it requires a substantial revision of government nutritional guidelines that have become entrenched as official dogma over the last few decades. At the time of this writing, the current Canadian food guide is still recommending a diet based on a foundation of grains and cereals. Although a complete reversal may be unlikely, it may be helpful to increase public awareness about the glycemic index and empower them to make healthier choices.

Secondly, additional longitudinal studies may be necessary for fully investigating the causal links between diabetes, obesity, and AD. This methodological design may be problematic due to several reasons. Longitudinal studies are often costly and time-expensive. In addition, such study designs are vulnerable to volunteer bias – that is, patients with the most severe symptoms may be too sick to participate, thus leaving researchers to underestimate the potential relationship between T2D, obesity, and AD.

Thirdly, this would also require the medical system to shift toward a preventative and integrated model of care. Possible psychological interventions may include periodical cognitive screening for high-risk individuals, such as patients with T2D (Whitmer et al, 2008). Such measures ensure that patients in the prodromal phase of AD can be de-

tected and benefit from early treatment. Other possibilities include integrated healthcare settings where psychologists, physicians, and dieticians work together to provide continuity of care.

Lastly, this would require psychology to shift toward a medical model in its practice. In other words, psychological interventions such as CBT may benefit from incorporating pharmacological and nutritional therapy in treating patients. Specifically, the use of CBT to treat anxiety and depression in patients with AD (Spector et al, 2014; Walker, 2004) may yield limited results as it neglects the physiological aspects of a diseased nervous system. For example, patients with uncontrolled hypoglycemia experience symptoms of anxiety and negative rumination (Wredling et al, 1992), not unlike a patient with generalized anxiety disorder. Such patients may benefit from using nutritional therapy to correct the underlying blood sugar fluctuations, rather than solely using CBT to target rumination symptoms. Of particular interest is also the connection between endogenous insulin resistances in patients with psychotic depression (Okamura et al, 2000). This suggests that psychological interventions that solely target the abnormal thought patterns and emotions associated with brain diseases, such as AD, may be insufficient for a therapeutic approach. Possible future intervention may include the addition of nutritional or pharmacological therapy to CBT for better patient outcomes.

References

- Aiello, L., & Wheeler, P. (1995). The expensive-tissue hypothesis: The brain and the digestive system in human and primate evolution. *Current Anthropology*, 36(2), 199-221. Retrieved from <http://www.jstor.org/stable/2744104>
- Alagiakrishnan, K., & Sclater, A. (2012). Psychiatric disorders present-

- ing in the elderly with type 2 diabetes mellitus. *The American Journal of Geriatric Psychiatry*, 20(8), 645-652. doi: 10.1097/JGP.0b013e31823038db
- Alagiakrishnan, K., Sankaralingam, S., Ghosh, M., Mereu, L., & Senior, P. (2013). Antidiabetic drugs and their potential role in treating mild cognitive impairment and Alzheimer's disease. *Discovery Medicine*, 16(90), 277-286. Retrieved from <http://www.discoverymedicine.com/Kannayiram-Alagiakrishnan/2013/12/05/antidiabetic-drugs-and-their-potential-role-in-treating-mild-cognitive-impairment-and-alzheimers-disease/>
- Alberti, K. G. M., Zimmet, P., & Shaw, J. (2005). The metabolic syndrome—A new worldwide definition. *The Lancet*, 366(9491), 1059-1062. [http://dx.doi.org/10.1016/S0140-6736\(05\)67402-8](http://dx.doi.org/10.1016/S0140-6736(05)67402-8)
- Arvanitakis, Z., Wilson, R. S., Bienias, J. L., Evans, D. A., & Bennett, D. A. (2004). Diabetes mellitus and risk of Alzheimer disease and decline in cognitive function. *Archives of Neurology*, 61(5), 661-666. doi:10.1001/archneur.61.5.661
- Barnard, N. D., Bush, A. I., Ceccarelli, A., Cooper, J., de Jager, C. A., Erickson, K. I., Fraser, G., Kesler, S., Levin, S., Lucey, B., Morris, M., Squittik, I. R., & Morris, M. C. (2014). Dietary and lifestyle guidelines for the prevention of Alzheimer's disease. *Neurobiology of Aging*, 35, S74-S78. <http://dx.doi.org/10.1016/j.neurobiolaging.2014.03.033>
- Bell, S. J., & Sears, B. (2003). Low-glycemic-load diets: Impact on obesity and chronic diseases. *Critical Reviews in Food Science and Nutrition*, 43 (4) 357-377. <http://dx.doi.org/10.1080/10408690390826554>
- Bente, L., & Gerrior, S. A. (2002). Selected food and nutrient highlights of the 20th century: US food supply series. *Family Economics and Nutrition Review*, 14(1), 43. Retrieved from <http://proxy.lib.sfu.ca/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=a9h&AN=7164479&site=ehost-live>
- Biessels, G. J., Bravenboer, B., & Gispen, W. H. (2004). Glucose, insulin and the brain: Modulation of cognition and synaptic plasticity in health and disease: a preface. *European Journal of Pharmacology*, 490(1), 1-4. <http://dx.doi.org/10.1016/j.ejphar.2004.02.057>
- Bird, D. (2014). Alzheimer Disease Overview. *GeneReviews*. Retrieved from: http://www.ncbi.nlm.nih.gov/books/NBK1161/?report=reader#_NBK1161_pubdet_
- Burkitt, D. P. (1973). Some diseases characteristic of modern western civilization: A possible common causative factor. *Clinical Radiology*, 24(3), 271-280. doi:10.1016/S0009-9260(73)80037-6
- Clark, A. (1982). Foraging behavior of a vertebrate omnivore (*Rattus rattus*): Meal structure, sampling, and diet breadth. *Ecology*, 63(3), 763-772. doi: 10.2307/1936797
- Cleave, T. (1974). *The saccharine disease: Conditions caused by the taking of refined carbohydrates, such as sugar and white flour*. Bristol: John Wright & Sons, Ltd.
- Cordain, L., Eades, M. R., & Eades, M. D. (2003). Hyperinsulinemic diseases of civilization: More than just Syndrome X. *Comparative Biochemistry and Physiology Part A: Molecular & Integrative Physiology*, 136(1), 95-112. [http://dx.doi.org/10.1016/S1095-6433\(03\)00011-4](http://dx.doi.org/10.1016/S1095-6433(03)00011-4)
- Cordain, L., Eaton, B., Brand Miller, J., Mann, N., & Hill, K. (2002). Original Communications-The paradoxical nature of hunter-gatherer diets: Meat-based, yet

- non-atherogenic. *European Journal of clinical nutrition*, 56(1), 42. doi: 10.1038/sj=ejcn=1601353
- Cordain, L., Eaton, S. B., Sebastian, A., Mann, N., Lindeberg, S., Watkins, B. A. & Brand-Miller, J. (2005). Origins and evolution of the Western diet: health implications for the 21st century. *The American Journal of Clinical Nutrition*, 81(2), 341-354. Retrieved from <http://ajcn.nutrition.org/content/81/2/341.short>
- Craft, S., & Watson, G. S. (2004). Insulin and neurodegenerative disease: shared and specific mechanisms. *The Lancet Neurology*, 3(3), 169-178. [http://dx.doi.org/10.1016/S1474-4422\(04\)00681-7](http://dx.doi.org/10.1016/S1474-4422(04)00681-7)
- Crawford, M. A. (1992). The role of dietary fatty acids in biology: their place in the evolution of the human brain. *Nutritional Review*, 50(4), 3-11. doi: 10.1111/j.1753-4887.1992.tb 01283.x
- Danial, N., Hartman, L., Stafstrom, E., & Thio, L. L. (2013). How does the ketogenic diet work? Four potential mechanisms. *Journal of child neurology*, 28(8),1027-1033. doi: 10.1177/0883073813487598
- Dennett, D. C. (1995). Darwin's dangerous idea. New York, NY: Simon and Schuster.
- Despres, J. P. (1992). Abdominal obesity as important component of insulin-resistance syndrome. *Nutrition*, 9(5), 452-459. Retrieved from <https://www.ncbi.nlm.nih.gov/pubmed/8286886>
- Després, J. P., & Lemieux, I. (2006). Abdominal obesity and metabolic syndrome. *Nature*, 444(7121), 881-887. doi:10.1038/nature05488
- Dhamija, R., Eckert, S., & Wirrell, E. (2013). Ketogenic diet. *The Canadian Journal of Neurological Sciences*, 40(02), 158-167. <https://doi.org/10.1017/S0317167100013676>
- Dobzhansky, T. (2013). Nothing in biology makes sense except in the light of evolution. *The American Biology Teacher*, 75(2), 87-91. <http://dx.doi.org/10.2307/4444260>
- Ferri, C. P., Prince, M., Brayne, C., Brodaty, H., Fratiglioni, L., Ganguli, M., Hall, K., Hasegawa, K., Hendrite, H., Huang, Y., Jorm, A., Mathers, C., Menzies, P., Rimmer, E., Sczafuoca, M., & Jorm, A. (2006). Global prevalence of dementia: A Delphi consensus study. *The Lancet*, 366(9503), 2112-2117. [http://dx.doi.org/10.1016/S0140-6736\(05\)67889-0](http://dx.doi.org/10.1016/S0140-6736(05)67889-0)
- Fouché, F. P. (1923). Freedom of Negro Races from Cancer. *British Medical Journal*. 1(3261), 1116. Retrieved from <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2316860/>
- Frisardi, V., Solfrizzi, V., Seripa, D., Capurso, C., Santamato, A., Sancarlo, D., Vendemialec G., Pilotto A., & Panza, F. (2010). Metabolic-cognitive syndrome: A cross-talk between metabolic syndrome and Alzheimer's disease. *Ageing Research Reviews*, 9(4), 399-417. <http://dx.doi.org/10.1016/j.arr.2010.04.007>
- Frölich, L., Blum-Degen, D., Bernstein, H. G., Engelsberger, S., Humrich, J., Laufer, S., Muschner, A., Thalheimer A., Türk S., Hoyer R., Zöchling, W., Boissl K., Jellinger P & Riederer. (1998). Brain insulin and insulin receptors in aging and sporadic Alzheimer's disease. *Journal of Neural Transmission*, 105(4-5), 423-438. doi: 10.1007/s007020050068
- Grey, N., & Kipnis, D. M. (1971). Effect of diet composition on the hyperinsulinemia of obesity. *New England Journal of Medicine*, 285(15), 827-831. doi: 10.1056/NEJM197110072851504
- Havrankova, J., Brownstein, M., & Roth, J. (1981). Insulin and insulin receptors in rodent brain. *Diabetologia*, 20(3), 268-273. doi:10.1007/BF00254492
- Jellinger, K. A. (2006). Alzheimer 100

- Highlights in the history of Alzheimer research. *Journal of Neural Transmission*, 113(11), 1603-1623. doi: 10.1007/s00702-006-0578-3
- Kahn, B. B., & Flier, J. S. (2000). Obesity and insulin resistance. *The Journal of Clinical Investigation*, 106(4), 473-481. doi:10.1172/JCI10842
- Kratz, M., Baars, T., & Guyenet, S. (2013). The relationship between high-fat dairy consumption and obesity, cardiovascular, and metabolic disease. *European Journal of Nutrition*, 52(1), 1-24. doi: 10.1007/s00394-012-0418-1
- Krishnan, S., Rosenberg, L., Singer, M., Hu, F. B., Djoussé, L., Cupples, L. A., & Palmer, J. R. (2007). Glycemic index, glycemic load, and cereal fiber intake and risk of type 2 diabetes in US black women. *Archives of Internal Medicine*, 167(21), 2304-2309. doi:10.1001/archinte.167.21.2304
- Langan, S. J., Deary, I. J., Hepburn, D. A., & Frier, B. M. (1991). Cumulative cognitive impairment following recurrent severe hypoglycaemia in adult patients with insulin-treated diabetes mellitus. *Diabetologia*, 34(5), 337-344. doi:10.1007/BF00405006
- Leonard, W. R., Robertson, M. L., Snodgrass, J. J., & Kuzawa, C. W. (2003). Metabolic correlates of hominid brain evolution. *Comparative Biochemistry and Physiology Part A: Molecular & Integrative Physiology*, 136(1), 5-15. [http://dx.doi.org/10.1016/S1095-6433\(03\)00132-6](http://dx.doi.org/10.1016/S1095-6433(03)00132-6)
- Liu, S., Willett, W. C., Stampfer, M. J., Hu, F. B., Franz, M., Sampson, L., Hennekens, C. & Manson, J. E. (2000). A prospective study of dietary glycemic load, carbohydrate intake, and risk of coronary heart disease in US women. *The American Journal of Clinical Nutrition*, 71(6), 1455-1461. Retrieved from <http://ajcn.nutrition.org/content/71/6/1455.short>
- Luchsinger, J. A., Tang, M. X., Shea, S., & Mayeux, R. (2004). Hyperinsulinemia and risk of Alzheimer disease. *Neurology*, 63(7), 1187-1192. <http://dx.doi.org/10.1212/01.WNL.0000140292.04932.87>
- Ludwig, D. S. (2002). The glycemic index: physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. *Jama*, 287(18), 2414-2423. doi:10.1001/jama.287.18.2414
- Ludwig, D. S., Majzoub, J. A., Al-Zahrani, A., Dallal, G. E., Blanco, I., & Roberts, S. B. (1999). High glycemic index foods, overeating, and obesity. *Pediatrics*. 103(3), 26. doi: 10.1542/peds.103.3.e26
- Ma, W., Berg, J., & Yellen, G. (2007). Ketogenic diet metabolites reduce firing in central neurons by opening KATP channels. *The Journal of Neuroscience*, 27(14), 3618-3625. doi:10.1523/JNEUROSCI.0132-07.2007
- Miller, J. C. (1994). Importance of glycemic index in diabetes. *The American Journal of Clinical Nutrition*, 59(3), 747-752. Retrieved from <http://ajcn.nutrition.org/content/59/3/747S.short>
- Morrison, C. D., Pistell, P. J., Ingram, D. K., Johnson, W. D., Liu, Y., Fernandez-Kim, S. O., White, C., Purpera, M., Urang, R., Bruce-Kellerand, A., & Keller, J. N. (2010). High fat diet increases hippocampal oxidative stress and cognitive impairment in aged mice: Implications for decreased Nrf2 signaling. *Journal of Neurochemistry*, 114(6), 1581-1589. doi: 10.1111/j.1471-4159.2010.06865.x
- Nam, Y., Lee, J., Kim, R., Cha, S., Song, D., Lim, K., Lee, C. & Huh, K. B. (1997). Effect of obesity on total and free insulin-like growth factor (IGF)-1, and their relationship to IGF-binding protein (BP)-1, IGFBP-2, IGFBP-3,

- insulin, and growth hormone. *International Journal of Obesity & Related Metabolic Disorders*, 21(5). Retrieved from <http://www.nature.com.proxy.lib.sfu.ca/ijo/journal/v21/n5/abs/0800412a.html>
- Okamura, F., Tashiro, A., Utumi, A., Imai, T., Suchi, T., Tamura, D. & Hongo, M. (2000). Insulin resistance in patients with depression and its changes during the clinical course of depression: minimal model analysis. *Metabolism*, 49(10), 1255-1260. doi:10.1053/meta.2000.9515
- Orr, J. B., & Gilks, J. L. (1931). Studies of nutrition. The physique and health of two African tribes. Special Report Series. Medical Research Council, (155). doi:10.1001/jama.1931.02730200061030
- Pathan, A. R., Gaikwad, A. B., Viswanad, B., & Ramarao, P. (2008). Rosiglitazone attenuates the cognitive deficits induced by high fat diet feeding in rats. *European Journal of Pharmacology*, 589(1), 176-179. <http://dx.doi.org/10.1016/j.ejphar.2008.06.016>
- Pimenta, W., Mitrakou, A., Jensen, T., Yki-Järvinen, H., Daily, G., & Gerich, J. (1996). Insulin secretion and insulin sensitivity in people with impaired glucose tolerance. *Diabetic Medicine: A journal of the British Diabetic Association*, 13(9), 33-36. Retrieved from <http://europepmc.org/abstract/med/8894478>
- Pistell, P. J., Morrison, C. D., Gupta, S., Knight, A. G., Keller, J. N., Ingram, D. K., & Bruce-Keller, A. J. (2010). Cognitive impairment following high fat diet consumption is associated with brain inflammation. *Journal of Neuroimmunology*, 219(1), 25-32. <http://dx.doi.org/10.1016/j.jneuroim.2009.11.010>
- Profenno, L. A., Porsteinsson, A. P., & Faraone, S. V. (2010). Meta-analysis of Alzheimer's disease risk with obesity, diabetes, and related disorders. *Biological psychiatry*, 67(6), 505-512. <http://dx.doi.org/10.1016/j.biopsych.2009.02.013>
- Qiu, W. Q., & Folstein, M. F. (2006). Insulin, insulin-degrading enzyme and amyloid- β peptide in Alzheimer's disease: Review and hypothesis. *Neurobiology of Aging*, 27(2), 190-198. <http://dx.doi.org/10.1016/j.neurobiolaging.2005.01.004>
- Reaven, G. M., & Laws, A. (Eds.). (1999). *Insulin resistance: the metabolic syndrome X* (Vol. 12). Springer Science & Business Media.
- Reger, M. A., Henderson, S. T., Hale, C., Cholerton, B., Baker, L. D., Watson, G. S., & Craft, S. (2004). Effects of β -hydroxybutyrate on cognition in memory-impaired adults. *Neurobiology of Aging*, 25(3), 311-314. [http://dx.doi.org/10.1016/S0197-4580\(03\)00087-3](http://dx.doi.org/10.1016/S0197-4580(03)00087-3)
- Reinhardt, R., & Bondy, C. A. (1994). Insulin-like growth factors cross the blood-brain barrier. *Endocrinology*, 135(5), 1753-1761. <http://dx.doi.org/10.1210/endo.135.5.7525251>
- Rho, J. M., & Stafstrom, C. E. (2012). The ketogenic diet as a treatment paradigm for diverse neurological disorders. *Frontiers in pharmacology*, 3, 59. <http://dx.doi.org/10.3389/fphar.2012.00059>
- Saher, G., Brügger, B., Lappe-Siefke, C., Möbius, W., Tozawa, R. I., Wehr, M. C., Wieland, F., Ishibashi, S., & Nave, K. A. (2005). High cholesterol level is essential for myelin membrane growth. *Nature Neuroscience*, 8(4), 468-475. doi:10.1038/nn1426
- Samaha, F. F., Iqbal, N., Seshadri, P., Chicanos, K. L., Daily, D. A., McGrory, J. Williams T., Williams, M., Gracely, D & Stern, L. (2003). A low-carbohydrate as compared with a low-fat diet in severe obesity. *New England Journal of Medicine*, 348(21), 2074-2081. doi: 0.1056/NEJMoa022637

- Schaefer, O. (1971). When the Eskimo comes to town. *Nutrition Today*, 6(6), 8-16. Retrieved from http://journals.lww.com/nutritiontodayonline/Citation/1971/11000/When_The_Eskimo_Comes_To_Town.3.aspx
- Sommerfield, A. J., Deary, I. J., & Frier, B. M. (2004). Acute hyperglycemia alters mood state and impairs cognitive performance in people with type 2 diabetes. *Diabetes Care*, 27(10), 2335-2340. <http://dx.doi.org/10.2337/diacare.27.10.2335>
- Spector, A., Orrell, M., Lattimer, M., Hoe, J., King, M., Harwood, K., Qazi A. & Charlesworth, G. (2012). Cognitive behavioural therapy (CBT) for anxiety in people with dementia: Study protocol for a randomised controlled trial. *Trials*, 13(1), 197. doi: 10.1186/1745-6215-13-197
- Statistics Canada. (2016). Body mass index, overweight or obese, self-reported, adult, by sex, provinces and territories (Number of persons). Retrieved from <http://www.statcan.gc.ca/tables-tableaux/sum-som/l01/cst01/health82a-eng.htm>
- Statistics Canada. (2016). Diabetes, by age group and sex (Number of persons). (2016). Retrieved from <http://www.statcan.gc.ca/tables-tableaux/sum-som/l01/cst01/health53a-eng.htm>
- Ströhle, A., & Hahn, A. (2011). Diets of modern hunter-gatherers vary substantially in their carbohydrate content depending on environments: Results from an ethnographic analysis. *Nutrition Research*, 31(6), 429-435. <http://dx.doi.org/10.1016/j.nutres.2011.05.003>
- Trowell, H. C. & Burkitt, D.P. (1981). (Eds.). *Western diseases, their emergence and prevention*. Massachusetts, MA: Harvard University Press.
- USDA Food Composition Database. (2016). Foods List. Retrieved 16 October 2016, from <https://ndb.nal.usda.gov/ndb/search/list>.
- Volek, J. S., & Feinman, R. D. (2005). Carbohydrate restriction improves the features of Metabolic Syndrome. Metabolic Syndrome may be defined by the response to carbohydrate restriction. *Nutrition & Metabolism*, 2(1), 1. doi: 10.1186/1743-7075-2-31
- Walker, D. A. (2004). Cognitive behavioural therapy for depression in a person with Alzheimer's dementia. *Behavioural and Cognitive Psychotherapy*, 32(04), 495-500. <https://doi.org/10.1017/S1352465804001663>
- Wallwork, J. C. (1986). Zinc and the central nervous system. *Progress in Food & Nutrition Science*, 11(2), 203-247. doi: 10.1016/j.stem.2012.01.017
- Weiler, P. G. (1987). The public health impact of Alzheimer's disease. *American Journal of Public Health*, 77(9), 1157-1158. Retrieved from <http://ajph.aphapublications.org/doi/pdf/10.2105/AJPH.77.9.1157>
- Weyer, C., Bogardus, C., Mott, D. M., & Pratley, R. E. (1999). The natural history of insulin secretory dysfunction and insulin resistance in the pathogenesis of type 2 diabetes mellitus. *The Journal of Clinical Investigation*, 104(6), 787-794. doi:10.1172/JCI7231.
- Whitmer, R. A., Gustafson, D. R., Barrett-Connor, E., Haan, M. N., Gunderson, E. P., & Yaffe, K. (2008). Central obesity and increased risk of dementia more than three decades later. *Neurology*, 71(14), 1057-1064. <http://dx.doi.org/10.1212/01.wnl.0000306313.89165.ef>
- Whitmer, R. A., Karter, A. J., Yaffe, K., Quesenberry, C. P., & Selby, J. V. (2009). Hypoglycemic episodes and risk of dementia in older patients with type 2 diabetes mel-

litus. *Jama*, 301(15), 1565-1572.
<http://dx.doi.org/10.1212/01.wnl.0000306313.89165.ef>

Wills, C. (2008). Evolution theory and the future of humanity. In Bostrom & Cirkovic (Eds.), *Global Castatrophic Risk* (48-60). New York, NY: Oxford University Press.

Wredling A., Theorell T., Roll M., Lins S & Adamson K. (1992). Psychosocial state of patients with IDDM prone to recurrent episodes of severe hypoglycemia. *Diabetes Care*, 15(4), 518-521. <http://dx.doi.org/10.2337/diacare.15.4.518>

Yancy, W. S., Olsen, M. K., Guyton, J. R., Bakst, R. P., & Westman, E. C. (2004). A low-carbohydrate, ketogenic diet versus a low-fat diet to treat obesity and hyperlipidemia: a randomized, controlled trial. *Annals of Internal Medicine*, 140(10), 769-777. doi: 10.7326/0003-4819-140-10-200405180-00006

Yudkin, J. (1972). *Pure, white and deadly*. New York, NY: Penguin Books.

Yudkoff, M., Daikhin, Y., Nissim, I., Lazarow, A., & Nissim, I. (2004). Ketogenic diet, brain glutamate metabolism and seizure control. *Prostaglandins, Leukotrienes and Essential Fatty Acids*, 70(3), 277-285. <http://dx.doi.org/10.1016/j.plefa.2003.07.005>

THIS PUBLICATION IS PROUDLY SPONSORED BY:



psychology student union
SIMON FRASER UNIVERSITY

SFU PSYCHOLOGY

