University of Alberta

Clinical Implications of Historical Development of the DSM through Examining Two Main Disorders

by

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A thesis submitted to the Faculty of Graduate Studies and Research
in partial fulfillment of the requirements for the degree of

Doctor of Philosophy

in

Psychological Studies in Education

Educational Psychology

Fall 2013
Edmonton, Alberta

I dedicate this dissertation to my wife, Julianne, who has travelled with me on this journey every step of the way.

Abstract

As a gateway to support for those who experience significant distress or impairment as a result of cognitive, affective, developmental, social, environmental, or other personal/interpersonal challenges, mental disorder diagnosis is a common practice in mental health professions. In order to practice effectively and ethically, diagnosticians should develop a keen understanding of the diagnostic guidelines they follow in research and practice. The Diagnostic and Statistical Manual for Mental Disorders (DSM) is a dominant diagnostic classification used throughout the world. Because it impacts innumerable lives in significant ways, the DSM should be used judiciously by diagnosticians. By understanding the history and development of the DSM within a clinical backdrop, diagnosticians can better understand the fundamental strengths and limitations of this hegemonic manual. In addition, understanding the history and development of specific mental disorders can help diagnosticians better understand the conditions they diagnose and facilitate a more rigorous and careful diagnostic process. This thesis uses paper format, where each chapter stands as its own study. Using a paper thesis format, dissemination via peer-reviewed journals is more easily facilitated so that the papers may be available to a wide body of readership in order to promote more judicious use of the DSM and mental disorder diagnosis. Variations of four chapters in this paper-format dissertation have been published in peer-reviewed journals, and a fifth has been accepted and is in press. In addition to a review of the historical developments of the DSM, I review the historical developments of autism and Asperger's Disorder,

developmental disorders within the current edition of the DSM, and Fetal Alcohol Spectrum Disorder (FASD), a developmental disorder that is not in the current edition of the DSM but has been proposed for a future edition. Finally, recommendations for ethical and effective diagnostic practice using the DSM or other diagnostic guidelines are provided.

I acknowledge with gratitude my doctoral supervisor, Dr. George Buck, for his unbounded support over the past several years. I also thank the faculty members in the Psychological Studies in Education program in the Department of Educational Psychology, University of Alberta, particularly Drs. Martin Mrazik and Henry Janzen, for their academic insights and professional examples. They have inspired me to practice with integrity and passion.

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List of Acronyms

AD – Asperger's Disorder

APA – American Psychiatric Association

ARND – Alcohol-Related Neurodevelopmental Disorder

ASD – Autism Spectrum Disorder

DSM – Diagnostic and Statistical Manual of Mental Disorders

FAS – Fetal Alcohol Syndrome

FASD – Fetal Alcohol Spectrum Disorder

HFA – High Functioning Autism

ICD – International Classification of Diseases

ND-PAE – Neurobehavioural Disorder associated with Prenatal Alcohol Exposure

NOS – Not Otherwise Specified

PAE – Prenatal Alcohol Exposure

PDD – Pervasive Developmental Disorder

pFAS – Partial Fetal Alcohol Syndrome

The Historical Development of the Diagnostic and Statistical Manual of Mental Disorders (DSM)

Introduction

The importance of diagnostic classification in addressing concerns and formulating effective treatments for individuals with mental disorders cannot be overstated (Grob, 1991). Diagnoses are important in determining appropriate intervention strategies and accessing services. A host of organizations, including government-funded programs, insurance companies, managed health care programs, and employee assistance programs often require a diagnosis of a mental disorder before supports or funding are provided for clients (Kirk & Kutchins, 1988; Mead, Hohenshil, & Singh, 1997). For better or for worse, a diagnosis of a mental disorder can therefore be described as the gateway to recovery and support for many patients (House, 2001). Diagnoses are also used in experimental and quasi-experimental research as treatment groups, and often matched control groups, are stratified by diagnosis.

The Diagnostic and Statistical Manual of Mental Disorders (DSM) is an ever-growing and evolving compendium of mental disorders, their classifications, and their diagnostic criteria. Currently in its fourth edition (DSM-IV-TR), the manual describes a mental disorder as aberrant thinking, feeling, or behaving that involves distress, impaired functioning, or significantly increased risk of negative outcome (APA, 2000). There are currently over three-hundred diagnoses and their criteria described in the text.

Classifications of mental disorders have emerged and developed across continents and throughout history (King, 1999). Since the turn of the twentieth century, interest in psychiatric nosologies has grown immensely. Numerous nosologies in North America, some standardized and some home-grown, preceded the development of the first edition of the DSM in 1952 (Grob, 1991). Some of these were directly involved in the development of the DSM (i.e. The Statistical Manual for the Use of Institutions for the Insane in 1918). It was partly in response to different diagnostic "languages" spoken in different nosologies in North America that that spurred the creation of the DSM (APA, 1952). Without a standard classification system researchers and clinicians were confounded by different diagnostic languages. The DSM has met an important need in defining a common diagnostic language for both clinicians and researchers (Jampala, Zimmerman, Sierles, & Taylor, 1992).

There are few texts as influential and widely used as the DSM. The DSM is a prevailing diagnostic classification that is used worldwide in both research and clinical practice (Maser, Kaelber, & Weise, 1991; Mezzich, 2002). Because of its widespread use by clinicians in the diagnosis of mental disorders, "very few professional documents compare to the DSM in affecting the welfare of countless persons" (Rogler, 1997, p. 9). Yet, as described by Follette and Houts (1996), "the degree of influence of the DSM is way out of proportion with the science supporting it" (p. 1129).

Despite, and perhaps because of, its widespread use, the DSM has come under heavy criticism. The validity of the concept of mental illness itself has been

questioned for some time, and diagnoses of mental disorders have been viewed as marginalizing and oppressive (Szasz, 1960). The claim of an atheoretical orientation to diagnosis by its authors has been questioned given its heavy focus on medical and biological research using positivistic and empirical approaches as means of validating the disorders therein (Cooper, 2004; Follette & Houts, 1996). However, positivistic and empirical approaches have done little to confirm the validity or even the reliability of the majority of disorders in the DSM (Hyman, 2010).

Ideals aside, use of the DSM has become increasingly important amongst medical and non-medical helping professionals. If mental disorder diagnosis is the gateway to support, the DSM could be described as the key to the gate. In fact, many programs and third-party payers require not just a mental disorder diagnosis to begin services, but an Axis I DSM-IV-TR mental disorder diagnosis, including a DSM code (Goldman & Grob, 2006; Rappo, 2002; Rushton, Felt, & Roberts, 2002). For this reason, use of the DSM has become increasingly common amongst non-medical mental health professionals, including social workers (Kirk & Kutchins, 1988; Newman, Dannenfelser, & Clemmons, 2007), mental health counsellors (Cosgrove, 2005; Mead et al., 1997), clinical psychologists (Smith & Kraft, 1983; Zammit, 1995), and school psychologists (McBurnett, 1996).

Despite its hegemonic status (Anand & Malhi, 2011), the use of the DSM has arguably fallen prey to the whims of institutional efficiency. Many third-party payers and government funded programs (e.g. Alberta Education) require the use of diagnostic codes before supports and funding are dispersed. Alberta Education,

for example, currently utilises its own coding system to identify students in need of specialized education (Alberta Education, 2011). In my professional experience conducting psychoeducational assessments in schools, I have found that many teachers and even parents communicate via this coding system. For example, teachers will often inquire whether a student experiences "code 54", which represents a learning disability. The DSM-IV-TR uses a coding system to identify and organize the disorders therein. Amongst Learning Disorders, for example, a Reading Disorder is given code 315.00, Mathematics Disorder code 315.1, Disorder of Written Expression code 315.2, and the ever-flexible Learning Disorder Not Otherwise Specified code 315.9 (APA, 2000). Although mental disorder diagnoses and codes are more efficient from an institutional standpoint, they do little to describe the client's actual impairment and remedial needs (Goldman & Grob, 2006). Yet it is the code and often the type of code, rather than the client's need for support, that opens the door to support and, more specifically, funding (Rushton et al., 2002). Rather than structuring support based on need, the client in essence is at-risk of being re-structured to fit a diagnostic code in order to meet eligibility requirements. In practice, this can result in a hunt for a diagnosis (or misdiagnosis) in an attempt to meet the needs of the client (Rappo, 2002), which certainly has ramifications for the ethical and professional conduct of users (Braun & Cox, 2005; Rappo, 2002). For these reasons, these coding requirements have serious implications for how the DSM is used in practice.

Because the DSM impacts innumerable lives on individual and collective levels, it must be used judiciously. Yet, apart from peer evaluation, there is little

monitoring of its everyday use amongst professionals. The user qualifications described in DSM-IV-TR are unspecific and scant, warning that, "use of these criteria requires specialized clinical training that provides both a body of knowledge and clinical skills" (APA, 2000, p. XXXVii). Decisions on what constitute adequate clinical training, knowledge, and clinical skills are presumably left to the user. There are no formal practices in place to monitor its use and purchase and use of the manual is not limited to credentialed professionals. Presumably a diagnosis could be made by anyone at any time so long as s/he claim (or are perceived to have) authority by his or her audiences to do so.

Because judicious use of the DSM falls predominantly on the user, it is critical that users acquire adequate and appropriate training for this important manual. Cosgrove (2005) provided a number of helpful recommendations for teaching students in mental health counselling programs about using the manual through a critical lens. Cosgrove cited the importance of challenging the epistemology that mental disorders pre-exist and that they are awaiting discovery and clarification upon empirical validation. Rather, Cosgrove added, mental disorders are constructions of reality that are subject to biases. The DSM, as an authoritative manual on mental disorders, is intricately tied to how mental disorders are constructed and understood.

There are few better ways of relating how the DSM is a constitutive manual than by exploring its history. The DSM has undergone many significant changes in content, orientation, organization, and volume since its first edition in 1952. These changes have arguably been brought about not only by evolving

research but by socio-political influences (Schacht, 1985). Understanding the history and development of the DSM can help clinicians and researchers better understand the diagnostic language they are speaking, as well as its strengths and limitations. If nothing else, knowing the history of the DSM can help professionals think critically about the constructionist nature of the manual and remind them to use it carefully.

In this paper, I will critically evaluate the history and development of the DSM with the aim to increase knowledge and understanding of the underlying strengths and weaknesses of the manual amongst its users. As stated above, the judicious use of the DSM by diagnosticians is of utmost importance. This begins by recognizing not only the DSM's fundamental strengths but its fundamental limitations as well, and these will be explored in my study. Although its history has been reviewed elsewhere (Collier, 2008; Decker, 2007; Grob, 1991; Grob, 1994; Jablensky, 2007; Kendler, 2009; Mayes & Horwitz, 2005; Rogler, 1997), it does not appear that the evolution of the DSM has been explored with a clinical backdrop, which is the intent of this paper.

The DSM has undergone enormous scrutiny, opposition, and condemnation by some (Cooper, 2004), and rightfully so; a manual that is so influential in countless lives should be subject to intense review and criticism. Arguably, the DSM has not passed, but has thus far survived the test of scrutiny, buoyed in part by societal institutions that mandate its use. Yet, it continues reign as a dominant classification system. Regardless of the ideals of the evolving mental disorder epistemology, the practical truth is that the DSM is not going

anywhere anytime soon. Psychodiagnosticians need a keen understanding of the developments of what is perhaps the most important manual in their offices, beyond a familiarity of what is printed in its text. It is my hope that the content of my dissertation may signal clinicians to evaluate and re-evaluate the DSM, its underlying concepts, and their use of it.

As an undergraduate student, I was enthused when I perused for the first time a copy of the DSM-IV-TR (APA, 2000). I was impressed by the volume of the text and felt that it must have been an exhaustive guide to all the mental disorders that ever had been "discovered". I am still impressed with the DSM-IV-TR but over time have come to appreciate its limitations. I first flipped to the diagnostic criteria for autism, a Pervasive Developmental Disorder (PDD) of which I had much interest. When scanning the adjacent pages, I came across Asperger's Disorder, another PDD similar to, but qualitatively and categorically distinct from autism. Little did I know at the time that both diagnoses would likely be subsumed under one condition, Autism Spectrum Disorder (ASD), as suggested in the changes proposed by the American Psychiatric Association (APA) as well as by many leading researchers (Volkmar, State, & Klin, 2009). If I had been presented with this idea at the time I believe I would have questioned, "How could there be Asperger's Disorder one year and not the next? How could a mental disorder cease to exist?" Understanding the history of mental disorders can provide insight into their developments, their inclusion or exclusion from the DSM, and resulting controversies.

Understanding the history and development of specific mental disorders in the DSM can also help diagnosticians better understand the individual conditions they diagnose. Conceptualizations of mental disorders and consequently diagnostic criteria change over time, whether they are the result of increased knowledge and empirical evidence, changing epistemologies, or socio-political pressures (Cooper, 2004). Understanding and being critical of the continual development of such conceptualizations should help facilitate a more rigorous and careful diagnostic process amongst individual clinicians.

In addition to a critical review of the DSM, I will evaluate the evolution of autism and Asperger's Disorder, developmental disorders within the current edition of the DSM. Specifically, I will evaluate how the history of these disorders may have contributed to their inclusion as distinct disorders in DSM-IV and DSM-IV-TR, and how that might change with the advent of DSM-V. With the relatively recent inclusion and (probable) exclusion of a major diagnosis with a great deal of clinical relevance such as Asperger's Disorder (Ghaziuddin, 2011), the historical study of this disorder provides a rich opportunity to demonstrate how the history of a disorder, rather than empirical research supporting its validity, may have had a greater influence on whether it is included in the DSM.

Recognizing that future editions of the DSM including the upcoming fifth edition are working documents (Kupfer, Regier, & Kuhl, 2008), it is quite probable that beyond DSM-V many disorders will probably be modified, new disorders and diagnostic criteria are likely to be defined, and some disorders will probably be subsumed under other categories or removed entirely. Fetal Alcohol

Syndrome (FAS), a diagnostic category under the umbrella of Fetal Alcohol Spectrum Disorders (FASD) (Chudley et al., 2005), is noted as a diagnosis proposed for DSM-V by sources outside APA (APA, n.d.), and may be given consideration for future editions. Although research on prenatal alcohol exposure has only gained momentum since the mid-1970s, there is much evidence of a neurocognitive profile for FASD that causes significant delays in many functional areas (Kodituwakku, 2009), and there is merit in including this diagnosis in the DSM based on the manual's emphasis on empirically validated disorders (APA, 2000).

A primary purpose for my evaluation of ASD and FASD in this paper dissertation is that of clinical interest. As a Registered Psychologist in the Province of Alberta I have maintained an interest in the diagnosis (and misdiagnosis) of these disorders. In addition, as a member of the FASD diagnostic clinic team in my city of residence, the study of FASD specifically provides an additional advantage of helping me think critically about the professional guidelines I follow in my own diagnostic practices. Broadly speaking, a critical analysis of the DSM as a whole is also directly beneficial as differential and co-morbid diagnoses are constant considerations in psychodiagnostic practice.

Additionally, it was necessary to pare down to evaluations of a couple disorders to ensure thorough evaluations, given that the DSM-IV-TR classifies over 300 mental disorders and that there are other disorders with empirical support outside the DSM. In addition to sharing my clinical interest, ASD and FASD are both significant lifelong developmental disabilities predominantly

diagnosed in childhood. ASD may involve a teratogenic effect (Newschaffer et al., 2007), which is definite in FASD. Both tend to involve cognitive, adaptive, and executive dysfunction, as well as difficulty with social skills (Bishop, Gahagan, & Lord, 2007) and safety (Strickland, McAllister, Coles, & Osborne, 2007).

Structure of Dissertation

The histories of the DSM, ASD, and FASD will be evaluated and synthesized in a paper thesis format rather than a traditional format. This thesis using a paper format includes an introductory section describing an outline and rationale for the project. Following this introductory section I will include a section outlining the methods of research, describing historical research as a type of qualitative research with important operational distinctions from other forms of qualitative inquiry. Each subsequent section, exploring the histories of DSM, ASD, and FASD, will be assigned its own bibliography, and each can stand alone as its own study. Synthesis of results with existing literature will be completed after historical analyses to allow the historical evidence to guide the search for relevant literature rather than my influenced notions of what is relevant, which is a common strategy in qualitative inquiry (Johnson & Christensen, 2007). A final chapter will be presented, including a general discussion, a synthesis of chapters, and conclusions. In addition, the final chapter will include recommendations for clinicians and researchers for more judicious use of the DSM. Using a paper thesis format, dissemination of the research via peer-reviewed journals is more easily facilitated. This is appropriate given my goal to publish these chapters so

that they might be available to a wide body of readership, including psychodiagnosticians, so that this dissertation may promote more judicious use of the DSM and mental disorder diagnosis.

Bibliography

Alberta Education. (2011). *Special education coding criteria 2011/2012*.http://education.alberta.ca/media/825847/spedcodingcriteria.pdf

Anand, S., & Malhi, G. S. (2011). From manual to bible: The questionable hegemony of DSM IV *The Australian and New Zealand Journal of Psychiatry, 45*(5), 348-350. doi:10.3109/00048674.2011.576814

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders*. Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders - text revision* (4th ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (n.d.). *Proposed draft revisions to DSM disorders and criteria.* Retrieved June 5, 2010, from www.dsm5.org

Bishop, S., Gahagan, S., & Lord, C. (2007). Re-examining the core features of autism: A comparison of autism spectrum disorder and fetal alcohol spectrum disorder. *Journal of Child Psychology & Psychiatry & Allied Disciplines, 48*(11), 1111-1121.

Braun, S. A., & Cox, J. A. (2005). Managed mental health care: Intentional misdiagnosis of mental disorders. *Journal of Counseling and Development : JCD, 83*(4), 425.

Chudley, A. E., Conry, J., Cook, J. L., Loock, C., Rosales, T., & LeBlanc, N. (2005). Fetal alcohol spectrum disorder: Canadian guidelines for diagnosis. *Canadian Medical Association Journal, 172*(Suppl), S1-S21.

Collier, E. (2008). Historical development of psychiatric classification and mental illness. *British Journal of Nursing, 17*(14), 890-894.

Cooper, R. (2004). What is wrong with the DSM? *History of Psychiatry, 15*(57), 005-025. doi:http://dx.doi.org/10.1177/0957154X04039343

Cosgrove, L. (2005). When labels mask oppression: Implications for teaching psychiatric taxonomy to mental health counselors. *Journal of Mental Health Counseling, 27*(4), 283-296.

Decker, H. S. (2007). How Kraepelinian was Kraepelin? how Kraepelinian are the neo-Kraepelinians?--from Emil Kraepelin to DSM-III. *History of Psychiatry, 18*(71), 337-360.

Follette, W. C., & Houts, A. C. (1996). Models of scientific progress and the role of theory in taxonomy development: A case study of the DSM. *Journal of Consulting & Clinical Psychology, 64*(6), 1120-1132.

Ghaziuddin, M. (2011). Asperger disorder in the DSM-V: Sacrificing utility for validity. *Journal of the American Academy of Child & Adolescent Psychiatry, 50*(2), 192-193. doi:http://dx.doi.org/10.1016/j.jaac.2010.11.013

Goldman, H. H., & Grob, G. N. (2006). Defining 'mental illness' in mental health policy. *Health Affairs, 25*(3), 737.

Grob, G. N. (1991). Origins of DSM-I: A study in appearance and reality. *American Journal of Psychiatry, 148*(4), 421-431.

Grob, G. N. (1994). *The mad among us*. New York: Free Press.

House, A. E. (2001). Effective use of DSM-IV with children. In E. R. Welfel, & R. E. Ingersoll (Eds.), *The mental health desk reference: A practice-based guide to diagnosis* (pp. 213-218). New Jersey: John Wiley & Sons.

Hyman, S. E. (2010). The diagnosis of mental disorders: The problem of reification *Annual Review of Clinical Psychology, 6,* 155-179. doi:10.1146/annurev.clinpsy.3.022806.091532

Jablensky, A. (2007). Living in a Kraepelinian world: Kraepelin's impact on modern psychiatry. *History of Psychiatry, 18*(71 Pt.3), 381-388.

Jampala, V. C., Zimmerman, M., Sierles, F. S., & Taylor, M. A. (1992). Consumers' attitudes toward DSM-III and DSM-III-R: A 1989 survey of psychiatric educators, researchers, practitioners, and senior residents, *Comprehensive Psychiatry, 33*(3), 180-185. doi:10.1016/0010-440X(92)90027-N

Johnson, B., & Christensen, L. B. (2007). Educational research: Quantitative, qualitative, and mixed approaches.

Kendler, K. S. (2009). An historical framework for psychiatric nosology. *Psychological Medicine, 39*(12), 1935-1941.

King, L. J. (1999). A brief history of psychiatry: Millennia past and present. *Annals of Clinical Psychiatry, 11*(1), 3-12.

Kirk, S. A., & Kutchins, H. (1988). Deliberate misdiagnosis in mental health practice. *Social Service Review, 62*(2), 225-237.

Kodituwakku, P. W. (2009). Neurocognitive profile in children with fetal alcohol spectrum disorders. *Developmental Disabilities Research Reviews, 15*(3), 218-224.

Kupfer, D. J., Regier, D. A., & Kuhl, E. A. (2008). On the road to DSM-V and ICD-11. *European Archives of Psychiatry & Clinical Neuroscience, 258*(Suppl 5), 2-6.

Maser, J. D., Kaelber, C., & Weise, R. E. (1991). International use and attitudes toward DSM-III and DSM-III-R: Growing consensus in psychiatric classification. *Journal of Abnormal Psychology, 100*(3), 271-279.

Mayes, R., & Horwitz, A. V. (2005). DSM-III and the revolution in the classification of mental illness. *Journal of the History of the Behavioral Sciences, 41*(3), 249-267.

McBurnett, K. (1996). Development of the DSM-IV: Validity and relevance for school psychologists. *School Psychology Review, 25*(3), 259-273.

Mead, M. A., Hohenshil, T. H., & Singh, K. (1997). How the DSM system is used by clinical counselors: A national study. *Journal of Mental Health Counseling, 19*(4), 383.

Mezzich, J. E. (2002). International surveys on the use of ICD-10 and related diagnostic systems. *Psychopathology, 35*(2-3), 72-75.

Newman, B. S., Dannenfelser, P. L., & Clemmons, V. (2007). The diagnostic and statistical manual of mental disorders in graduate social work education: Then and now. *Journal of Social Work Education, 43*(2), 297-307.

Newschaffer, C. J., Croen, L. A., Daniels, J., Giarelli, E., Grether, J. K., Levy, S. E., . . . Windham, G. C. (2007). The epidemiology of autism spectrum disorders. *Annual Review of Public Health, 28*, 235-258.

Rappo, P. D. (2002). Coding for mental health and behavioral problems: The arcane elevated to the ranks of the scientific. *Pediatrics, 110*(1), 167.

Rogler, L. H. (1997). Making sense of historical changes in the diagnostic and statistical manual of mental disorders: Five propositions. *Journal of Health and Social Behavior, 38*(1), 9-20.

Rushton, J. L., Felt, B. T., & Roberts, M. W. (2002). Coding of pediatric behavioral and mental disorders. *Pediatrics, 110*(1 Pt 1), e8. Retrieved from

Schacht, T. E. (1985). DSM-III and the politics of truth. *American Psychologist, 40*(5), 513-521.

Smith, D., & Kraft, W. A. (1983). DSM-III: Do psychologists really want an alternative? *American Psychologist, 38*(7), 777-785.

Strickland, D. C., McAllister, D., Coles, C. D., & Osborne, S. (2007). An evolution of virtual reality training designs for children with autism and fetal alcohol spectrum disorders. *Topics in Language Disorders, 27*(3), 226-241.

Szasz, T. S. (1960). The myth of mental illness. *American Psychologist, 15*(2), 113-118.

Volkmar, F. R., State, M., & Klin, A. (2009). Autism and autism spectrum disorders: Diagnostic issues for the coming decade. *Journal of Child Psychology and Psychiatry, 50*(1-2), 108-115.

Zammit, G. K. (1995). The diagnosis of mental disorders using the DSM-IV. In G. K. [. Zammit, & J. W. [. Hull (Eds.), *Guidebook for clinical psychology interns* (pp. 105-120). New York, NY, US: Plenum Press; US.

Methods of Research

Historical research, as described by Hooker (2010), is a form of qualitative research. Like other qualitative researchers, historians are interested in subjective experience, ethnography, and phenomenology (Mark, 1996). Historians and qualitative researchers use similar techniques and often attain similar looking results. While quantitative researchers embrace objectivity, qualitative and historical researchers recognise the subjectivity inherent in research and often synthesize it in their writing.

Despite similarities, there are fundamental differences between historical research and other qualitative paradigms. First, the study of history appears to have developed as a distinct discipline from that of qualitative inquiry as a whole. Though there is much crossover, history appears more closely aligned with literary traditions and the humanities, whereas other qualitative research methods appear to have stemmed to a greater extent from the social sciences (Berger, Feldner, & Passmore, 2003; Denzin & Lincoln, 2005). As such, the degree of focus on methodology between the two disciplines differs quite substantially. In qualitative research, many formal procedures in design, data collection, analysis, interpretation, and reporting have been constructed for various methodologies (Creswell, 1998; Patton, 2002). For the historian, the research design, process of data collection, writing, and other research activities are not formalized (Berkhofer, 1995). Rather, the product of historical research better resembles a literary work, typically absent of the organizational formats (i.e. methods, procedures, results, conclusions) often seen in quantitative and some qualitative

approaches (Hooker, 2010). In writing of history, analysis, interpretations, and ultimately persuasiveness relies as much or more on the writing than on the data, making the act of writing the central premise for the quality of a historical review and analysis (Berger et al., 2003).

Although not a strict demarcation, the type of data used in historical research tends to differ from that of other qualitative paradigms. Research in other qualitative domains tends to extract, review, analyse, and interpret data based on ideas in the present. Historical information in this type of research tends to be contextual and perhaps interesting, but typically does not constitute the central premise of the research (Hooker, 2010). In historical research, events and developments of the past, and social change and continuities, are central premises, though they can be relevant and meaningful to the present context. Because I hope that clinicians and other professionals who read my thesis will be moved to engage in judicious psychodiagnostic processes, modern-day meaning of historical developments are important. In addition to historical analyses, my thesis will have present-day relevance in our social context and will include recommendations for psychodiagnosticians moving forward.

Historical analyses can be valuable in health and social science research to promote societal change. Hooker (2010) suggests that historical research can assist health researchers to learn about the efficiency and consequences of processes (i.e. interventions) in the past, to avoid past errors and reinventing the wheel, and to be more influential advocates for change. Hooker cites studies of tobacco control where understanding history was used as part of advocating for

policy change. Understanding history and its research process, Hooker argues, helped advocates consider deep and novel analyses and to become more conversant in the broader social discourse that frames the social landscape, thus becoming more efficient in navigating the political landscape. Thus, knowledge and more importantly, an understanding of history in relation to the "here and now" strengthen advocates' discourse by framing it in a way to facilitate and promote political change. In the broad field of mental disorder diagnosis, and even with regard to the development of the DSM specifically, advocates for change are needed at political and community levels. Advocates in the field of mental disorder diagnosis and the DSM would improve their advocacy efforts by learning and understanding its history.

Much can be gained by reviewing the history of the DSM and the mental disorders therein, but historical information needs to be interpreted cautiously. Presentism is the tendency to interpret history based on current values rather than on knowledge of values of the past (Mayr, 1990). This idea was articulated skillfully by Herbert Butterfield in his book The Whig Interpretation of History (1965). Butterfield explained that the historian "is riding after a whole flock of misapprehensions if he goes to hunt for the present in the past" (p. 10). Reviews of the history of the DSM and mental disorders can be subject to such misapprehensions. For example, homosexuality as a disorder was included as a supplementary term for sexual deviation in DSM-I (APA, 1952) and as a specified type of sexual deviation in DSM-II (APA, 1968) [later changed to sexual orientation disorder in the same manual in 1973]. Ego-dystonic homosexuality,

included in DSM-III (APA, 1980) but removed in DSM-III-R (APA, 1987), does not refer to homosexuality as a disorder per se, but that homosexuality is for the individual unwanted and a source of distress. Unfortunately, literature reviewing the history of homosexuality in the DSMs has been "whiggish", criticizing work that was done decades ago based on present-day values (e.g., Kirby, 2003). When reviewing history, presentism needs to be avoided, and in the words of Butterfield an historian should aspire to tell an historical story free from this bias, "where everything is understood and all things are forgiven" (p. 3). In this context, eschewing presentism does not involve condoning past atrocities or despicable acts. Rather, effort is made to avoid characterizing historical figures in a negative light when they would not have been characterized in that fashion in the context of the time.

The selection of which historical sources to use and which to set aside is a subjective task based on the needs of the researcher (Mark, 1996). Selecting too much information can obscure the research focus and the central premise of the research study (Mark, 1996). The selection and exclusion of information, therefore, will occur through the process of writing. This is similar to a qualitative approach of writing as a method of knowing (Richardson, 2003).

In addition, the collection of historical data is an important consideration. Literature describing the history of something can become a work of fiction in part when the sources of information used lack credibility. Primary sources can be described as first-hand accounts of historical events. Secondary sources, are accounts of events not witnessed by the reporter of the source (Best & Kahn,

1998). Secondary sources should be used only when primary sources are not available as they may be distorted based on incomplete or misinterpreted accounts. In my thesis, I will strive to refer to primary sources. If these are not available, I will indicate that secondary sources are being used and provide rationale and contextual support when doing so. Fortunately, the main documents of interest to my study (i.e. the DSM manuals) are not only easily accessible but are published and preserved electronically and in both print and electronic form by its publishers as well.

Although relics and oral testimony constitute appropriate information sources in historical writing, my sources of information stem predominantly from written documents and responses and criticisms to those documents. Although it has declined in popularity amongst researchers in the social sciences since the advent of other qualitative methodologies, documentary research is considered an effective way of recognizing changes and constants through history and linking historical events to the present context (McCulloch, 2004).

To verify authenticity, genuineness, and accuracy, historical documents should be subject to external and internal criticism (Best & Kahn, 1998). External criticism involves establishing the authenticity of the data, whether it is true or counterfeit. With the advent of electronic communication, many copies and approximations of historical documents are available online. These copies, however, may not be completely accurate. For example, the text from a document may have been altered or entered incorrectly. For this reason, I will acquire primary copies of documents in print when available. An exception is when the

publisher of a historical document has provided a scanned copy of the document online. Internal criticism, or historical reliability, is a means of determining the accuracy of a document. Although a document can be authentic it may be of little value if the writer's trustworthiness is in question. Historic reliability of primary historic documents will be determined by considering the source of the document, its author, and its historic context. Like choosing which documents to include in this historical analysis, assessing their reliability is a subjective task (McCulloch, 2004).

Recognising the subjectivity of the researcher in historical inquiry, like qualitative inquiry, I make no qualms in stating that I bring my opinions and worldview to the process. As described by Mark (1996), "One of the problems of historical interpretation in any discipline is that it's difficult not to be a cheerleader for your own field" (p. 42). From a phenomenological-hermeneutics standpoint, my interpretations are embedded in the interpretive process (Laverty, 2003) and attempting to bracket-out my interpretations lessens meaning (LeVasseur, 2003). Although some objectivity will be sought in the collection and interpretation of historical data, my intent is not to portray a completely objective history. Rather, my intent is to spur my readers into deeper and novel analyses of the DSM and mental disorder diagnosis (Hooker, 2010).

Bibliography

APA. (1952). *Diagnostic and statistical manual of mental disorders*. Washington, DC: American Psychiatric Association.

APA. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: American Psychiatric Association.

APA. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Association.

APA. (1987). *Diagnostic and statistical manual of mental disorders - revised* (3rd ed.). Washington, DC: American Psychiatric Association.

Berger, S., Feldner, H., & Passmore, K. (Eds.). (2003). *Writing history: Theory and practice* Oxford: Oxford University Press.

Berkhofer, R. F. (1995). *Beyond the great story: History as text and discourse* . Cambridge, Mass.: Belknap Press of Harvard University Press.

Best, J. W., & Kahn, J. V. (1998). *Research in education* (8th Ed. ed.). Needham Heights, MA: Allyn & Bacon.

Butterfield, H. (1965). *The whig interpretation of history*. New York: W.W. Norton.

Creswell, J. W. (1998). *Qualitative inquiry and research design: Choosing among five traditions*. Thousand Oaks, Calif.: Sage Publications.

Denzin, N. K., & Lincoln, Y. S. (2005). The discipline and practice of qualitative research. In N. K. Denzin, & Y. S. Lincoln (Eds.), *The SAGE handbook of qualitative research* (pp. 1-32). Thousand Oaks: Sage Publications.

Hooker, C. (2010). History and social change in health and medicine. In I. Bourgeault, R. Dingwall & R. de Vries (Eds.), *The SAGE handbook of qualitative methods in health research* (pp. 265-286). Los Angeles: Sage Publications.

Kirby, M. (2003). The 1973 deletion of homosexuality as a psychiatric disorder: 30 years on. *Australian & New Zealand Journal of Psychiatry, 37*(6), 674-677.

Laverty, S. M. (2003). Hermeneutic phenomenology and phenomenology: A comparison of historical and methodological considerations. *International Journal of Qualitative Methods, 2*(3), 1.

LeVasseur, J. J. (2003). The problem of bracketing in phenomenology. *Qualitative Health Research, 13*(3), 408.

Mark, M. (1996). Qualitative aspects of historical research. *Bulletin for the Council for Research in Music Education, 130*, 38-43.

Mayr, E. (1990). When is historiography whiggish? *Journal of the History of Ideas, 51*(2), 301-309.

McCulloch, G. (2004). *Documentary research in education, history, and the social sciences*. London: Routledge Falmer.

Patton, M. Q. (2002). *Qualitative research and evaluation methods* . Thousand Oaks, Calif.: Sage Publications.

Richardson, L. (2003). Writing as a method of inquiry. In Y. S. Lincoln, & N. K. Denzin (Eds.), *Turning points in qualitative research: Tying knots in a handkerchief* (pp. 379-396). Walnut Creek, CA: AltaMira Press.

Mental Disorder Beginnings

Psychiatry and psychology have a long history dating back to pre-historic times. Archeological evidence indicates a number of cases where disks of bone were removed from skulls (King, 1999a). This process, called trepanation, is thought to be a treatment of psychiatric symptoms. Evidence of psychiatric thought continued into ancient Greece, the Roman Period, the Middle Ages, the Renaissance, and so on (King, 1999a).

The idea of biological causes or "chemical imbalance" in psychiatric conditions is not a new idea. In ancient Greece, Hippocrates suspected that illnesses of a psychological nature had a medical basis. Hippocrates also described symptoms of several mental disorders, including hysteria (somatization) and, what is now termed, post-partum depression. Hippocrates also described phobia, a term derived from Phobos, a Greek god utilized to frighten enemies (King, 1999a). Psychological differences were also presented by Hippocrates in the four humours, imbalances in: a) blood (sanguine), b) phlegm (phlegmatic), c) yellow bile (choleric), and d) black bile (melancholic), which led to disease. Galen expanded on this idea and suggested that the humours were associated with personality (Axis II, as introduced in DSM III). Excess of blood, for example, was associated with cheerfulness, and excess black bile with sadness. Galen also focussed on treating illness in a holistic way (Mesulam & Perry, 1972).

Avicenna, an Arab physician influenced by Greek philosophers, including Hippocrates and Galen, authored the Canon of Medicine in which he described disorders of the nervous system and the psyche (King, 1999a). As an example, Avicenna described a disorder of "Love", a condition similar to severe depression

and obsessive thoughts. Love disorder was thought to be caused by repeated thoughts of a person of the opposite sex, and was manifest by anxiety, depression, and several physiological symptoms such as irregular pulse and heart palpitations (Mesulam & Perry, 1972). This condition as described by Avicenna appears to resemble adjustment disorder (Shoja & Tubbs, 2007). Avicenna also observed that anger, restlessness, and violence led to a transition in some patients from melancholy to mania (Vakili & Gorji, 2006), resembling bi-polar disorder. Avicenna's Canon also covers other areas related to mental disorder, including deficiency in memory and imagination, mental confusion, frivolity and stupidity, and lycanthropy (related to melancholia). Avicenna also described in length a number of hallucinations and delusional states which he attributed to melancholy (Dols, 2006).

There are many other characters in ancient medicine who described a number of disorders that may resemble conceptualizations of mental disorders at present. The history of the discipline of psychiatry is broad and extensive, and can be reviewed elsewhere (King, 1999a; King, 1999b). In this paper, an evaluation of historical data will focus on mental disorders and their precursors since the eighteenth century.

Eighteenth to Early-Twentieth Centuries

Although the first edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM) was not published until 1952, mental disorders were topics of interest in the preceding centuries. The advent of institutionalization, though periodically viewed as a dark period in the history of psychiatry (King,

1999b), provided substantial opportunity to collect data and learn about mental disorders in a clinical context. Mental disorders began to be viewed through a medical lens, spurring the development of psychiatry as a profession. The work of influential figures in the development of psychiatry, such as Phillipe Pinel and Emil Kraepelin, was centred in the study of mental disorders.

The debate as to whether the mentally disabled are better treated in institutions or the community has been occurring since the eighteenth century. At present, inclusion and integration into the community is the preferred standard, but this viewpoint is not new. In the eighteenth century, the mentally disabled were supported within the community and by their own families when possible. Increased population growth in urban centres created a strain on communities to care for the mentally disabled. From this need institutions were formed to "fulfill humanitarian and moral obligations" (p. 17) to the disabled in an efficient way (Grob, 1994). Unfortunately, conditions in many institutions seemed to degrade to the point that they appeared neither humane nor moral.

Philippe Pinel, who, beginning in the late 18th century, promoted the humane treatment of the mentally ill, found that inhabitants of the institutions Bicetre and Salpetriere endured appalling conditions, including being caged and chained. Pinel ordered that the chains be removed from the insane and subsequently promoted institutions where the mentally ill would be safe and treated respectfully (Harris, 2003; Weiner, 1992). One of Pinel's most important contributions was the concept of moral treatment, which involves increasing morale as a therapeutic measure (Kauffman, 1976). Samuel Woodward, first

president of the Association of Medical Superintendents of American Institutions for the Insane (presently called the American Psychiatric Association [APA]), was superintendent of the influential mental hospital in Worcester, Massachusetts. The idea that insanity was curable, or at least treatable, stemmed from successful rehabilitations resulting from medical treatments and the aforementioned moral treatments at this hospital (Grob, 1994). Dorothea Dix (1843), who advocated for the mentally disordered who were ill-treated in almshouses and asylums, stated that:

> Hospitals are the only places where insane persons can be at once humanely and properly controlled. Poorhouses converted into madhouses cease to effect the purposes for which they were established, and instead of being asylums for the aged, the homeless, and the friendless, and places of refuge for orphaned or neglected childhood, are transformed into perpetual bedlams. (para. 58)

Dix did much as an advocate for "suffering humanity" especially when one considers the political disadvantage women held at the time.

Although institutionalization during the eighteenth and nineteenth centuries can be viewed as barbaric from the modern lens, many of these negative aspects may be exaggerated. First, we may be subject to the presentist bias. Much has changed in the past few centuries regarding the status of the individual. Second, much of the authoritative writing on institutionalization was based on secondary sources (Kauffman, 1976; Prins, 1987). Allderidge (2004), for example, indicated that only two accounts of Bethlem Royal Hospital (Bedlam)

come from primary sources, and those being rather inaccessible at that. Allderidge acknowledged that many injustices likely occurred at Bedlam but added that positive treatment was rarely if ever reported through these accounts, that case examples frequently left out important context, and that profoundly negative accounts were presented as the rule when they were likely the exception. Third, there is much evidence to suggest that asylums were providing schooling and educational programs for children and adults with intellectual and behavioural disorders (Bockoven, 1956; Kauffman, 1976). The view that institutions did not live up to their "humane and moral" commitments should not be discounted but should be evaluated critically.

Pinel, Kraepelin, and other psychiatrists such as Wilhelm Griesinger and Eugen Bleuler authored psychiatric nosologies. These nosologies were essentially created from a top-down approach, using *a priori* assumptions to generate classifications (Kendler, 2009). These nosologies addressed biological, relational, and environmental factors that influenced presentation of symptoms (Grob, 1991). In reality, psychiatrists had little to go on outside of symptoms and extraneous factors. Interestingly, the DSM did not formally incorporate environmental factors into its classifications until the third edition when the multi-axial approach was introduced.

Individual nosologies put forth by psychiatrists at this time tended to be more holistic and centred on the individual. These nosologies provided an important starting point for the development of a diagnostic language for psychiatrists. There were, however, a number of challenges inherent in an array of

different nosologies. First, different nosologies meant different diagnostic languages were spoken, impeding communication between psychiatrists. Second, diagnostic similarities and differences between nosologies were difficult to distinguish because each nosology was based on different constructs of mental disease as conceptualized by different authors. Third, prevalence rates of mental disorders could not be determined without a standard nosology. These challenges would impede refinement and validation of these psychiatric nosologies, collaboration, prevalence estimates, and research. With a standardized nosology, psychiatrists would be able to learn and speak the same diagnostic language.

Coming to a standard psychiatric nosology would be challenging. Kendler (2009) articulated this challenge well by relating the development of biological taxonomy to psychiatric nosology. When biological taxonomies emerged in the 16th and 17th centuries, each emphasized different traits that were considered essential. What is the essential trait, for example, of a whale? That it is mammalian, that it lives in water, perhaps its size? Once essential traits can be determined (or at least justified) classification can proceed. For example, if habitat was determined to be the essential trait, whales could be grouped with sea animals rather than mammals.

Classification of mental disorders, unfortunately, is more complex. In his treatise, Isaac Ray (1871) suggested that no classification of mental disease could be "rigourously correct; for such divisions have not been made in nature and cannot be observed in practice" (p. 84). Determining which of those traits are

more essential than others is still a concern as numerous disorders in DSM-V are expected to undergo fundamental changes.

Psychiatric nosology increasingly became a topic of interest at the turn of the century. A number of factors seem to have influenced this shift, including social and political trends. Developing fields in social sciences, statistics, and the federal census, for example, provided the impetus to look at phenomena in a broad, generalizable way, mental disorders included. Subsequently, there was particular attention paid to ethnic and racial factors in a special census in 1904 to collect data on the institutionally insane (Grob, 1991). These factors, together with the notion of heritability of intelligence (Goddard, 1912) helped fuel the mental hygiene and eugenics movements.

In 1917, the Committee on Statistics of the American Medico-Psychological Association (later called the American Psychiatric Association) recommended a uniform classification system of mental disease. At the time, mental disorder classification was chaotic and did not allow for the collection of meaningful data. Clinicians and researchers were diagnosing mental disorders with little consistency. There was great confusion and variability in diagnoses of mental disorders because diagnostic systems were about as varied as the institutions and individuals that created them (Kendall, 1975). This problem was further compounded over time, as different diagnostic nomenclature spread across North America. This resulted in what was described in DSM-I (APA, 1952) as "a polyglot of diagnostic labels and systems, effectively blocking communication and the collection of medical statistics" (p. V). In addition, the committee feared

that having such a disordered way of classifying mental disease would discredit the field of psychiatry. Subsequently, the association published the Statistical Manual for the Use of Institutions for the Insane (Committee on Statistics of the American Medico-Psychological Association in collaboration with the National Committee for Mental Hygiene, 1918), which delineated mental disorders into 22 groups. The manual became the definitive psychiatric nosology and continued in a number of editions until the 10th edition in 1942.

Some psychiatrists were opposed to a psychiatric nosology. Adolf Meyer, former president of the APA, and "Dean of American Psychiatry", was opposed to a nosology demarcating "a one-word diagnosis marking the individual" (Meyer, 1919, as cited in Grob, 1991). Rather, Meyer viewed mental illness in holistic terms, and was a proponent of understanding the life histories of patients to understand the etiologies of mental disorders (Sabshin, 1990). Meyer introduced a psychiatric nosology based on reaction patterns, but it was largely unused by psychiatrists. Meyer abandoned the nosology because he found each psychiatric case was unique and should be studied on its own terms (Neill, 1980).

A significant shift in psychiatric nosology occurred in the United States as a result of World War II. In the Armed Forces, each case of morbidity required a diagnosis by a psychiatrist, but only about 10% of cases fell within the nomenclature of traditional diagnoses as described in the Statistical Manual (APA, 1952). Psychiatrists serving in the military also found that the early identification and treatment of mental illness in non-institutional settings could help alleviate and prevent more serious mental health problems. This resulted not

only in a rekindling of the institution/community debate of treating the mentally ill, but also in increased optimism in the treatment of mental disorders (Grob, 1991). Because it was recognized through these wartime efforts that environmental stressors contribute to mental illness and that intervention could alleviate psychological problems, it also reawakened interest in psychodynamic and psychoanalytic theories, which emphasizes the psychological processes that moderate environmental forces with biological drives. This led to a shift away from the medical model of psychiatry.

In terms of the development of the DSM, one of the most important changes was that previous terminology, which was expressed in pathological language, underwent significant changes. The new terminology focused less on biological bases of behaviour and more on developmental, environmental, and relational factors. Further updates to the Statistical Manual were put on hold. In addition, the United States Army made extensive revisions to the standard nomenclature, which was eventually adopted by all American Armed Forces. The U.S. Veterans Administration followed suit with their own nomenclatures, similar to those of the Armed Forces.

The 6th revision of the International Statistical Classification (International Classification of Diseases, or ICD) was produced in 1948 (WHO, 1948), which included for the first time a section on mental disorders entitled "Mental, Psychoneurotic, and Personality Disorders" (p. 106). The International Classification delineated these disorders into three broad groups of a) Psychoses, b) Psychoneurotic Disorders, and c) Disorders of Character, Behaviour, and

Intelligence, with 26 categories and 61 sub-categories in a rubric style classification. In the end, at least three nomenclatures were widely used, none of which was in line with the International Statistical Classification (APA, 1952). The journey toward a standard classification had taken a detour.

DSM

The first edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-I) was an important development toward a standard nosology of mental disorders. This manual offered "a completely new classification in conformity with newer scientific and clinical knowledge, simpler in structure, easier to use and virtually identical with other national and international nomenclatures" (APA, 1952, p. 1).

Disorders in the DSM-I were presented descriptively, unlike the rubric-like presentation of mental disorders in ICD-6. DSM-I delineated 106 mental disorders into two broad groupings, a) Disorders Caused by or Associated with Impairment of Brain Tissue Function, and b) Disorder of Psychogenic Origin or Without Clearly Defined Physical Cause or Structural Change in the Brain. In this way, the DSM-I implied causality in its delineation of mental disorders. The first grouping was subdivided into Acute Brain Disorders, Chronic Brain Disorders, and Mental Deficiency. The second was subdivided into Psychotic Disorders (including affective and schizophrenic reactions), Psychophysiologic Autonomic and Visceral Disorders (Psychophysiologic reactions, which appear related to somatization), Psychoneurotic Disorders (including Anxiety, Phobic, Obsessive Compulsive, and Depressive reactions), Personality Disorders (including Schizoid

personality, Antisocial reaction, and Addiction), and Transient Situational Personality Disorders (including Adjustment reaction and Conduct disturbance). It is interesting to note that Learning and Speech disturbances are categorized as Special Symptom reactions under Personality Disorders.

The DSM-I was highly influenced by the prevalence of psychodynamic theory in North America and therefore referred to disorders as 'reactions' (King, 1999b). Its move toward a psychodynamic orientation represented changes within psychiatry in the United States. The profession of psychiatry itself was shifting in a psychodynamic direction, highly influenced by the advent of psychotherapy post-World War II.

After publication of the DSM, it became necessary to coordinate it with the ICD, which proved to be a daunting task based on the different orientations and purposes of the manuals, the former used to provide nomenclature for clinical activities, and the latter for statistical classification. The seventh revision of the ICD was slated in 1955, but the mental disorder section of ICD was not updated until the following edition, which was published in 1968 as ICD-8 (Millon, 1986).

DSM-II

While working with the WHO on the development of the mental disorder section of ICD-8, the APA initiated revisions of the DSM to bring it in line with the International Classification (APA, 1968). By the end, however, several minor differences between the nosologies remained to keep the DSM in line with its use in the United States (Millon, 1986).

Both the DSM-I and DSM-II held similar theoretical stances, grounded in psychodynamics. There were, however, some noteworthy differences between the two manuals. In DSM-II there was an attempt to remove particular theoretical stances in the nomenclature. Although general psychodynamic formulations of mental disorders were retained, nomenclature was carefully selected to avoid terms implying causality (APA, 1968). The term "reactions" was removed from diagnostic labels as it espoused a "psychobiologic" orientation (Rogler, 1997). Other theoretical terms, particularly those with psychoanalytic formulations, were not removed, including the terms neuroses and psychophysiologic disorders. In this way, the shift away from a theoretical orientation was only partial.

DSM-III

Another significant shift in psychiatric nosology took place in the time between the publications of the 2nd and 3rd editions of the DSM. By the 1960s, psychiatry as a profession was predominantly psychodynamic, which resulted in some ideological put perhaps unrealistic thinking. Successes in returning soldiers to the front in WWII created perhaps an unrealistic expectation of the curability of mental illness. With its psychodynamic roots, psychiatric diagnosis became increasingly viewed as unreliable, overly subject to interpretation, and not clearly defined. The unreliability of diagnosis came under scrutiny, and there was growing public contempt in the United States, particularly over conflicting testimonies of psychiatrists in insanity defense pleas. As a result, professional and public perception of the field of psychiatry began to deteriorate. In part due to these pressures, the profession of psychiatry underwent significant theoretical

changes toward an empirical, positivistic orientation. The field of psychiatry reverted to an orientation based on the ideas of Emil Kraepelin (Kihlstrom, 2002).

Kraepelin is well known for his distinction of manic-depressive insanity and dementia praecox (later named schizophrenia by Eugen Bleuler). Kraepelin's views of mental disorder, "often quoted, occasionally misquoted and at times hotly debated" (Jablensky, 2007, p. 381), significantly influenced the field of psychiatry. Kraepelin kept longitudinal data on his patients, thus allowing for the comparison of clinical outcomes over time. Some of Kraepelin's core ideas include relating psychiatry with medicine, using descriptive language, linking patho-anatomical observation with psychopathological observation, and observing psychiatry through an empirical lens.

Kraepelin's influence on psychiatry re-emerged in the 1960s, about 40 years after his death, with a small group of psychiatrists at Washington University in St. Louis, Missouri, who were dissatisfied with psychodynamically-oriented American psychiatry. Eli Robins, Samuel Guze, and George Winokur, who sought to return psychiatry to its medical roots, were called the neo-Kraepelinians (Klerman, 1978). They were dissatisfied with the lack of clear diagnoses and classification, low inter-rater reliability among psychiatrists, and blurred distinction between mental health and illness. To address these fundamental concerns, these psychiatrists advocated descriptive and epidemiological work in psychiatric diagnosis, and to avoid speculating on etiology. This orientation closely matches Kraepelin's published views of psychiatry (Decker, 2007).

Klerman, who coined the term neo-Kraepelinians, outlined the neo-Kraepelinian credo (1978):

1. Psychiatry is a branch of medicine.

2. Psychiatry should utilize modern scientific methodologies and base its practice on scientific knowledge.

3. Psychiatry treats people who are sick and who require treatment for mental illness.

4. There is a boundary between the normal and the sick.

5. There are discrete mental illnesses. Mental illnesses are not myths. There is not one but many mental illnesses. It is the task of scientific psychiatry, as of other medical specialties, to investigate the causes, diagnosis, and treatment of these mental illnesses.

6. The focus of psychiatric physicians should be particularly on the biological aspects of mental illness.

7. There should be an explicit and intentional concern with diagnosis and classification.

8. Diagnostic criteria should be codified, and a legitimate and valued area of research should be to validate such criteria by various techniques. Further, departments of psychiatry in medical schools should teach these criteria and not depreciate them, as has been the case for many years.

9. In research efforts directed at improving the reliability and validity of diagnosis and classification, statistical techniques should be utilized. (p. 104-105)

This credo aligns closely with Kraepelin's published views of psychiatry (Jablensky, 2007).

In 1972, John Feighner and his "neo-Kraepelinian" colleagues published a set of diagnostic criteria based on a synthesis of research, pointing out that the criteria were not based on opinion or tradition. In addition, explicit criteria were used to increase reliability (Feighner et al., 1972). The classifications therein became known as the 'Feighner criteria'. This publication by Feighner et al. became a landmark paper, eventually becoming the most cited article published in a psychiatric journal (Decker, 2007). Blashfield (1982) suggests that Feighner's article was highly influential, but that the large number of citations (over 140 per year at that point, compared with an average of about 2 per year) may have been in part due to a disproportionate number of citations from within the "invisible college" of the neo-Kraepelinians.

The change in the theoretical orientation of American psychiatry toward an empirical foundation is perhaps best reflected in the 3rd edition of the DSM. Robert Spitzer, Head of the Task Force on DSM-III, was previously associated with the neo-Kraepelinians (Blashfield, 1982) and many were on the DSM-III Task Force (Decker, 2007), but Spitzer denied being neo-Krapelinian himself. In fact, Spitzer facetiously resigned from "the neo-Kraepelinian college" (p. 952) on account that he did not subscribe to some of the tenets of the neo-Kraepelinian credo presented by Klerman (Spitzer, 1982). Nevertheless, the DSM-III appeared to adopt a neo-Kraepelinian standpoint and in the process revolutionized psychiatry in North America.

The DSM-III, compared with its predecessors, was a vastly different manual. It was centred on descriptive criteria, removing etiological theory, presenting psychiatry in a medical model, and emphasizing follow-up and family histories. It sought to increase the reliability of diagnosis and facilitate communication amongst mental health professionals. The introduction of the DSM-III emphasizes the importance of having a common diagnostic language:

> Clinicians and researchers must have a common language with which to communicate about the disorders for which they have professional responsibility…The efficacy of various treatment modalities can be compared only if patient groups are described using diagnostic terms that are clearly defined. (APA, 1980, p. 1)

According to many clinicians and researchers worldwide, significant advantages of the DSM-III over prior editions included the manual's atheoretical stance, clinical descriptions that were more comprehensive and systematic, discrete categories, and the multiaxial format (Maser, Kaelber, & Weise, 1991). In this way, The DSM-III succeeded in many ways in following a neo-Kraepelinian approach. The inclusion of the multiaxial format acknowledges biopsychosocial factors that can impact a patient's mental health. Spitzer explained how the multiaxial system came about, which interestingly was in response to criticism:

> The actual impetus for this was to meet the mounting criticism that by developing such a large and seemingly authoritative diagnostic manual, American psychiatry was giving the impression that the only important part of a psychiatric evaluation was making (or not making) a psychiatric

> diagnosis. By providing a multiaxial system that included physical disorders (Axis III), psychosocial stressors (Axis IV), and level of functioning (Axis V) enabled DSM-III to be presented as within a broad biopsychosocial model—rather than the narrow diagnostic model that its critics feared…What values justify such a strategy? It is better to win (by offering your critics something) than to lose (offer them nothing and have the entire project stopped—as several times seemed possible). (Spitzer, 2001, p. 357-358)

Contrary to a neo-Kraepelinian standpoint, expert consensus was often used to inform decision-making. Empirical research was utilized when possible but much of the categorization was based on clinical judgment (APA, 1980; Spitzer, 1991). Had the DSM-III Task Force limited themselves to disorders with sound empirical backing (16 disorders based on the Feighner criteria), it would not have provided criteria for the vast majority of disorders in DSM-III, which arguably have clinical and research value.

Shortly after the publication of DSM-III, it became clear that some of the diagnostic criteria therein were contradictory and vague (APA, 1987). In addition, the validity of the disorders were explored through field trials (Widiger & Clark, 2000). Subsequently, a revised edition of the DSM-III was published in 1987 which included some revised descriptions of diagnostic criteria and a new appendix of "Proposed Diagnostic Categories Needing Further Study".

DSM-IV

The structure and theoretical orientation of the DSM-IV was largely unchanged from the DSM-III; a descriptive orientation and multiaxial system remained. The number of mental disorders increased from over 200 in DSM-III to over 300 in DSM-IV. The threshold for approval of a diagnosis in DSM-IV, however, was more conservative, requiring more empirical backing (Widiger & Clark, 2000). In this way, perhaps, the DSM-IV seems to have inched closer to a neo-Kraepelinian ideal compared with the DSM-III. Little else, however, distinguishes the two manuals.

The DSM-IV - Text Revision was published to ensure that information in the DSM-IV remained up-to-date. No substantive changes were made to the diagnostic criteria set out in DSM-IV, and neither new disorders nor new subtypes were considered. Rather, literature-based updates were made in several sections, including Prevalence, Course, Familial Patterns, Associated Features and Disorders, and Specific Culture, Age, and Gender Features (APA, 2000, p. XXX).

DSM-V

Although DSM-IV was still warm from the press when researchers began commenting and speculating about DSM-V (i.e., Schuckit, 1994), the initial phase of the DSM-V planning process began in 1999 with a series of conferences co-sponsored by APA and the National Institute of Mental Health (NIMH). These conferences culminated in six “white papers” intended to stimulate discussion and research for the eventual DSM revision process. These white papers, published as book chapters, addressed a number of research directions, including re-

conceptualizing mental disorder, establishing a classification based on etiology, introducing dimensional rather than categorical classifications, and re-evaluation of the multiaxial format (Kupfer, First, & Regier, 2002).

Although the neo-Kraepelinian approach to classification in DSM-III and DSM-IV improved reliability and communication among clinicians and researchers, it was clear that it was more limited in establishing validity of mental disorders. Etiology was not unimportant from a neo-Kraepelinian standpoint. Rather, it was suggested that validity of syndromes could be improved through clinical descriptions, laboratory studies, delimitation of disorders, follow-up, and family histories, which would then establish etiology (Robins & Guze, 1970). Establishing etiology by this means, however, was problematic due to high rates of co-morbidity between disorders and the lack of any biological marker that could identify a single mental disorder (Kupfer, Regier, & Kuhl, 2008). Incorporating etiology into DSM-V was therefore considered.

The inclusion of discrete categories in DSM-III also emerged from the neo-Kraepelinian standpoint, which has been challenged in DSM-V. A number of researchers indicated that a more dimensional approach in DSM-V was needed because co-morbidity rates suggest common underlying dimensions to current disorders. The dimensional approach has the advantage of being more effective in defining impairment, symptom severity, and quality of life issues (Helzer et al., 2008), which are not well described under the current classification.

At present, the DSM-IV-TR and ICD-10 represent the dominant diagnostic languages in the world. Traditionally, revisions to the DSMs and ICDs

have occurred independently (Kupfer et al., 2008), which has created a number of problems. The majority of disorders in both manuals have differences between them, with about 21% having conceptually-based differences (First, 2009). Such differences undermine the credibility of the field of psychiatry, and having two different classification systems can impeded international collaboration efforts. From 2003 to 2008, the APA and the World Health Organization (WHO) held a series of international conferences aimed at generating research leading to revisions to their respective diagnostic classifications. Harmonization of these classifications has been identified as an important goal of the revisions of both manuals. One step that has been proposed in DSM-V is the amalgamation of axes I, II, and III into one axis that contains all psychiatric and general medical conditions. This would bring the DSM more in line with the ICD approach.

In February, 2010, the APA released Proposed Draft Revisions to DSM Disorders and Criteria (APA, n.d.). Many of these proposed changes reflect the shift toward etiologically-based, dimensional diagnoses. For example, one proposed change is the inclusion of an anxiety dimension across all mood disorders. In the categorical approach in DSM-III and DSM-IV, anxiety is identified as a separate and distinct construct from other mood disorders, whereas the proposed changes in DSM-V suggest that anxiety may be a common underlying factor. A significant shift from a categorical to a dimensional orientation is reflected in the proposed changes for Pervasive Developmental Disorders. Presently, Autistic Disorder, Asperger's Disorder, and Pervasive Developmental Disorder – Not Otherwise Specified are distinct categories in the

DSM-IV-TR. The proposed changes would eliminate these categories and place these disorders within the classification Autism Spectrum Disorder. Perhaps the most significant change may take place with the Personality Disorders, where diagnoses may be based on underlying traits that require a dimensional approach. Not all disorders are proposed to incorporate such changes, but the etiological, dimensional approach of the DSM-V is likely to impact how clinicians, researchers, and the general public view mental disorders given the influence of the DSM (First & Frances, 2008).

Conclusion

Due to its iconic status (rather than out of reverence), the DSM has frequently been referred to as "the bible" for mental disorder diagnosis (Anand & Malhi, 2011). The DSM is used in clinical and research contexts throughout the world and few texts match its influential power. The DSM has undergone an interesting history, and understanding that history can help clinicians and researchers better understand and use this important text.

There are a number of factors that spurred the development of the first edition of DSM, with perhaps most important being the need for a common diagnostic language. The DSM provided this within the North American context. Even from the beginning, however, there were fundamental differences between the DSM and the ICD, both of which developed in different contexts for different purposes (Jablensky, 2009). Although some efforts have been made to harmonize the manuals, changes to prior editions of the DSM have occurred independently of revisions to the ICD (Kupfer et al., 2008).

Having one standard, worldwide diagnostic language, however, was probably less important when the first edition of the DSM was published in the mid-twentieth century. Worldwide communication was less sophisticated, so international collaboration was less common. It was therefore probably more acceptable for clinicians and researchers across the sea to speak a different diagnostic language. Had international collaboration been as prominent in the mid-twentieth century as it is today, it is possible that the already existing ICD-6 would have been adopted in North America, and the highly influential DSM may have never come into existence.

As international collaboration becomes increasingly more common, continued harmonization of the DSM and ICD is needed. This does not mean, however, that the DSM and ICD are likely to adopt an identical nosology. Given the prominence of both manuals, it is unlikely that one will be relinquished. For this reason, the endeavor to establish one standard, worldwide nosological language is unlikely to ever come to pass. However, having two standard nosological languages reminds us that there are many classifications in both manuals that are provisional and even arbitrary (Jablensky, 2009). Perhaps having two harmonized but separate manuals gives the advantage of reminding us not to become too comfortable with our existing nosologies.

A dimensional orientation to DSM-V may result in some communication growing pains. Using a dimensional approach requires the broadening of diagnostic language, and for a time many clinicians and researchers may become lost in the translation. For example, according to our current manual, differences

between Autistic Disorder and Asperger's Disorder are clearly delineated (Asperger's Disorder presents with the same deficits of Autistic Disorder except for cognitive delay and impairment in communication). With the proposed Autism Spectrum Disorder, differences between individuals and groups within that spectrum may be more difficult to delineate. It is essential that differences on dimensions be quantified so that individuals and groups can be delineated reliably and so that communication efforts are not impeded.

There have been concerns that incorporating a dimensional approach would increase false positive diagnoses dramatically because of the breadth of diagnosable profiles (Frances, 2010). This concern, however, is already present with our current nosology in the form of the Not Otherwise Specified (NOS) category for some disorders, which is perhaps not surprisingly the most frequently provided diagnosis in clinical practice (Clark, Watson, & Reynolds, 1995). For example, Pervasive Developmental Disorder (PDD) in DSM-IV-TR includes five possible diagnoses; Autistic Disorder, Asperger's Disorder, Rett's Disorder, Childhood Disintegrative Disorder, and PDD – NOS. A diagnosis of PDD – NOS may be given when there is marked impairment in the features associated with a PDD but when the criteria is not met for a specific PDD. Although Autistic Disorder and Asperger's Disorder have generated most research interest, prevalence estimates suggest that diagnoses of PDD – NOS outnumber all other PDDs combined (Fombonne, 2005). Fittingly, the proposed change to these disorders in DSM-V is subsuming them within Autism Spectrum Disorder, except Rett's Disorder, which is proposed for possible removal.

NOS is a limited dimensional concept in a categorical classification. The advantage of the dimensional approach proposed in DSM-V over NOS in DSM-IV-TR is that the dimensional approach can be quantified to estimate where on the dimension a patient lies. This is not the case in the categorical, quasi-dimensional NOS. Given the high prevalence but limitations on specificity of the current NOS system, false positive diagnoses are not likely to increase with the dimensional approach. Rather, false positives may decrease, or if not, spectrum diagnoses will at least be more functionally specific. The crux of the effectiveness of the dimensional approach lies in if and how it is quantified.

Using quantifiable ranking scales can create a more powerful diagnostic tool (Helzer, Kraemer, & Krueger, 2006). Psychotic disorders such as schizophrenia are proposed to be subsumed under an attenuated psychotic symptoms syndrome, with an assessment of symptom severity on a four-point scale within nine dimensions (i.e. hallucinations, disorganization, etc.). This type of dimensional approach should facilitate clinically meaningful diagnoses while maintaining communication between professionals. A similar system should be proposed for other spectrum disorders within DSM-V by identifying the most salient features of those disorders and constructing a scale for each symptom using criteria that are as objective as possible. Such a system would take advantage of the strengths of both dimensional and categorical approaches.

The definition of mental disorder presented in DSM-IV-TR (APA, 2000, p. XXXi) indicates that a syndrome or pattern that causes distress or disability can constitute a mental disorder. Establishing the level of distress or disability,

however, has been a challenge with the categorical approach. The dimensional approach in DSM-V may prove valuable in determining levels of disability, distress, and other quality of life factors (Helzer et al., 2008).

The evolution of each edition of the DSM presents an interesting trend. The first edition, influenced by circumstances of WWII and the treatability of mental illness, emphasized psychodynamic theory and implied causality within its diagnostic categories. DSM-II took a step toward an empirical, positivistic orientation by removing assumptions of causality, but retained its psychodynamic orientation. DSM-III took significant strides toward a logical positivistic and empirical orientation. "Quantification and objectification moved rapidly, perhaps at times too rapidly, across our entire field. Implicitly, theory building receded and an effort was made to move toward an empirical, quantitative direction where logical positivism and its empirical modes prevailed" (Sabshin, 1990, p. 1272). DSM-IV was a very similar manual to the DSM-III, and emphasized a slightly more empirical approach, requiring more empirically-based criteria for diagnosis. The development of DSM-V thus far suggests that it is likely to retain most of the changes brought about by DSM-III. However, it is likely to emphasize etiology to a greater degree, signaling a slight return toward a DSM-I orientation (though not to the same degree as DSM-I emphasized causality). The development of DSM, from beginning to present, resembles an historic pendulum, from DSM-I on the one hand emphasizing psychodynamics and causality, to DSMs-III and IV emphasizing empiricism and logical positivism. The proposed changes in etiological and dimensional-based classification for DSM-V appear to represent a

shift toward the centre. Time will tell whether the pendulum continues to swing and whether we are approaching an ideal nosology.

Bibliography

Allderidge, P. (2004). Bedlam: Fact or fantasy? In W. F. Bynum, R. Porter & M. Shepherd (Eds.), *The anatomy of madness: Essays in the history of psychiatry* (pp. 17). London: Routledge.

Anand, S., & Malhi, G. S. (2011). From manual to bible: The questionable hegemony of DSM IV *The Australian and New Zealand Journal of Psychiatry, 45*(5), 348-350. doi:10.3109/00048674.2011.576814

APA. (1952). *Diagnostic and statistical manual of mental disorders*. Washington, DC: American Psychiatric Association.

APA. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: American Psychiatric Association.

APA. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Association.

APA. (1987). *Diagnostic and statistical manual of mental disorders - revised* (3rd ed.). Washington, DC: American Psychiatric Association.

APA. (2000). *Diagnostic and statistical manual of mental disorders - text revision* (4th ed.). Washington, DC: American Psychiatric Association.

APA. (n.d.). *Proposed draft revisions to DSM disorders and criteria.* Retrieved June 5, 2010, from www.dsm5.org

Blashfield, R. K. (1982). Feighner et al., invisible colleges, and the matthew effect. *Schizophrenia Bulletin, 8*(1), 1-12.

Bockoven, J. S. (1956). Moral treatment in american psychiatry. *Journal of Nervous & Mental Disease, 124*(3), 292-321.

Clark, L. A., Watson, D., & Reynolds, S. (1995). Diagnosis and classification of psychopathology: Challenges to the current system and future directions. *Annual Review of Psychology, 46*, 121-153.

Committee on Statistics of the American Medico-Psychological Association in collaboration with the National Committee for Mental Hygiene. (1918). *Statistical manual for the use of institutions for the insane*. New York: National Committee for Mental Hygiene.

Decker, H. S. (2007). How Kraepelinian was Kraepelin? how Kraepelinian are the neo-Kraepelinians?--from Emil Kraepelin to DSM-III. *History of Psychiatry, 18*(71 Pt 3), 337-360.

Dix, D. (1843). *Memorial to the legislature of Massachusetts.* Retrieved from http://www.disabilitymuseum.org/lib/docs/737.htm

Dols, M. W. (2006). Medical madness. *Journal of Muslim Mental Health, 1*(1), 77-95.

Feighner, J. P., Robins, E., Guze, S. B., Woodruff, R. A. Jr, Winokur, G., & Munoz, R. (1972). Diagnostic criteria for use in psychiatric research. *Archives of General Psychiatry, 26*(1), 57-63.

First, M. B. (2009). Harmonisation of ICD-11 and DSM-V: Opportunities and challenges. *British Journal of Psychiatry, 195*(5), 382-390.

First, M. B., & Frances, A. (2008). Issues for DSM-V: Unintended consequences of small changes: The case of paraphilias. *The American Journal of Psychiatry, 165*(10), 1240-1241.

Fombonne, E. (2005). The changing epidemiology of autism. *Journal of Applied Research in Intellectual Disabilities, 18*(4), 281-294.

Frances, A. (2010). The first draft of DSM-V. *BMJ, 340*, 1168. Retrieved from http://dx.doi.org/10.1136/bmj.c1168

Goddard, H. H. (1912). *The Kallikak family: A study in the heredity of feeble-mindedness.* New York, NY, US: MacMillan Co.

Grob, G. N. (1991). Origins of DSM-I: A study in appearance and reality. *American Journal of Psychiatry, 148*(4), 421-431.

Grob, G. N. (1994). *The mad among us*. New York: Free Press.

Harris, J. C. (2003). Pinel orders the chains removed from the insane at bicetre. *Archives of General Psychiatry, 60*(5), 442.

Helzer, J. E., Kraemer, H. C., & Krueger, R. F. (2006). The feasibility and need for dimensional psychiatric diagnoses. *Psychological Medicine, 36*(12), 1671-1680.

Helzer, J. E., Kraemer, H. C., Krueger, R. F., Wittchen, H., Sirovatka, P. J., & Regier, D. A. (Eds.). (2008). *Dimensional approaches in diagnostic classification: Refining the research agenda for DSM-V.* Washington, DC: American Psychiatric Association.

Jablensky, A. (2007). Living in a Kraepelinian world: Kraepelin's impact on modern psychiatry. *History of Psychiatry, 18*(71 Pt 3), 381-388.

Jablensky, A. (2009). Towards ICD-11 and DSM-V: Issues beyond 'harmonisation'. *British Journal of Psychiatry, 195*(5), 379-381.

Kauffman, J. M. (1976). Nineteenth century views of children's behavior disorders: Historical contributions and continuing issues. *The Journal of Special Education, 10*(4), 335-349.

Kendall, R. E. (1975). *The role of diagnosis in psychiatry.* London: Basil Blackwell.

Kendler, K. S. (2009). An historical framework for psychiatric nosology. *Psychological Medicine, 39*(12), 1935-1941.

Kihlstrom, J. F. (2002). To honor Kraepelin...: From symptoms to pathology in the diagnosis of mental illness. In L. E. Beutler, & M. L. Malik (Eds.),

Rethinking the DSM: A psychological perspective (pp. 279-303). Washington, DC, US: American Psychological Association.

King, L. J. (1999a). A brief history of psychiatry: Millennia past and present. *Annals of Clinical Psychiatry, 11*(1), 3-12.

King, L. J. (1999b). A brief history of psychiatry: Milliennia past and present--part II. *Annals of Clinical Psychiatry.Vol 11(2), 47-54.*

Klerman, G. L. (1978). The evolution of a scientific nosology. In J. C. Shershow (Ed.), *Schizophrenia: Science and practice* (pp. 91-121). Cambridge, MA: Harvard University Press.

Kupfer, D. J., Regier, D. A., & Kuhl, E. A. (2008). On the road to DSM-V and ICD-11. *European Archives of Psychiatry & Clinical Neuroscience, 258*(Suppl 5), 2-6.

Kupfer, D. J., First, M. B., & Regier, D. A. (2002). A research agenda for DSM--V. *(2002).* Washington, DC, US: American Psychiatric Association.

Maser, J. D., Kaelber, C., & & Weise, R. E. (1991). International use and attitudes toward DSM-III and DSM-III-R: Growing consensus in psychiatric classification. *Journal of Abnormal Psychology, 100*(3), 271-279.

Mesulam, M. M., & Perry, J. (1972). The diagnosis of love-sickness: Experimental psychophysiology without the polygraph. *Psychophysiology, 9*(5), 546-551.

Millon, T. (1986). On the past and future of the DSM-III: Personal recollections and projections. In T. Millon, & G. L. Klerman (Eds.), *Contemporary directions in psychopathology: Toward the DSM-IV* (pp. 29-70). New York, NY, US: Guilford Press.

Neill, J. R. (1980). Adolf Meyer and American psychiatry today. *American Journal of Psychiatry, 137*(4), 460-464.

Prins, H. (1987). Literature review: Understanding and managing insanity: Some glimpses into historical fact and fiction. *British Journal of Social Work, 17*(1), 91-98.

Ray, I. (1871). *A treatise on the medical jurisprudence of insanity.* (5th ed.). Boston: Little, Brown, and Company.

Robins, E., & Guze, S. B. (1970). Establishment of diagnostic validity in psychiatric illness: Its application to schizophrenia. *American Journal of Psychiatry, 126*(7), 983-987.

Rogler, L. H. (1997). Making sense of historical changes in the diagnostic and statistical manual of mental disorders: Five propositions. *Journal of Health and Social Behavior, 38*(1), 9-20.

Sabshin, M. (1990). Turning points in twentieth-century american psychiatry. *American Journal of Psychiatry, 147*(10), 1267-1274.

Schuckit, M. A. (1994). DSM-IV: Was it worth all the fuss? *Alcohol & Alcoholism, 2*(Supplement. 2), 459-469.

Shoja, M. M., & Tubbs, R. S. (2007). The disorder of love in the canon of Avicenna (A.D. 980-1037). *American Journal of Psychiatry, 164*(2), 228-229.

Spitzer, R. L. (2001). Values and assumptions in the development of DSM-III and DSM-III-R: An insider's perspective and a belated response to Sadler, Hulgus, and Agich's "on values in recent American psychiatric classification". *Journal of Nervous & Mental Disease, 189*(6), 351-359.

Spitzer, R. L. (1982). "Feighner et al., invisible colleges, and the Matthew effect": Letter. *Schizophrenia Bulletin, 8*(4), 592.

Spitzer, R. L. (1991). An outsider-insider's views about revising the DSMs. *Journal of Abnormal Psychology, 100*(3), 294-296.

Vakili, N., & Gorji, A. (2006). Psychiatry and psychology in medieval persia. *Journal of Clinical Psychiatry, 67*(12), 1862-1869.

Weiner, D. B. (1992). Philippe Pinel's "memoir on madness" of December 11, 1794: A fundamental text of modern psychiatry. *American Journal of Psychiatry, 149*(6), 725-732.

WHO. (1948). *Manual of the international statistical classification of diseases, injuries, and causes of death.* (6th ed.). Geneva, Switzerland: World Health Organization.

Widiger, T. A., & Clark, L. A. (2000). Toward DSM--V and the classification of psychopathology. *Psychological Bulletin, 126*(6), 946-963.

Qualitative or Quantitative Differences between Asperger's Disorder and Autism?

Historical Considerations

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Qualitative or Quantitative Differences between Asperger's Disorder and Autism?

Historical Considerations

In 1943, Leo Kanner described a disorder similar to, but distinct from childhood schizophrenia. This disorder, true to its name, was referred to as autism, and included in the Diagnostic and Statistical Manual of Mental Disorders, 3rd Ed. (DSM-III). In 1981, Hans Asperger's account of Autistic Psychopathy (1944) was introduced to North America (Frith, 1991). This led to the apparent discovery of a new disorder similar to autism. Named after Asperger, it was included in the DSM-IV as a qualitatively distinct disorder from autism. Since its inclusion in DSM-IV, however, there has been a great deal of research examining whether Asperger's Disorder (AD) should be considered a disorder on an autistic spectrum, or whether its characteristics warrant it as related to but distinct from autism.

AD and autism are highly related, and are both considered Pervasive Developmental Disorders (PDDs). Determining whether a qualitative distinction exists between AD and autism specifically, and autism generally, has several clinical and research implications. If both disorders are distinct they may also differ in etiology, which may impact early identification and biological markers for the disorder(s), or prevention through physiological means. In addition, prognoses and interventions would likely differ between Asperger's and autism if they were qualitatively distinct (Macintosh & Dissanayake, 2004).

Prevalence rates of PDD suggest that the diagnostic distinction between autism and AD is hazy. According to Fombonne (2005), autism is believed to be

present in approximately 13/10,000 people, whereas AD is at about 2.6/10,000 people. PDD Not Otherwise Specified (PDD-NOS) involves patients who present with autistic symptoms but do not qualify for a diagnosis of a specific PDD (i.e. autism, AD, Rett's Disorder). Interestingly, the less-specific diagnosis of PDD-NOS accounted for about 21/10,000, nearly twice as much as those with autism, and about eight times higher than AD. In effect, PDD-NOS, which has no specific diagnostic criteria, outnumbers the combination of all other PDDs that do have specific diagnostic criteria. This suggests that the diagnostic criteria at present for PDDs are too specific or do not capture the primary features of the disability, and that significant functional deficits are still experienced by a higher proportion of individuals that do not meet specific criteria for AD and autism. Diagnostic criteria for AD and autism need to be re-evaluated, and by looking at the history of AD and autism its influences become clearer.

Leo Kanner

In his landmark paper in 1943, *Autistic Disturbances of Affective Contact*, Leo Kanner described 11 children who demonstrated obsessive and repetitive behaviours, social deficits, and echolalia. Although the children's behaviours were somewhat consistent with childhood schizophrenia, they seemed different from other recorded incidences of childhood schizophrenic patients. Children with schizophrenia demonstrated average development prior to the onset of their disorders, however, according to Kanner, his patients seemed to exhibit "extreme aloneness" from birth (p. 248) (although research at present has not identified autism in children at birth, factors in the first year of life, such as intense interest

toward objects, has been shown retrospectively in children with autism, i.e. Maestro et al., 2006). In addition, Kanner's patients seemed peculiar because they related obsessively with objects but avoided any affective contact with people. Relating with people was tolerated by the children by focusing on the person's hand or foot as a detached object, with social reciprocity apparently lacking entirely.

Kanner's descriptions of the 11 children he studied appear consistent with the present diagnosis of autism, and his paper was arguably influential on the development of the diagnostic criteria in the long run. Nine years after Kanner's seminal paper, the first edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM) was published (APA, 1952). The second edition was published in 1968 (APA). In these first two editions of the DSM, autism was not categorized as a disorder. However, elements of modern-day diagnostic criteria for autism were present in the criteria for schizophrenic disorders, most notably Schizoid Personality and Schizophrenia – Childhood Type. Under Schizoid Personality, these manuals establish "autistic thinking" (i.e. coldness, emotional detachment, aloofness) as criteria (p. 42., 1968; p. 35, 1952). DSM-II describes Schizophrenia – Childhood Type as a condition manifested by "autistic, atypical, and withdrawn behavior" (p. 35, 1968). Autistic-type thinking and behavior, as we know it today, were considered schizophrenic symptoms well before the onset of the DSM (Frith, 1991), which may explain the time lag between the first published clinical description of autism in 1943 and its inclusion in DSM-III in 1980.

Autistic disorder first appeared as a disorder distinct from schizophrenia in the DSM-III, under the name Infantile Autism (APA, 1980), which was later changed to Autism in 1987. The authors indicated that some camps still considered autism a schizophrenic disorder, and that infantile autism was the earliest form of schizophrenia. Research in family studies, however, suggested that autism and schizophrenia were distinct disorders. The DSM-III criteria for infantile autism included deficits in the development of language, atypical patterns of speech when present, lack of responsiveness to others, unusual attachments and interests, and resistance to change, with an onset before 30 months of age. These criteria are remarkably similar to the diagnostic criteria for autism in the DSM-IV, which are qualitative impairments in social interactions and communication, and restricted repetitive and stereotyped patterns of behavior, interests, and activities, with onset prior to age three (2000).

Kanner provided many clinical descriptions in his study that are congruent with DSM-III and DSM-IV criteria, even in terminology. The title of Kanner's publication, *Autistic Disturbances of Affective Contact*, provided the name autism for the disorder. The terms "autistic" and "autism" are derived from the Greek word "autos", meaning "self", and were used to describe some characteristics of schizophrenia (Frith, 1991). In the context of autism as a disorder, it is meant to convey an apparent disconnect from the social world outside of themselves. Kanner described some of his child patients, "like in a shell", "acting as if people weren't there", and "perfectly oblivious to everything about him" (1943, p. 242), which seem to describe the apparent self-absorption associated with the term

"autistic". Kanner also described features of communicative impairment; "As far as the communicative functions of speech are concerned, there is no fundamental difference between the eight speaking and the three mute children" (p. 243); and repetitive behavior such as echolalia; "language was deflected in a considerable measure to a self-sufficient, semantically and conversationally valueless or grossly distorted memory exercise…When sentences are finally formed, they are for a long time mostly parrot-like repetitions of heard word combinations" (p. 243). The DSM-IV contains 4 specific symptoms in each of the diagnostic categories of impairments in social interaction, communication, and stereotyped patterns of behavior, totaling 12 symptoms. A careful reading of Kanner's article will uncover reference to each of those 12 symptoms in his clinical descriptions. Kanner also noted in his cases that the children's development were stable, not regressive like in childhood onset schizophrenia. This is reflected in the diagnostic criteria of onset of symptoms prior to 30 months in DSM-III, or before three years in DSM-IV. The only diagnostic criteria for autism that cannot be related to Kanner's article involve differential diagnosis, in that the symptoms are not better accounted for by Rett's Disorder or Childhood Disintegrative Disorder, because those disorders were not yet recognized at the time of Kanner's article.

Although Kanner describes at length the deficits associated with "autistic disturbances", he does point out some apparent and potential strengths of the children. He noted the children displayed good cognitive potential and strong episodic memory, and that those who spoke demonstrated an excellent vocabulary. The relative strengths that these children demonstrated in memory

and vocabulary (or in DSM-type language, the lack of deficits in these areas) were not incorporated into the DSM-III criteria for autism.

Hans Asperger

In 1944, Hans Asperger published his account, *Autistic Psychopathy in Childhood*, which is remarkably similar to Kanner's publication a year earlier (Frith, 1991). In it, Asperger describes in his children the same deficits of social interaction and stereotyped behavior that Kanner described. In Frith's translation of Asperger's article (1991), it is noted that "relations with the outside world are extremely limited" (p. 42), that a patient "could not engage in the lively reciprocity of normal social interaction" (p. 45), and that the children had "abnormal fixations" with objects (p. 81). Like Kanner, Asperger also noted that the autistic differed from the schizophrenic in that cognitive and social development was not regressive but stable in the former. Qualitative impairments in communication, unlike Kanner's findings, are not apparent in Asperger's work. In fact, Asperger reported some extraordinary abilities in mathematics and relative strengths in some communicative skills, noting that some children spoke like adults, though in a rigid stereotyped fashion.

Although Asperger's description of autistic disorder may have been recognized in Germany, his work was not introduced to North America until 1981, by Wing. This came 37 years after his article was first published, and one year after the publication of the DSM-III. Despite a strong interest in the disorder in North America, Asperger's original article was not translated into English until ten years after it was introduced (Frith, 1991). In this respect, it is astounding to

think that Asperger's and Kanner's articles, though nearly identical in description, were not directly compared until nearly 50 years after their publication dates. In fact, Asperger published one year after Kanner but may have been engaged in the investigation of autism well before him (Lyons & Fitzgerald 2007). Their accounts of their patients were virtually parallel in both description and timing, yet culminated in two distinct, though similar disorders in the DSM-IV. This may be in part related to the fact that they were introduced to the English-speaking world decades apart.

The relative focus on patients' verbal strengths in Asperger's report seems to have had a direct impact on the DSM-IV criteria for Asperger's Disorder (AD). The diagnostic criteria for AD differs most notably from autism in that communication delay is not present. Interestingly, similar verbal strengths were identified by Kanner in some, but not all of his cases (1943). Yet this distinction was not made in the DSM-III (before Asperger's work reached North America), probably due to the orientation of the DSM to focus on deficits rather than strengths. When Asperger's cases were introduced to North America, they may have been perceived as novel because they were recognized much later than Kanner's work, and the orientation of Asperger's article focused more so on children's strengths. In retrospect, it may be that a new disorder or a variant of autism was not introduced to North America as was thought at the time. Rather, the autism Hans Asperger discussed may have been the same autism that Kanner referred to. Wing, who first introduced Asperger's work to North America,

indicated that there was no evidence that any qualitative differences existed between the two disorders, but this assertion was largely ignored (2000).

The apparent, though restricted verbal strengths described in both Kanner's and Asperger's accounts seem consistent with High Functioning Autism (HFA) and/or Asperger's Disorder (according to DSM), which at present is a topic of diagnostic controversy and the central theme of this paper. In DSM-IV, AD was distinguished from autism, but apart from the absences of communicative impairment and cognitive delay, Asperger's Disorder is virtually identical to autism diagnostically. HFA does not appear in the DSM as a diagnosis per se, but is considered to be autism absent of cognitive delay, or in other words, an IQ above 70 (Ghaziuddin & Mountain-Kimchi, 2004). The distinction, if any, between HFA and AD is blurry.

AD/HFA Research

Since the introduction of AD to North America in 1981, there has been substantial research (and resulting controversy) regarding differences, if any, between AD and HFA. Early research in this area suggested that children with AD exhibit greater verbal ability than those with HFA (e.g. Ozonoff, Rogers, & Pennington, 1991). There were, however, some problems with much of the research. Many studies that found qualitative differences between AD and HFA were conducted before formalized diagnostic procedures were created for AD (i.e. DSM-IV). When studies were replicated using DSM-IV criteria, the differences were often no longer present. Even when using DSM-IV criteria in comparison studies, finding differences between AD and HFA groups did not necessarily

mean that the disorders were distinct (Macintosh & Dissanayake, 2004). For example, DSM-IV distinguishes HFA from AD as having a presence of language delay. Therefore, if a researcher partitions participants into HFA and AD groups based on DSM-IV, the supposed HFA group will necessarily present with language delay relative to the supposed AD group. Finding group differences in verbal ability between the groups should not be surprising, nor does it present evidence of a distinction between HFA and AD, because that would be the basis of how they were partitioned. In other research, AD and HFA participants were not matched for IQ, and patients with higher IQ tended to receive a diagnosis of AD versus HFA. In addition, the majority of studies evaluated participants in childhood or early adolescence; few studies evaluated late adolescence and adulthood, and those that did tended to find no differences between AD and HFA groups (Howlin, 2003).

Taking the DSM-IV criteria of Asperger's at face value, a delay in language or cognitive functioning (IQ<70) should not be present in an AD diagnosis. In HFA, cognitive delay should also be absent (Ghaziuddin & Mountain-Kimchi, 2004), but a general delay in language should be present. Therefore, matching for cognitive functioning, patients with AD should perform significantly better in language functioning than those with autism, *and* on par with controls. However, most recent research in this area indicates that this appears to not be the case. Patients with AD are often on par with HFA groups in both language and cognitive ability, but significantly lower than controls in

language, especially in studies that use older children, adolescents, and adults as the sample (Howlin, 2003).

Differences in cognitive capacity between AD and autism may not be indicative of qualitative distinctions between the disorders. Mayes and Calhoun (2004) compared children with AD and autism on IQ and other variables, such as frequency of autistic symptoms and social concerns. Although they found that lower IQ was related to more autistic symptoms and social problems, these effects were nullified when IQ and age were statistically removed. This suggests that autistic symptoms and social problems were attributable to IQ and age, rather than differences intrinsic to autism. This result seems consistent with research suggesting that differences between AD and HFA diminish as children get older (Howlin, 2003).

Although full-scale IQs of children with AD and HFA are similar in many studies, their intellectual profiles may differ to some degree. Ghaziuddin and Mountain-Kimchi (2004) compared the intellectual profiles of AD and HFA patients using the Wechsler scales. They found that patients with AD tended to have a higher Verbal IQ than Performance IQ, and that their VIQ were usually higher than the VIQ of patients with HFA. The IQ profiles of HFA patients were varied, with an equal number showing relative strengths in VIQ and PIQ. In addition, the patients with AD performed significantly better on Arithmetic, Information, and Vocabulary subtests than the HFA patients. These results suggest that patients diagnosed with AD are likely to perform better on verbal tasks than performance tasks, and that their verbal abilities tend to be stronger

than the verbal abilities of HFA patients. The authors note, however, that both AD and HFA groups had individuals whose profiles were typical of the other group. This suggests that although verbal ability may be greater in AD as a whole, that the diagnostic utility of VIQ and PIQ for differentiating the disorders is questionable.

Another problematic area in distinguishing AD from HFA is the diagnostic criteria for Asperger's of a lack of general delay in language, because language delay is not clearly nor objectively defined. In addition, many people diagnosed with AD present with language delay later in life; conversely, many with autism do not (Macintosh & Dissanayake, 2004). Bennett et al. (2008) evaluated the use of structural language impairment for discriminating between populations on the autism spectrum. Structural language impairment refers to deficits in grammar or syntax, not semantic or pragmatic uses of language. This is more specific and measurable than general language delay noted in DSM-IV. Participants were tested for language skills at ages 4 to 6 and again two years later. Subsequently, children's autistic symptoms and adaptive functioning were assessed once every two years until ages 15 to 17. In this way, the researchers were able to evaluate the long-term outcomes of the participants in relation to their language skills early in life. The researchers found that structural language impairment at ages 6 to 8 was more predictive of autistic symptoms and adaptive dysfunction in adolescence than was a diagnosis of AD or autism based on DSM-IV criteria. This result suggests that impaired use of syntax and grammar, at ages 6 to 8, is a better predictor of autistic tendencies than general delay in language by

age 3, as reported retrospectively by parents. In addition, structural language impairment at ages 6 to 8 was more predictive of autistic behaviour and adaptive functioning than the same impairments at ages 4 to 6, suggesting a "catch-up" effect as children reach primary school age. This catch-up may make an analysis of language delay prior to age three meaningless (Woodbury-Smith, Klin, & Volkmar, 2005).

Howlin (2003) obtained similar findings in her study, where adults with PDD were separated into an autism group (early language delay present), and an AD group (early language delays absent), and were matched for age, nonverbal IQ, and gender. Howlin found that there were no significant differences on the Autism Diagnostic Inventory – Revised (ADI-R) between the autism and AD groups, suggesting no differences in autistic symptomology based on the presence or absence of early language delay. This result indicates that early language development may not be a good indicator of autistic symptoms later in life, and that differences between autism and AD are ambiguous.

Another discriminating factor between AD and autism in DSM-IV is onset of language delay. A child will be given an AD diagnosis only if there is no language delay *prior* to a certain age. For example, a child who used single words by age two years will meet onset criteria for AD. However, a diagnosis of autism takes precedence where there is a conflict of criteria, such as if that child used single words by age two years, but did not use communicative phrases by age three years. This approach is problematic in that diagnoses are slanted toward autism on the basis of development. In addition, information regarding a child's

language development is usually obtained retrospectively, which presents with a number of issues. Dates of developmental milestones may not be remembered, minor developmental delays may be inflated, and present diagnostic realities may distort parents' memories of their children's development (Woodbury-Smith, Klin, & Volkmar, 2005).

The supposition that children with AD would be free of language delay is somewhat mysterious, as Asperger described language and communication dysfunction amongst his participants (Frith, 1991). This apparent absence of language delay in early years may be tied to the supposed absence of qualitative impairments in communication, which also distinguishes AD from autism in DSM-IV. However, research has been unequivocal in demonstrating deficits in social communication for AD (Howlin, 2003; Woodbury-Smith, Klin, & Volkmar, 2005), and Bennett et al.'s findings (2008) suggest that communication impairment is in fact present for AD. With most recent research suggesting homogeneity of language and communication dysfunction between AD and HFA, qualitative distinctions between the disorders virtually disappear.

A DSM-IV diagnosis for either AD or autism requires the presence of qualitative impairments in social interaction. However, because several diagnostic criteria for AD in the DSM-IV have come into question, research comparing AD with HFA in social skills and interaction is needed to determine whether there are differences in these areas. Because communication impairment in AD is supposed to be absent, most researchers comparing social skills hypothesize that children with AD will demonstrate better social functioning.

Barbaro and Dissanayake (2007) compared children with AD and HFA on self-presentational display rules, where they were evaluated for their ability to regulate their outer expressions of emotion. AD and HFA groups did not differ in their uses of self-presentational display rules, which were less effective than those used by typically developing children. This result indicates that children with AD and HFA seem to be on par in the regulation of outward emotional expression.

Social skills deficits can sometimes lead to behavioural challenges. Macintosh and Dissanayake (2006a) compared children with AD and HFA in levels of cooperation, assertion, responsibility, and self-control. They found no differences between the AD and HFA groups, but found that both groups showed deficits compared with typically developing children. They also noted that both AD and HFA groups were at increased risk of co-morbid disorders, such as depression.

Some studies, however, have found differences in social skills between children with AD and HFA. Ghaziuddin (2008) classified characteristics of social interaction of children, with IQs above 70, into three categories: a) aloof participants, who were indifferent toward most social situations and other children; b) passive participants, who responded to questions appropriately but did not initiate social contact; and c) active but odd participants, who spontaneously initiated social interaction but did so in an inappropriate manner. Ghaziuddin found that the majority of children diagnosed with autism were described as aloof and passive, and that the majority of children with AD were described as active but odd. This result, while not necessarily demonstrating differences in social

skill, do suggest differing social characteristics between AD and autism. Macintosh and Dissanayake (2006b) also found differences in characteristics of social interaction between AD and HFA. Observing children's social interactions in everyday settings, the researchers found that children with AD and HFA demonstrated similar social behaviours in spontaneous peer interactions, social competence, and time spent interacting, which were all below that of typically developing children. However, the authors did find that children with AD demonstrated more social bids and more conversation during social interaction, suggesting increased social motivation and increased expressive language. Taken together, the findings of Ghaziuddin and Macintosh and Dissanayake suggest differences in social preferences or perhaps personality, not social skill. For example, extraverted children with autistic symptoms may be more likely to be diagnosed with AD because communication is more apparent, though not necessarily impaired. Conversely, introverted children of similar IQ may be more apt to receive a diagnosis of autism, because the presence of communication and language skill is less apparent.

Discussion

Up until now, there seems to be no consistent evidence of any significant differences in symptomology, or social, emotional, or psychiatric problems between AD and HFA. Frith (2004) asserts that Asperger Disorder appears to be a variant of autism typically diagnosed in patients that are verbally and/or cognitively higher-functioning, and is not a separate disorder. Most recent empirical research supports this claim.

The recognition of HFA calls into question the validity of DSM-IV criterion of absence of cognitive delay for AD. If AD is a distinct disorder from autism, there is little use for this cognitive criterion because many patients with autism are not cognitively delayed (based on an IQ above 70).

The AD diagnostic criterion in DSM-IV regarding absence of language delay can be problematic for a number of reasons. First, language delay is neither specific nor well-defined. Examples of language delay absence in the text include "single words used by age 2 years, communicative phrases used by age 3 years" (p. 84). But those abilities do not necessarily preclude the presence of a language delay. For example, an echolalic child may use single words by age 2 but with no communicative meaning or intent. Second, the presence of language delay in the first 3 years of life does not necessarily translate into lifelong language impairment, nor does the apparent absence of language delay in toddlers necessarily lead to lifelong verbal ability within the average or functional range. Differences in verbal ability between AD and HFA have disappeared as early as primary school age in some studies (Howlin, 2003). Third, because recognition of language delay is age-sensitive (i.e. by age 2 years) the burden is in part left on the parent to determine whether the child demonstrated a language delay. If, for example, a child vocalizes advanced terminology relative to age, the parent may perceive relative verbal strength, even if that child speaks the term inappropriately or as a stereotyped pattern of behaviour. Fourth, because language delay is age-sensitive, clinicians must rely on retrospective reports of language development, which may not accurately represent true language functioning. As it stands in

DSM-IV, the presence or absence of "language delay" seems to be the primary (if not only) discriminating variable between AD and HFA. Language delay, however, lacks definitive meaning and is subject to invalid retrospective reporting, and is likely not well-fit as a discriminating variable between disorders (Macintosh & Dissanayake, 2004).

What may be difficult to conceptualize is that verbal deficit is considered a primary characteristic of autism, and when an apparently autistic child shows relatively enhanced verbal ability we might feel the need to re-classify that child. In addition, verbal ability seems highly relatable to cognitive functioning (e.g., a person with higher VIQ may give a first impression of precociousness compared to a person with higher PIQ). Therefore, when a child appears precocious at first glance, there may be an assumption that the child has strong verbal ability (therefore, not autistic), which may not be the case. A child can "speak like an adult" (i.e. children in Kanner's and Asperger's studies), but still show deficits in verbal ability, such as the use of grammatical rules and syntax (Bennett et al., 2008). This long-standing impression of verbal deficits in autism may have skewed our impressions of the actual disorder. Language impairment, for example, may be a secondary characteristic of social abstinence. Further research is needed in this area to better understand the fundamental aspects of autism, and to determine whether our current framework best fits the actual disorder.

It should not be surprising to observe greater cognitive ability and verbal acumen among some people with autism compared with others with autism; such differences in verbal ability exist within typically developing populations.

Bennett et al. (2008) seemed to recognize that variations in development of patients with autism do not mean that a different diagnostic category is needed. These researchers suggest that "the best way to think of these conditions is not as different disorders but as parallel and potentially overlapping developmental pathways" (p. 618). Therefore, it may be more valid to think about patients with Asperger's as patients with autism that are precocious and/or verbally gifted relative to other patients with autism.

Apparent strengths in verbal ability may be designated to children with AD, relative to HFA, because they are more apt to be heard. Similar deficits in communication have been demonstrated for children with AD and HFA (Bennett et al., 2008), as well as deficits in social skills (Barbaro & Dissanayake 2007; Macintosh & Dissanayake, 2006a). However, social motivation (Macintosh & Dissanayake, 2006a) and a propensity to speak (Ghaziuddin, 2008) have been shown to be greater in AD. This may not represent a distinct diagnostic difference, rather differences in personality. Children more likely to be diagnosed with AD rather than autism may be more extraverted than other autistic children. Differences in extraversion/introversion have been well-established in personality theory and in typically developing populations; it should not be surprising that some children with HFA would be more extraverted than others.

Based on up-to-date empirical research, it appears that AD and autism are not qualitatively distinct disorders, but are different quantitative manifestations of the same disorder. This suggests that AD is part of the autism spectrum, and is not qualitatively distinct. The differences in cognitive, language, and social

ability between AD and autism may be a function of individual variability in these areas, not the presence of unique disorders. Since individual variability is apparent in typically developing populations, such variability could be found within the autistic population as well. A child with autism may be with relatively high verbal ability and cognitive functioning, and/or may present with an extraverted personality. Because the DSM-IV criteria at present distinguish AD as a lack of language delay and cognitive functioning, such a child may be more likely to be diagnosed with AD. Other children with autism may present with lower cognitive and language functioning, and receive a diagnosis of autistic disorder. Children with higher relative cognitive functioning but an introverted personality may be deemed as HFA. More research is needed to understand whether individual variation accounts for the apparent differences in children and adults with AD and autism, and whether AD belongs on the autistic spectrum.

Bibliography

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., TR). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders*. Washington, DC: American Psychiatric Association.

Barbaro, J., & Dissanayake, C. (2007). A comparative study of the use and understanding of self-presentational display rules in children with high functioning autism and Asperger's disorder. *Journal of Autism & Developmental Disorders, 37*(7), 1235-1246.

Bennett, T., Szatmari, P., Bryson, S., Volden, J., Zwaigenbaum, L., Vaccarella, L., et al. (2008). Differentiating autism and Asperger syndrome on the basis of language delay or impairment. *Journal of Autism and Developmental Disorders, 38*(4), 616-625.

Fombonne, E. (2005). The changing epidemiology of autism. *Journal of Applied Research in Intellectual Disabilities, 18*(4), 281-294.

Frith, U. (2004). Emanuel Miller lecture: Confusions and controversies about Asperger syndrome. *Journal of Child Psychology and Psychiatry, 45*(4), 672-686.

Frith, U. (1991). Autism and Asperger syndrome. Cambridge: Cambridge University Press

Ghaziuddin, M. (2008). Defining the behavioral phenotype of Asperger syndrome. *Journal of Autism and Developmental Disorders, 38*(1), 138-142.

Ghaziuddin, M., & MountainKimchi, K. (2004). Defining the intellectual profile of Asperger syndrome: Comparison with high-functioning autism. *Journal of Autism and Developmental Disorders, 34*(3), 279-284.

Howlin, P. (2003). Outcome in high-functioning adults with autism with and without early language delays: Implications for the differentiation between autism and Asperger syndrome. *Journal of Autism & Developmental Disorders, 33*(1), 3.

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child,* (2), 217-250.

Lyons, V., & Fitzgerald, M. (2007). Asperger (1906-1980) and Kanner (1894-1981), the two pioneers of autism. *Journal of Autism and Developmental Disorders, 37*(10), 2022-2023.

Macintosh, K., & Dissanayake, C. (2006). A comparative study of the spontaneous social interactions of children with high-functioning autism and children with Asperger's disorder. *Autism, 10*(2), 199-220.

Macintosh, K., & Dissanayake, C. (2006). Social skills and problem behaviours in school aged children with high-functioning autism and Asperger's disorder. *Journal of Autism & Developmental Disorders, 36*(8), 1065-1076.

Macintosh, K. E., & Dissanayake, C. (2004). The similarities and differences between autistic disorder and Asperger's disorder: A review of the empirical evidence. *Journal of Child Psychology & Psychiatry & Allied Disciplines, 45*(3), 421-434.

Maestro, S., Muratori, F., Cesari, A., Pecini, C., Apicella, F., & Stern, D. (2006). A view to regressive autism through home movies: Is early development really normal? *Acta Psychiatrica Scandinavica, 113*(1), 68-72.

Mayes, S. D., & Calhoun, S. L. (2004). Influence of IQ and age in childhood autism: Lack of support for DSM-IV Asperger's disorder. *Journal of Developmental and Physical Disabilities, 16*(3), 257-272.

Ozonoff, S., Rogers, S. J., & Pennington, B. F. (1991). Asperger's syndrome: Evidence of an empirical distinction from high-functioning autism. *Journal of Child Psychology and Psychiatry, 32*(7), 1107-1122.

Wing, L. (2000). Past and Future of Research on Asperger Syndrome. In A. Klin, F. K. Volkmar, & S. S. Sparrow (Eds.), *Asperger syndrome* (pp. 418–432). New York: The Guildford Press.

Wing, L. (1981). Asperger's syndrome: A clinical account. *Psychological Medicine,* 11, 115-13

Woodbury-Smith, M., Klin, A., & Volkmar, F. (2005). Asperger's syndrome: A comparison of clinical diagnoses and those made according to the ICD-10 and DSM-IV. *Journal of Autism and Developmental Disorders, 35*(2), 235-240.

Were our Forebears Aware of Prenatal Alcohol Exposure and its Effects? A Review of the History of Fetal Alcohol Spectrum Disorder

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Were our Forebears Aware of Prenatal Alcohol Exposure and its Effects? A Review of the History of Fetal Alcohol Spectrum Disorder

The discovery of the effects of prenatal alcohol exposure on the developing fetus is relatively new. In an unpublished thesis in Paris, 1957, Rouquette described malformations in children born to alcoholic mothers and fathers, and noted that maternal alcoholism could be a serious threat to the fetus (Lemoine, 1994; Streissguth, 1997). Eleven years later, Paul Lemoine, also in France, published a clinical description of 127 children born to predominantly alcoholic mothers (Lemoine et al., 1968). He noted physical malformations, developmental delays, and behavioural problems in the "Lemoine kids" (Lemoine, 2003). In a series of studies in Seattle, Washington five years later, teratologists Kenneth Jones and David Smith described similar characteristics in a handful of children (Jones et al., 1973; Jones et al., 1974), and coined the term Fetal Alcohol Syndrome (FAS) (Jones & Smith, 1973). These seminal articles brought worldwide attention to the disorder and initiated a new branch of research and clinical practice into the impacts of alcohol on the developing fetus (Streissguth, 1997). The research literature has since evolved to capture a broader view of the effects of prenatal alcohol exposure beyond FAS. The term fetal alcohol spectrum disorders (FASD) is now used to describe a wide range of deficits associated with prenatal alcohol exposure (Koren et al., 2003).

In addition to work exploring the nature of FASD, research connecting FASD to historical records and events emerged. Jones and Smith, in their second article about the newly discovered disorder, provided a brief look at some

historical accounts that apparently alluded to the effects of alcohol consumption during pregnancy (Jones & Smith, 1973). In 1975, only two years after FAS was given its name, Warner and Rosett conducted a comprehensive review of British and American literature that seemed connected to this newly identified disorder. They referred to the findings of Jones and Smith as a rediscovery of the effects of alcohol on the developing fetus, and suggested that the effects of maternal drinking have been suspected for centuries.

History of FASD

According to Davis (1980), a biblical passage may be the first reference to the effects of prenatal alcohol exposure. In the Old Testament it is recorded that an angel spoke to the mother of Samson and said, "Thou shalt conceive, and bear a son. Now therefore, beware, I pray thee, and drink not wine nor strong drink" (Judges 13:3-4). At first glance, this passage appears to be an exhortation to avoid alcohol so that the unborn son may not be exposed to its impacts. For this reason, it has been cited in numerous papers as an indication that there was at least a rudimentary knowledge of the effects of prenatal alcohol in biblical times (Calhoun & Warren, 2007). However, Abel (1997) noted that in context, the passage may not be referring to the deleterious effects of alcohol on the development of the fetus. A broader look at the same passages in the 13th chapter of Judges provides some important context:

> "Thou shalt conceive, and bear a son. Now therefore beware, I pray thee, and drink not wine nor strong drink, and eat not any unclean thing: For, lo, thou shalt conceive, and bear a son; *and no razor shall come on his head*:

> for *the child shall be a Nazarite unto God from the womb*." (Judges 13:3-5, italics added)

According to another biblical scripture, "When either man or woman shall separate themselves to vow a vow of a Nazarite, to separate themselves unto the Lord: *He shall separate himself from wine and strong drink*" (Numbers 6:2-3). In addition, for a Nazarite "there shall no razor come upon his head" (Numbers 6:5). This passage seems to correspond with the warning given to Samson's mother, quoted above. Therefore, the exhortation to not consume strong drink was more likely based on the consecration of Samson as a Nazarite "from the womb" than a reference toward the effects of alcohol on the developing fetus.

Aristotle is often quoted as being aware of the effects of prenatal alcohol exposure: "foolish, drunken, or haire-brain women most often bring forth children like unto themselves, morose and languid" (as cited in Warner & Rosett, 1975). But there are a couple problems with that assumption. First, this statement seems more indicative of an awareness of the tendency for children to turn out like their parents, rather than of the effects of prenatal alcohol exposure. The term "drunken" is used as a descriptor or characteristic of certain women, along with "foolish" and "haire-brain" (i.e. what they are, not what they do). According to this statement, these women are likely to have children that are also "foolish, drunken, or haire-brain." It does not imply a direct impact of alcohol exposure (drunkenness) on children, but indicates that children are likely to turn out like their parents. Whether through genetic transmission or environmental influence, it is not specified. The second, and more significant, problem with this statement

is that it may not have been spoken by Aristotle at all. In his 1621 publication *Anatomy of Melancholy*, Robert Burton cited Aristotle's *Problemata* and attributed this statement to him (Warner & Rosett, 1975). However, Abel (1999a) noted that this statement does not appear in *Problemata*, nor does it appear in Aristotle's other writings. He also explained that similar quotes mentioned by Burton were not attributable to the figures he cited (i.e. Aulus Gellius and Plutarch). Abel asserts that the Aristotelian quote shared by Burton was Burton's synopsis of what the Greek philosopher would have said about the effects of alcohol, not an actual quote of Aristotle.

Although Aristotle did not appear to address the effects of prenatal alcohol exposure on fetal development, he did discuss its effects on fertility and gender. Based on the premise that all things were composed of fire and water, Aristotle asserted that men were made of a greater proportion of fire than women, and conversely, women were made of more water than men (Abel, 1999b). Men were considered more intelligent, courageous, strong, healthy, and morally superior because they had more natural heat. Aristotle believed that if the man had consumed too much alcohol that he became cold (i.e. shivers). During coitus, he would be unable to heat the woman's womb, and as a result would conceive a girl.

Abel (1999a) pointed out fundamental differences between views about alcohol and fertility in antiquity and our present understanding of the teratogenic effects of alcohol. He noted that the focus of ancient Greeks and Romans was on paternal drinking, whereas ours is on maternal drinking. This may have been the case in antiquity because of the ideological superiority of males and a limited

understanding of human reproduction. In addition, the Greeks and Romans believed conception to be the period where alcohol had its active effect, whereas we focus on drinking throughout pregnancy. In antiquity, although too much alcohol was thought to be detrimental, moderate drinking was encouraged, whereas presently no amount of alcohol is considered safe for pregnant women. Although there are apparent fundamental flaws in these views in antiquity, they did recognize a relationship of some kind between alcohol consumption and reproduction.

From antiquity until the 18th century, there appears to be a lag in recorded information relating alcohol to human reproduction, though thinking regarding this topic may have been present. In England from 1720 to 1750, there was ample opportunity to observe the effects of alcohol on reproduction. The country lifted restrictions on distilling, and cheap gin flooded the country. England became mired by a "gin epidemic" (Warner & Rosett, 1975). The resulting social problems led to a movement calling for gin restrictions. For example, Sedgewick stated in 1725 that "half the train of chronological Diseases" afflicting infants were caused "by the Debauchery of the Mother...So that from the whole, the Regulation of the Mother, during her Pregnancy, is an Affair of the highest Moment and Consideration". A year later, College of Physicians called gin "a cause of weak, feeble, and distempered children". These quotes suggest that the mother's conduct generally, and gin specifically, negatively affected children's health. In addition, increased stillbirth and infant mortality were noticed at that time.

William Hogarth's painting *Gin Lane* reflects many of the social concerns related to the gin epidemic, and interest in the painting has resurged since the advent of historical research in FASD. The painting depicts a myriad of social problems, including drunkenness, misery, poverty, starvation, abuse, suicide, and infanticide. The central figure is a syphilitic woman who unknowingly drops her infant as she reaches for snuff. Apart from the obvious social ills described in the painting, some researchers suggest that it is a testament that an awareness of the effects of prenatal alcohol existed at that time (Shibley & Pennington, 1998). For example, Rodin (1981) suggested that the infant falling from the woman's arms showed abnormal palpebral features, characteristic of FAS. Additional facial features of FAS, however, were not identified for the child, and similar features were not described in the other figures in the painting (Abel, 2001).

Another painting by Hogarth, *Beer Street*, indicates that an awareness of the effects of prenatal alcohol exposure was not apparent, contrary to the claims of many researchers who hold *Gin Lane* as an important symbol of the history of FASD in 18th century England (Abel, 2001). Themes of *Beer Street* include commerce, wealth, industry, and happiness, and are in sharp contrast with the significant social ills demonstrated in *Gin Lane*. This suggests that Hogarth was commenting on the deleterious effects of prenatal alcohol exposure, but was commenting on the effects of the "vile" gin, contrasting with the "invigorating" beer.

Research of the impacts of alcohol and pregnancy began to emerge in North America in the 19th century. Benjamin Rush, who lobbied against alcohol

use during pregnancy to avoid dependence, was influential as the temperance movement in the United States took shape. In England, Thomas Trotter proposed that alcohol could cause mental deficiency in children, an innovative idea that was not researched until decades after his time (Warner & Rosett, 1975). These two figures helped fuel the temperance debate.

The initiation of the temperance movement in the mid-19th century was highly driven by moral and religious attitudes, and resulted in several groups and societies debating the subject, such as the American Temperance Society in 1826. As the temperance movement took shape, an interest in studying the effects of alcohol on human development emerged in scientific literature in Europe and North America, with some studies evaluating the effects of prenatal alcohol exposure. A report for the House of Commons by a committee on drunkenness indicated that children of alcoholic mothers have "a starved, shrivelled, and imperfect look" (Warner & Rosett, 1975). Journals were formed specific to the temperance movement (i.e. *Journal of Inebriety*), and temperance began to be an accepted moral standard for many Americans. In the early 20th century, experimental studies of the effects of prenatal alcohol exposure in animals emerged, showing delayed growth, physical malformations, and high mortality, which indicate similar results in modern research.

A seminal study at the time of medical temperance was provided by Sullivan (1899). In his research, Sullivan compared 600 children of alcoholic mothers with controls and found higher rates of stillbirths and mortality among them. In addition, Sullivan found that alcohol consumption of fathers and

grandparents were not correlated with these effects. When alcoholic mothers who drank in previous pregnancies and had damaged offspring abstained under coercion (such as by imprisonment), their children did not experience the same rates of mortality. These results suggest that maternal alcohol consumption, not paternal, is a cause of stillbirth and infant mortality.

The eugenics movement began to take shape at the turn of the 20th century, about the same time that the medical temperance research was at its peak. Proponents of the eugenics movement suggested that "imbecility" was genetic and that restricting or eliminating the reproductive practices of "idiots", "imbeciles", and "morons" (three degrees of intellectual disability) would lead to better care and less strain on resources, or in some cases, a purer race. Many eugenists argued that alcohol was a "race poison" that could contribute to the downfall of humanity. However, some Darwinists, such as Archdall Reid, asserted that alcohol was "the cause of an evolution protective against itself" (Reid, 1903, p. 819), in that the effects of alcohol use through natural selection would offset the hereditary propensity for alcohol as a whole.

Goddard's study of the Kallikak family was central to the eugenics movement, as he recommended that the "feeble-minded" have their reproductive organs removed to prevent the procreation of feeble-minded children (Goddard, 1912). This, of course, was based on the premise that those like the Kallikak family would pass on feeble-mindedness to their children genetically. This movement gained momentum, and was still practiced into the mid-20th century, well after the medical temperance movement ended. Interestingly, after

retrospective analysis of Goddard's data, some researchers suggest that members of the Kallikak family suffered the effects of prenatal alcohol exposure (Karp et al., 1995), but those assertions are speculative. Still, it is somewhat unsettling to think that the Kallikak study, which fuelled the now defunct eugenics movement, may have to do with prenatal alcohol exposure, not strictly genetics, and that an alternate method of preventing "feeblemindedness" may have been to restrict alcohol use rather than ovariectomy and castration.

The research on alcohol and pregnancy of this area resulted in significant debates leading to, in many cases, personal attacks. Malcolm (1984) summarized a heated debate that occurred in 1903 between researchers opposed to the temperance movement (including Archdall Reid, quoted above) and those promoting abstinence from alcohol. C. Mercier, for example, admitted that in comparison with Reid he and his colleagues, "are but worms, but why do they address their opponents in the tone of Almighty God addressing a peculiarly ignorant and recalcitrant black beetle?" (as cited in Malcolm, 1984, 261).

Much of this research was encouraged and supported by groups and societies promoting abstinence, and became a moral, religious, and political quest, rather than a scientific quest for knowledge. Research was largely conducted to support the platform of these organizations. The Anti-Saloon League, for example, created periodicals, an encyclopedia, numerous flyers, and other publications to combat the use of alcohol in North America. The research they used was arguably biased and sensational. For example, one flyer uses statistics to demonstrate that children of alcoholic parents have higher mortality, without

accounting for other variables that might contribute to increased death rate, such as poverty (Anti-Saloon League, 1909). In addition, much of the imagery in these flyers can illicit an emotional reaction, such as children pictured next to tombstones.

The moral and religious undertones that fuelled research into the effects of prenatal alcohol exposure may have also contributed to its temporary downfall. When Prohibition came into effect, alcohol was largely considered a moot issue, resulting in a significant decrease in research of the effects of prenatal alcohol exposure. Later, scientists discounted research in alcoholism as moralistic and unscientific (Crowe, 1985). In addition, sociological explanations for the deficits experienced by children of alcoholic parents began to be forwarded, such as poverty and abuse (Elderton & Pearson, 1910). About 50 years after Prohibition and the near shut-down of studies into the effects of prenatal alcohol exposure, research again exploded when Jones & Smith provided a clinical description of the fetal alcohol syndrome (1973). Based on evidence to date, it is anticipated that this research topic should not fall out of favour again.

Discussion

According to this literature search, there is apparently only one comprehensive study of the history of FASD, by Warner and Rosett (1975), which occurred over 30 years ago. Another limitation of historical research in FASD is that the majority of rigorous articles on the topic were authored by only one researcher, Earnest Abel. Shibley and Pennington are excepted, as their review of the history of FASD emphasizes historical reliability, citing objectively,

and a return to original sources where possible (1998). Other exceptions may exist but were not found in literature searches. More sound, critical research and writing is needed regarding the history of FASD because many of the same erroneous assumptions are being made again, which tend to be an over-emphasis of the validity of historical data.

Although historical records and research in the late-19th and early-20th centuries discuss the effects of alcohol on fertility, it does not seem likely that these events directly influenced the discovery of fetal alcohol syndrome. Initial clinical descriptions of this condition (Jones et al., 1973; Lemoine et al., 1968) were conducted when these authors noticed differences within a population of children born to alcoholic mothers. It was exploratory, not confirmatory research, and references to previous studies and historical accounts were made after the fact. However, broader areas of alcoholism and teratogenics seem to be influential; Jones and Smith, for example, were teratogenists.

Clinical descriptions by Jones et al. (1973) and Lemoine et al. (1968) mirror in many ways conclusions found in alcohol and pregnancy research in the early 19th century. Stillbirths and infant mortality were identified frequently in early research. Growth delays were recognized, and physical and central nervous malformations were identified in animal studies. Children of alcoholic parents were identified as being cognitively impaired by Trotter and later researchers. Behavioural and moral "degeneracy" were thought to be associated with these children (Warner & Rosett, 1975). Even a propensity for addiction was identified, which has been shown to be a significant and common problem of children with

FASD (Streissguth et al, 2004). One significant attribute was identified by Jones et al. and Lemoine that was not apparent in early research; the characteristic facial features of FAS. These were identified by these modern researchers as short palpebral fissures, epicanthal folds, smooth philtrum, flat midface, short nose, and thin upper lip (Jones & Smith, 1973; Lemoine et al., 1968). Although their research was arguably more sound and unbiased than older research in prenatal alcohol exposure, they also introduced the Face of FAS to the world. Although Lemoine's research was "not taken seriously" (Lemoine, 2003), the Face of FAS may have played a role in increasing serious interest in this syndrome worldwide.

Although the construct of FAS/FASD has been questioned by some in the past (Armstrong, 1998), there is much evidence to suggest that alcohol has some teratogenic effect on the fetus that creates real and significant developmental concerns (Streissguth et al., 2004). There is substantial evidence to suggest that prenatal alcohol exposure can negatively impact the central nervous system (Kumada et al., 2007), and that these impacts often lead to functional deficits (Rasmussen, Horne, & Witol, 2006). It is therefore probable that unrecognized effects of prenatal alcohol exposure have impacted lives for centuries. It is possible, for example, that members of the Kallikak family had FASD (Karp et al., 1995). Although the presence of what is now known as FASD was likely present in times past, a knowledge of prenatal alcohol exposure specifically seemed to occur later, possibly as early as the late 19th century but certainly by the mid to late 20th century.

Many historical records have been taken out of context when reviewing the history of prenatal alcohol exposure, and the impacts of these histories on modern-day FASD research have likely been overestimated. Historical records do suggest an awareness of an interaction between alcohol and reproduction of some kind, as early as biblical times. However, these records do not necessarily suggest an awareness of the deleterious effects of alcohol on the developing fetus. Historical records regarding alcoholism and reproduction need to be interpreted critically, in context, and in consideration of the Zeitgeist, or the Spirit of the Times.

Bibliography

Abel, E. L. (1997). Was the fetal alcohol syndrome recognized in the ancient near east? *Alcohol & Alcoholism, 32*(1), 3-7.

Abel, E. L. (1999a). Was the fetal alcohol syndrome recognized by the Greeks and Romans? *Alcohol and Alcoholism, 34*(6), 868-872.

Abel, E. L. (1999b). "Who goes drunk to bed begets but a girl": The history of a renaissance medical proverb. *Journal of the History of Medicine, 54*, 5-22.

Abel, E. L. (2001). Gin lane: Did Hogarth know about fetal alcohol syndrome? *Alcohol & Alcoholism, 36*(2), 131-134.

Anti-Saloon League (1909). Flier no. 2: Child death rate higher in drinkers' families. Retrieved from http://www.wpl.lib.oh.us/AntiSaloon/index.html.

Armstrong, E.M. (1998). Diagnosing moral disorder: The discovery and evolution of fetal alcohol syndrome. *Soc. Sci. Med., 47*(12), 2025-2042.

Calhoun, F., & Warren, K. (2007). Fetal alcohol syndrome: Historical perspectives. *Neuroscience & Biobehavioral Reviews, 31*(2), 168-171.

Chudley, A. E., Conry, J., Cook, J. L., Loock, C., Rosales, T., & LeBlanc, N. (2005). Fetal alcohol spectrum disorder: Canadian guidelines for diagnosis. *Canadian Medical Association Journal, 172*(Suppl), S1-S21.

Crowe, L. (1985). Alcohol and heredity: Theories about the effects of alcohol use on offspring. *Social Biology, 32*(3-4), 146-161.

Davis, V. E. (1980). The fetus and alcohol. *Medical Journal of Australia, 1*, 558.

Elderton, E., & Pearson, K. (1910). *A first study of the influence of parental alcoholism on the physique and ability of the offspring.* London: Cambridge University Press.

Goddard, H. H. (1912). *The Kallikak family: A study in the heredity of feeble-mindedness.* New York, NY, US: MacMillan Co.

Jones, K. L., Smith, D. W., Ulleland, C. N., & Streissguth, A. P. (1973). Pattern of malformation in offspring of chronic alcoholic mothers. *Lancet, 7815*, 1267-1271.

Jones, K. L., & Smith, D. W. (1973). Recognition of the fetal alcohol syndrome in early infancy. *Lancet, 2*(7836), 999-1001.

Jones, K. L., Smith, D. W., Streissguth, A. P., & Myrianthopoulos, N. C. (1974). Outcome in offspring of chronic alcoholic women. *Lancet, 1*(7866), 1076-1078.

Karp, R. J., Qazi, Q. H., Moller, K. A., Angelo, W. A., & Davis, J. M. (1995). Fetal alcohol syndrome at the turn of the 20th century: An unexpected explanation of the Kallikak family. *Archives of Pediatrics & Adolescent Medicine, 149*(1), 45-48.

Koren, G., Nulman, I., Chudley, A.E., & Loocke, C. (2003). Fetal alcohol spectrum disorder. *CMAJ, 169*(11), 1181-1185.

Kumada, T., Jiang, Y., Cameron, D. B., & Komuro, H. (2007). How does alcohol impair neuronal migration? *Journal of Neuroscience Research, 85*(3), 465-470.

Lemoine, P. (1994). An historical note about the foetal-alcohol syndrome: A letter from professor Lemoine. *Addiction, 89,* 1021-1023.

Lemoine, P. (2003). The history of alcoholic fetopathies. *J FAS Int, 1*, e2.

Lemoine, P., Harrouseau, H., Borteyru, J. P., & Menuet, J. C. (1968). Les enfants de parents alcoholiques: Anomalies observees a propos de 127 cas. *Ouest Medical, 8*, 476-482.

Malcolm, M. T. (1984). Foetal alcohol syndrome: Historical aspects. *Alcohol and Alcoholism, 19*(3), 261-262.

Rasmussen, C., Horne, K., & Witol, A. (2006). Neurobehavioral functioning in children with fetal alcohol spectrum disorder. *Child Neuropsychology, 12*(6), 453-468.

Reid, G. A. (1903). Human evolution; with especial reference to alcohol. *British Journal of Medicine., 2*, 818-820.

Rodin, A. E. (1981). Infants and gin mania in 18th-century London. *JAMA, 245*(12), 1237-1239.

Shibley, I. A., & Pennington, S. N. (1998). Historical misrepresentation in science: The case of fetal alcohol syndrome. *Science & Engineering Ethics, 4*(4), 427-435.

Streissguth, A. P. (1997). *Fetal alcohol syndrome: A guide for families and communities*. Baltimore, MD: Paul H. Brookes Publishing Co.

Streissguth, A. P., Bookstein, F. L., Barr, H. M., Sampson, P. D., O'Malley, K., & Young, J. K. (2004). Risk factors for adverse life outcomes in fetal alcohol syndrome and fetal alcohol effects. *Journal of Developmental & Behavioral Pediatrics, 25*(4), 228-238.

Sullivan, W. C. (1899). A note on the influence of maternal inebriety on the offspring. *Journal of Mental Science., 45*, 489-503.

Warner, R. H., & Rosett, H. L. (1975). The effects of drinking on offspring: An historical survey of the American and British literature. *Journal of Studies on Alcohol, 36*(11), 1395-1420.

What might have been: Sullivan may have impacted modern prenatal alcohol research under different circumstances

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What might have been: Sullivan may have impacted modern prenatal alcohol research under different circumstances

William Sullivan's 1899 paper, *A Note on the Influence of Maternal Inebriety on the Offspring*, often cited as a precursor to modern fetal alcohol research, was published at a time when alcoholism was a topic of great interest in Europe and North America. Emerging theories of heredity, eugenics, and temperance movements drove alcoholism research from about 1860 to 1910. In addition to an upsurge of medical literature on alcoholism, numerous journals devoted to the topic were formed, e.g. Journal of Inebriety (Warner & Rosett, 1975). Benedict Morel's theory of hereditary degeneration was influential in the medical field generally, including research on alcohol and reproduction. This theory postulated that degenerative traits, of which alcoholism was thought to be one of the more obvious, pass on hereditarily and accumulate over generations, eventually leading to the extinction of the family bloodline by about the fourth generation (Morel, 1857). Because heredity was the transmitting mechanism for degenerative traits, paternal alcoholism was generally considered as detrimental as maternal alcoholism. While some researchers and activists were concerned that alcoholism would degenerate society as a whole, others suggested that through degeneracy and extinction of family lines the propensity for alcohol would limit itself (Reid, 1903). Because alcohol was readily available and its effects observable and treatable, the study of alcoholism as a degenerative condition was of particular interest, and most alcoholism research during this period supported

the degeneration theory. Sullivan strongly advocated the theory of degeneration in his alcoholism research (Bynum, 1984).

As a physician serving a Liverpool prison, Sullivan had access to life histories of alcoholic female inmates. His 1899 study involved two main components; an epidemiological study of female inebriates and their children, and a case study of 11 alcoholic mothers who were imprisoned while pregnant. Citing prior research summarizing the alcoholic tendencies of parents of "degenerates", Sullivan's aim was to distinguish alcoholism as the primary degenerative agent by investigating "not alcoholism in the ancestry of the degenerate, but degeneracy in the descendants of the alcoholic" (p. 490). The research design constructed by Sullivan, the epidemiological data he collected, and the analyses he conducted were quite impressive. Sullivan acknowledged limitations to the data while maintaining that results could be generalized to parental alcoholism. The validity of the study was enhanced by excluding inmates who were suspected of "other degenerative factors", such as tuberculosis or syphilis, or who were "markedly neurotic". When analyzing outcomes of 600 children born to 120 alcoholic mothers, he found that the majority of children died before age two. In addition, child mortality rates were higher amongst alcoholic mothers compared to sober female relatives.

Perhaps a more important aspect to Sullivan's paper was a case study of 11 alcoholic mothers who were forced to abstain from alcohol during pregnancy because of imprisonment. Amongst these mothers Sullivan found decreased mortality of children who were in utero during imprisonment relative to their

siblings who were likely exposed to alcohol prenatally. Although Sullivan acknowledged that other environmental factors related to parental alcoholism could play a role in child mortality rates or negative outcomes, he suggested not only that the impact of maternal alcohol consumption was considerably greater than that of paternal alcohol consumption, but that alcohol appeared to have a "direct toxic action on the embryo" (p. 499).

A direct action of alcohol on the developing fetus proposed by Sullivan has been confirmed in modern day prenatal alcohol research. Increases in still births (Strandberg-Larsen et al., 2008) and infant mortality (Strandberg-Larsen et al., 2009) have been confirmed amongst mothers who drink heavily or binge while pregnant. The impact of alcohol on the developing fetus extends beyond increased mortality, however, as research has confirmed a neurodevelopmental impact caused by heavy prenatal alcohol consumption. Although the neurocognitive profile continues to emerge, consequences of heavy prenatal alcohol exposure includes deficits in intellectual ability, attention and speed of information processing, executive functioning, language, visual perception and construction, learning and memory, and number processing (Kodituwakku, 2009).

Research on the neurodevelopmental effects of prenatal alcohol exposure exploded in the 1970s, spurred by papers published by dysmorphologists David Smith and Kenneth Lyons Jones of the University of Washington in Seattle, United States. These physicians and their colleagues described developmental delay, microcephaly, growth deficiency, and aberrant facial features in a handful of children born to alcoholic mothers (Jones et al., 1973; Jones et al., 1974), and

named the condition Fetal Alcohol Syndrome (FAS) (Jones & Smith, 1973). Researchers and clinicians soon observed that the physical and neurobehavioural consequences of prenatal alcohol exposure varied (Clarren & Smith, 1978). Currently, the term Fetal Alcohol Spectrum Disorder (FASD) is used to describe the range of deficits associated with prenatal alcohol exposure.

The Seattle team indicated in the first of their seminal articles that "This seems to be the first reported association between maternal alcoholism and aberrant morphogenesis in the offspring" (Jones et al., 1973, p. 1267), suggesting that they were unaware of Sullivan's publication. In their second article, however, Jones and Smith (1973) cited Sullivan's work as well as other historical references. Although his paper preceded modern fetal alcohol research by many decades, Sullivan's work did not appear to directly influence the emergence of modern prenatal alcohol research. Although Sullivan's findings were suggestive of a direct toxic effect of alcohol on the fetus, and although subsequent animal studies suggested the same (Stockard & Papanicolaou, 1918), the moral undertones of the temperance movement in the United Kingdom and the United States created a climate where alcoholism research was considered by many as moralistic and unscientific (Sanders, 2009). In addition, research may have been hindered by a developing but rudimentary understanding of genetics, heredity, toxicity, and teratology (Warren & Hewitt, 2009). A controversial yet influential 1910 paper by biometrician Karl Pearson and his colleague Ethel Elderton set the tone for research in parental alcoholism for the immediate future. They concluded that they found no marked biological relationship between intelligence, disease, or

physical health and parental alcoholism. Rather, they suggested that such deficits, more readily observed in alcoholic families, were due to social factors and poor care (Elderton & Pearson, 1910). World War I also had an impact on the availability of resources for research in this area. By 1919, when prohibition was put into effect in the United States, the perceived need for alcoholism research declined, with some researchers abandoning the topic entirely. Philip Pauly suggests that research in alcoholism and reproduction became ideologically driven. The end result was that by the 1930s the topic of alcohol and reproduction became "scientifically uninteresting" (Pauly, 1996, p. 2). The following half-century is marked by a lack of research on the topic, described by Warner and Rosett as "forgetfulness" (1975, p. 1408). In a different social/political environment perhaps Sullivan's findings could have been a springboard to further investigate the "direct toxic action" of prenatal alcohol exposure.

Although his contribution is acknowledged in modern prenatal alcohol research literature, Sullivan is typically not credited with what appears to be the first demonstrable evidence of a direct toxic effect of prenatal alcohol exposure on the fetus. Jones and Smith are often credited with discovering the harmful consequences of prenatal alcohol exposure, but it is more accurate to suggest that they brought the neurodevelopmental impacts of prenatal alcohol exposure to the forefront. In fact, the impetus for the Seattle team's research emerged with observations made by Christie Ulleland (1972), a student who was listed as a secondary author on the first paper. Yet in 1968, Paul Lemoine, a physician in Paris, France, published a clinical description of 127 children of alcoholic mothers

(Lemoine et al., 1968). His descriptions of physical malformations, developmental delays, and behavioural challenges were very similar when compared to the descriptions of 11 children by the Seattle team (Jones & Smith, 1973). Lemoine's study, however, did not appear to have any immediate influence. Lemoine also acknowledged that an unpublished thesis in 1957 by Jacqueline Rouquette in France, describing malformations of children born to alcoholic parents, preceded his work (Lemoine, 1994). The Seattle team was not the first to discover the consequences of prenatal alcohol exposure, as has been frequently cited in research articles glossing over the history of prenatal alcohol research (Shibley & Pennington, 1998). Rather, their contribution, and a critical one at that, was successfully generating clinical and research interest in the topic.

The recent evolution of research on alcohol and reproduction can provide important lessons for reviewing the histories of research in scientific publications; those who make a discovery are not always immediately credited with their discoveries; that discoveries made at an earlier time may or may not influence later re-kindling of research topics; that the evolution of knowledge is not necessarily linear or constant; and that social/political thinking may guide, promote, or hinder research. To avoid making inappropriate assumptions when writing on history, researchers should think critically about the primary and secondary sources they are citing, consider the contexts of their references, and ensure they are giving credit where credit is due.

Bibliography

Bynum, W.F. (1984). Alcoholism and degeneration in 19th century European medicine and psychiatry. *British Journal of Addiction,* 79(1), 59-70.

Clarren, S.K. & Smith, D.W. (1978). The fetal alcohol syndrome. *New England Journal of Medicine,* 298(19), 1063-1067.

Elderton, E. & Pearson, K. (1910). *A first study of the influence of parental alcoholism on the physique and ability of the offspring.* London: Cambridge University Press.

Jones, K. L., Smith, D. W., Ulleland, C. N., & Streissguth, A. P. (1973). Pattern of malformation in offspring of chronic alcoholic mothers. *Lancet, 7815,* 1267-1271.

Jones, K. L., & Smith, D. W. (1973). Recognition of the fetal alcohol syndrome in early infancy. *Lancet, 2*(7836), 999-1001.

Jones, K. L., Smith, D. W., Streissguth, A. P., & Myrianthopoulos, N. C. (1974). Outcome in offspring of chronic alcoholic women. *Lancet, 1*(7866), 1076-1078.

Kodituwakku, P.W. (2009). Neurocognitive profile in children with fetal alcohol spectrum disorders. *Developmental Disabilities Research Reviews,* 15(3), 218-224.

Lemoine, P. (1994). An historical note about the foetal-alcohol syndrome: A letter from professor Lemoine. *Addiction, 89*, 1021-1023.

Lemoine, P., Harrouseau, H., Borteyru, J. P., & Menuet, J. C. (1968). Les enfants de parents alcoholiques: Anomalies observees a propos de 127 cas. *Ouest Medical, 8*, 476-482.

Morel, B.A. (1857). *Traité des dégénérescences physiques, intellectuelles et morales de l'espèce humaine et des causes qui produisent ces variétés maladives.* Paris: J.B. Ballière.

Pauly, P.J. (1996). How did the effects of alcohol on reproduction become scientifically uninteresting? *Journal of the History of Biology, 29*(1), 1-28.

Reid, G.A. (1903). Human evolution; with especial reference to alcohol. *British Journal of Medicine, 2*, 818-820.

Sanders, J.L. (2009). Were our forebears aware of prenatal alcohol exposure and its effects? A review of the history of fetal alcohol spectrum disorder. *Can.J.Clin.Pharmacol, 16*(2), e288-95.

Shibley, I.A. & Pennington, S.N. (1998). Historical misrepresentation in science: the case of fetal alcohol syndrome. *Sci.Eng.Ethics, 4*(4), 427-435.

Stockard, C.R. & Papanicolaou, G.N. (1918). Further studies on the modification of the germ cells in mammals: the effect of alcohol on treated guinea-pigs and their descendants. *Journal of Experimental Zoology,* 27, 119-226.

Strandberg-Larsen, K., Nielsen, N.R., Gronbaek, M., Andersen, P.K., Olsen, J., & Andersen, A.M. (2008). Binge drinking in pregnancy and risk of fetal death. *Obstetrics and Gynecology,* 111(3), 602-609.

Strandberg-Larsen, K., Gronboek, M., Andersen, A.M., Andersen, P.K., & Olsen, J. (2009). Alcohol drinking pattern during pregnancy and risk of infant mortality. *Epidemiology, 20*(6), 884-891.

Sullivan, W.C. (1899). A note on the influence of maternal inebriety on the offspring. *Journal of Mental Science,* 45, 489-503.

Ulleland, C.N. (1972). The offspring of alcoholic mothers. *Annals New York Academy of Sciences, 197*(1), 167-169.

Warner, R.H. & Rosett, H.L. (1975). The effects of drinking on offspring: An historical survey of the American and British literature. *Journal of Studies on Alcohol, 36*(11), 1395-1420.

Warren, K.R. & Hewitt, B.G. (2009). Fetal alcohol spectrum disorders: When science, medicine, public policy, and laws collide. *Developmental Disabilities Research Reviews, 15*(3), 170-175.

Running Head: A Window of Opportunity

"A Window of Opportunity": The Proposed Inclusion of FASD in the DSM-5

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"A Window of Opportunity": The Proposed Inclusion of FASD in the DSM-5

Researchers have identified a host of cognitive deficits associated with exposure to alcohol in utero, including deficits in intellectual abilities, learning and memory, language, attention, executive functioning, visuospatial abilities, motor skills, and adaptive and social functioning (Riley & McGee, 2005). These primary cognitive deficits have been shown to be associated with a host of secondary disabilities, including mental illness (Famy, Streissguth, & Unis, 1998), psychiatric disorders (O'Connor & Paley, 2009), and numerous adverse life outcomes (Streissguth, Bookstein, Barr, Sampson, O'Malley, & Young, 2004).

Fetal Alcohol Syndrome (FAS), as a diagnostic term, emerged with the discovery of the negative effects of alcohol on the developing fetus (Jones & Smith, 1973). FAS conveys the most salient consequences of prenatal alcohol exposure (PAE) including facial dysmorphology, growth restriction, and central nervous system (CNS) impairment. It was soon recognized that alcohol exposure in utero could impact the developing brain even in the absence of characteristic facial features and growth restriction (Aase, Jones & Clarren, 1995). Fetal Alcohol Spectrum Disorder (FASD) is an umbrella term used to describe an array of neurocognitive and neurodevelopmental disabilities associated with PAE (Chudley, Conry, Cook, Loock, Rosales, & LeBlanc, 2005). Diagnostic terms such as Alcohol-Related Neurodevelopmental Disorder (ARND), Partial FAS, and Alcohol Related Birth Defects fall under the umbrella of FASD.

Although PAE is associated with these significant and costly deficits, diagnostic categories for FASD are not available in the current Diagnostic and

Statistical Manual of Mental Disorders, now in its fourth edition (DSM-IV-TR) (APA, 2000). FAS is a diagnosis included in the International Classification of Diseases-10th edition (ICD-10), under the heading Congenital Malformation Syndromes due to Known Exogenous Causes, Not Elsewhere Classified (WHO, 2004). Other disorders on the Fetal Alcohol Spectrum such as ARND, however, are not included in the ICD-10.

A disorder describing the negative consequences of PAE has been proposed for inclusion in DSM-5 to be published in May, 2013. In May, 2012, criteria for a newly-described condition, Neurobehavioral Disorder Associated with Prenatal Alcohol Exposure (ND-PAE), were proposed in section III of DSM-5 as a condition that requires further research, under the category of Substance Use and Addictive Disorders (APA, n.d.). Criteria include the presence of neurocognitive impairment, impairment in self-regulation, deficits in adaptive functioning, and confirmation of more than minimal alcohol exposure any time during gestation.

Disorders caused by Prenatal Alcohol Exposure – A significant issue

Due to the worldwide availability of alcohol, understanding the effects of PAE and its costs is a significant international and social issue (Clarren, Salmon & Jonsson, 2011). Prevalence of FAS has been studied in numerous countries throughout the world, with those countries whose citizens are at higher risk of binge drinking and lower socioeconomic status yielding the highest prevalence rates (Abel, 1995). Although prevalence estimates of FAS and FASD have been more conservative in the past, it is thought that they may be as high as 2-5%

amongst younger school children in the United States and some Western European countries (May et al., 2009). Support services are often needed for those diagnosed with the disorder, but many individuals in need of support do not qualify for such services (Carmichael Olson et al., 2009). For example, based on their sample of 415 patients with FASD, Streissguth et al. (2004) found that only 13% of them would qualify for disability service based on the typical cutoff of IQ less than 70 despite marked deficits in adaptive behaviour. Paradoxically, individuals with the disorder that appear higher functioning may actually fare more poorly in the long run due to difficulty accessing services. Having a diagnostic category dedicated to disorders caused by PAE in a nosological giant such as the DSM would do much to enhance diagnostic communication and enable access to much needed services for those diagnosed, their families, and their communities.

"A Window of Opportunity": The Campaign to Include FASD in the DSM

Although the deleterious effects of PAE have been recognized and well-researched since the 1970s, limited attention was paid to the inclusion of criteria for prenatal alcohol disorders in the DSM for the following two decades. FAS, by name, appeared as an etiologic factor to disability in DSM-III but was removed in DSM-III-R (APA, 1987). FAS was mentioned again briefly in DSM-IV and DSM-IV-TR as a general medical condition frequently associated with Learning Disorder, and as a medical consequence of heavy drinking during pregnancy (APA, 1994; APA, 2000). Mention of FAS in the DSMs do not include diagnostic criteria or codes but were indicated as contextual information. Discussion of FAS

and related disorders with regard to DSM often focussed on how individuals with the disorder might fit within the multiaxial classification system of DSM-III and DSM-IV (Streissguth & O'Malley, 2000). In a newsletter for the FAS Family Resource Institute, several prominent FASD diagnosticians indicated in interviews that they utilized the DSM-IV-TR for their clients who presented with a concurrent mental disorder but typically considered FASD general medical conditions (DeVries, 2006).

Momentum in promoting the inclusion of FASDs in the DSM-5 emerged through the presence of existing organizations. In 1996 the Interagency Coordinating Committee on Fetal Alcohol Syndrome (ICCFAS) was formed, chaired by the National Institute on Alcohol Abuse and Alcoholism (NIAAA). The intent of the ICCFAS (later renamed ICCFASD) was to improve communication and collaboration through the coordination of federal-level activities in the United States to address FASD-related issues, including health, education, developmental disability, research, justice, and social services. Numerous federal-level agencies participate on the ICCFASD, including the Department of Health and Human Services, the Centers for Disease Control and Prevention (CDC), Department of Education, and Department of Justice, United States.

The ICCFAS hosted an Early Childhood Neurobehavioral Assessment workshop in March, 2000 to further develop understanding of Alcohol Related Neurodevelopmental Disorder (ARND) and to address related diagnostic issues. A special session on Revising the Diagnostic and Statistical Manual of Mental

Disorders: Relevance to ARND was facilitated by Dr. Michael First, Editor of Text and Criteria for DSM-IV and co-chair and editor for work groups of DSM-IV-TR. Dr. First suggested that there was a "window of opportunity" for new diagnostic criteria for FAS to be included in DSM-5, and suggested several options for how this might be incorporated. First, the APA could include a "behavioral disorders due to prenatal exposure" category in DSM-5. Second, prenatal alcohol exposure could be listed in Axis III as a physical condition, with other diagnoses, such as learning disorder, listed as primary diagnoses. Third, FAS criteria could be included within the category of alcohol-induced disorders. Although disorders within this category were typically used to describe behaviour of adults under the influence of alcohol, it was suggested that there may be flexibility to consider alcohol within the context of a toxic exposure (First, 2000, March).

In that same year (2000), the CDC hosted the first National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effect. At this meeting there was some debate as to whether it would be advantageous to have a DSM code for FAS since the condition was listed in the ICD-9, and it was argued that the worst diagnosis for insurance reimbursement was a mental health code. Yet, it was further argued that addressing the potential inclusion of FAS in the DSM would make the condition diagnosable, thus reimbursable for adults, and that further consideration of the DSM for the Task Force was needed (CDC, 2000, December).

In 2004, a newly formed work group of the National Task Force, the Post-Exposure Work Group, advocated for recognition of FASD within government

and organizational bodies. The work group asserted that FAS needed to be a part of DSM-5 and drafted a letter from the Task Force to the APA inquiring about the inclusion of FAS (CDC, 2004, June). In addition to advocating the inclusion of FAS in the DSM, the work group focussed attention on ways of promoting eligibility for services for those with FASD. These included efforts to promote the inclusion of FAS in the Individuals with Disabilities Education Act (IDEA) (CDC, 2005, June) and to advocate for the provision of life-long supports for adults with FAS, particularly in addressing psychological, behavioural, and housing needs (CDC, 2004, June). The efforts of the work group continued until the eventual disbanding of the National Task Force in 2007.

In May, 2005, Dr. Susan Rich, a psychiatrist, co-authored an APA action paper with Dr. Roger Peele outlining issues related to FASD and recommended that the disorder be considered for future editions of the DSM (Rich, 2005). Action papers are documents that may be submitted by APA members to the APA Assembly for review and possible implementation within the association (Mamah, 2006). The authors proposed a diagnostic classification system similar to the Pervasive Developmental Disorders category of DSM-IV-TR, in that the term FASD would serve as the categorical heading but not as a diagnostic term. Rather, diagnostic terms would include existing terms FAS, ARND, and a newly proposed category called Fetal Alcohol-Induced Disorders. The action paper was endorsed by the National Organization on Fetal Alcohol Syndrome (NOFAS), NIAAA, and CDC (CDC, 2005, June). The APA Assembly 2005 approved the action paper unanimously, signaling an official starting point for the APA in

exploring the possibility of including disorders caused by prenatal alcohol exposure in the DSM. Yet, momentum within the APA created by this action paper and its unanimous approval by the Assembly appeared to wane; by 2008, Rich indicated that the action paper lay "dormant" within the DSM-5 Task Force (Rich, 2008, June).

In 2008, Dr. Howard Moss, a psychiatrist associated with NIAAA, submitted a white paper advocating for the inclusion of Alcohol-Related Neurobehavioral Syndrome in DSM-5. Dr. Moss argued that while FASD had not been a part of psychiatric taxonomy, psychiatrists routinely worked with these patients. The inability of psychiatrists to bill for services and diagnose patients with FASD was deemed to be a barrier to service (Brunk, 2009, August).

Michael First emphasized in the March, 2000 Early Childhood Neurobehavioral Assessment workshop that the APA tended to ignore reimbursement matters when revising the DSM. Yet, it appears that much of the impetus for including FASD in the DSM has been based predominantly on reimbursement issues and enabling of services. Many of the efforts of the Post-Exposure Work Group of the National Task Force were aimed at enabling services for individuals with FAS, and Moss emphasized that a DSM code was needed to add flexibility in the services psychiatrists could provide. In addition, advocacy groups, such as NOFAS and the Minnesota Organization on Fetal Alcohol Syndrome (MOFAS) issued position statements emphasizing that the absence of FASD in the DSM has resulted in treatment delivery problems and resource limitations for those with the disorder (MOFAS, n.d.; NOFAS, n.d.). It is

not surprising that service provision and reimbursement issues were central to advocacy efforts to include FASD in the DSM, given that services and reimbursements are elusive for those impacted by prenatal alcohol exposure (Carmichael Olson et al., 2009), and that a DSM code is often considered a gateway to treatment and support (Goldman & Grob, 2006; Rappo, 2002).

In 2008, the ICCFASD developed a Diagnostic Issues Work Group with the intent to revise and enhance current FASD diagnostic guidelines for research and medical practice. Much of the focus of the work group centred on ARND diagnosis, and whether it can be differentiated from other disorders given the absence of the characteristic facial features of FAS (ICCFASD, 2011, Oct-Nov). Early in 2010, the DSM revision group issued a call for comments on an early draft of DSM-5 that included FAS as a "Condition Proposed by Outside Sources". Although the Diagnostic Issues Work Group was not developed for the purpose of incorporating FASD into the DSM, the work group did develop a DSM Revision Subcommittee to address the issue. The subcommittee responded to the call for comments with a white paper that included preliminary diagnostic criteria in the style of DSM, based in part on data presented at a scientific workshop hosted by ICCFASD in May, 2009. The subcommittee subsequently submitted revised criteria to the DSM-5 Neurodevelopmental Disorders Work Group, and to the Substance-Related Disorders Work Group. In May, 2012 DSM-5 draft criteria were proposed for ND-PAE as a condition requiring further study under the category Substance Use and Addictive Disorders (APA, n.d.).

From FASD Guidelines to ND-PAE Criteria

In existing FASD diagnostic guidelines (Astley & Clarren, 2000; Chudley et al., 2005) four key domains are assessed; a) physical growth restriction, b) facial dysmorphology, c) CNS dysfunction, and d) confirmation of alcohol exposure. In the proposed ND-PAE criteria made publicly available from APA (n.d.), only two of those domains, CNS dysfunction and confirmation of alcohol exposure, were retained. This likely relates back to the underlying ideology of DSM, which tends to avoid incorporating physical signs in criteria. Rather, DSM criteria focus on behaviourally observable symptoms.

The criteria identified in the proposed DSM-5 revisions addressing maternal alcohol confirmation state:

> **A.** More than minimal exposure to alcohol at any time during gestation, including prior to pregnancy recognition. Confirmation of gestational exposure to alcohol may be obtained from any of the following sources: maternal self-report of alcohol use in pregnancy, collateral reports, or medical or other records. (APA, n.d.)

Confirmation of gestational alcohol exposure is the keystone of the FASD diagnostic process. Without confirmation of PAE, the presence of FASD cannot be confirmed. According to some FASD guidelines, however, a FAS diagnosis can be given when growth deficiency and facial features are fully expressed in the presence of CNS dysfunction (Chudley et al., 2005).

With regard to CNS dysfunction specifically, behaviourally observable symptoms and core features are listed below:

B. Neurocognitive impairment, as evidenced by one (or more) of the following:

1. global intellectual impairment (i.e., IQ of 70 or below, or a standard score of 70 or below on a comprehensive developmental assessment)
2. impairment in executive functioning (e.g., poor planning and organization; difficulty changing strategies or inflexibility; difficulty with behavioral inhibition)
3. impairment in learning (e.g., lower academic achievement than expected for intellectual level; requires special education services; specific learning disability)
4. impairment in memory (e.g., problems remembering information learned recently; repeatedly making the same mistakes; difficulty remembering lengthy verbal instructions)
5. impairment in visual spatial reasoning (e.g., disorganized or poorly planned drawings or constructions; problems differentiating left from right; problems aligning numbers in columns)

C. Impairment in self-regulation in one (or more) of the following:

1. impairment in mood or behavioral regulation (e.g., mood lability; negative affect or irritability; frequent behavioral outbursts)
2. attention deficit (e.g., difficulty encoding new information; difficulty shifting attention; difficulty sustaining mental effort)
3. impairment in impulse control (e.g., difficulty waiting turn; difficulty complying with rules; confabulating; taking possessions of others)

> **D.** Deficits in adaptive functioning as manifested in two (or more) of the following, including at least one of (1) or (2):
>
> 1. communication deficit (e.g., delayed acquisition of language; difficulty understanding spoken language; difficulty using language to express self so that the listener understands)
> 2. social impairment (e.g., overly friendly with strangers; difficulty reading social cues; difficulty understanding social consequences; acting "too young")
> 3. impairment in daily living (e.g., delayed toileting, feeding, or bathing; problems following rules of personal safety; difficulty managing daily schedule)
> 4. motor impairment (e.g., poor fine motor development; delayed attainment of gross motor milestones or ongoing deficits in gross motor function; problems in coordination and balance) (APA, n.d.)

Although the specific symptoms (i.e. impairment in executive functioning or memory) are addressed in the current FASD diagnostic guidelines, the ND-PAE criteria imply that neurocognitive impairment, impairment in self-regulation, and deficits in adaptive functioning are core features shared by all individuals with ND-PAE. This differs from current guidelines where significant deficits in CNS domains imply CNS dysfunction but not a core set of features. For example, using current guidelines, one client may receive a diagnosis based on significant deficits in learning, memory and executive functioning, whereas another client can receive an identical diagnosis based on significant deficits in attention, communication,

and daily living skills. In this way, ND-PAE is a step closer to defining a neurobehavioural profile shared by all individuals with FASD.

Like most disorders in the DSM, ND-PAE includes contextual criteria that relate to onset, functional impact, and distinctions from other conditions:

> **E.** The onset of the disturbance (symptoms in Criteria B, C, and D) is before 18 years of age.
>
> **F.** The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
>
> **G.** The disturbance is not better explained by the direct physiological effects associated with postnatal use of a substance (e.g., medication, alcohol or other drugs), another medical condition (e.g., traumatic brain injury, delirium, dementia), other known teratogens (e.g., Fetal Hydantoin syndrome), a genetic condition (e.g., Williams syndrome, Down syndrome, Cornelia de Lange syndrome), or environmental neglect. (APA, n.d.)

Another challenge in the diagnosis of FASDs involves differentiating effects of PAE from deficits caused by other factors, which can result in false positive diagnoses. Children exposed to alcohol in utero are often exposed to other risk factors that can lead to cognitive and behavioural problems (McLennan, 2011). For example, the majority of children with an FASD are raised in unstable home environments or in adoptive/foster care, which environments are often associated with childhood learning disabilities (Evans, 2001; Sullivan & Knutson, 2000).

Conclusion

The formal process of developing the criteria for ND-PAE began relatively recently. Formed in 2008, the Diagnostic Issues Work Group of the ICCFASD developed a DSM Revision Subcommittee to work on the criteria. Other efforts to advocate the inclusion of FASD in the DSM were documented in meetings of the National Task Force on FAS and FAE, facilitated by the CDC, and with advocacy groups such as NOFAS and MOFAS. Psychiatrists Susan Rich and Howard Moss also initiated efforts to include FASD in the DSM. Central to the efforts to incorporate FASD in the DSM was the recognition that FASD diagnoses are currently insufficient to obtain the services and supports clients and their significant others require (Carmichael Olson et al., 2009). A DSM diagnosis opens doors to services (Goldman & Grob, 2006; Rappo, 2002).

FASD is a lifelong disorder that causes clinically significant impairment and that results in numerous adverse life outcomes for those affected. Efforts to include FASD in the DSM have recognized the significant impact of PAE, the need for services and supports, and the potential for a DSM diagnosis to enable services. In addition, these efforts have promoted the need for standardized diagnosis to assist in building diagnostic capacity, establishing prevalence, informing prevention efforts, and facilitating research. Continued efforts to make ND-PAE a diagnosable condition in DSM-5 will further enhance understanding of this disorder and support those affected.

Bibliography

Aase, J. M., Jones, K. L., & Clarren, S. K. (1995). Do we need the term "FAE"? *Pediatrics, 95*(3), 428-430.

Abel, E. L. (1995). An update on incidence of FAS: FAS is not an equal opportunity birth defect. *Neurotoxicology & Teratology, 17*(4), 437-443.

APA. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Association.

APA. (1987). *Diagnostic and statistical manual of mental disorders - revised* (3rd ed.). Washington, DC: American Psychiatric Association.

APA. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: American Psychiatric Association.

APA. (2000). *Diagnostic and statistical manual of mental disorders - text revision* (4th ed.). Washington, DC: American Psychiatric Association.

APA. (n.d.). *Proposed draft revisions to DSM disorders and criteria.* Retrieved December 30, 2011, from www.dsm5.org

Astley, S. J., & Clarren, S. K. (2000). Diagnosing the full spectrum of fetal alcohol-exposed individuals: Introducing the 4-digit diagnostic code. *Alcohol & Alcoholism, 35*(4), 400-410.

Brunk, D. (2009, August). Should fetal alcohol spectrum disorder be included in DSM-5? *Clinical Psychiatry News*, pp. 26.

Carmichael Olson, H., Oti, R., Gelo, J., & Beck, S. (2009). "Family matters:" fetal alcohol spectrum disorders and the family. *Developmental Disabilities Research Reviews, 15*(3), 235-249.

CDC. (2000, December). National FAS task force minutes.

CDC. (2004, June). Records of the meeting of the national task force on fetal alcohol syndrome and fetal alcohol effect.

CDC. (2005, June). Records of the meeting of the national task force on fetal alcohol syndrome and fetal alcohol effect.

Chudley, A. E., Conry, J., Cook, J. L., Loock, C., Rosales, T., & LeBlanc, N. (2005). Fetal alcohol spectrum disorder: Canadian guidelines for diagnosis. *Canadian Medical Association Journal, 172*(Suppl), S1-S21.

Clarren, S. K., Salmon, A., & Jonsson, E. (2011). Introduction. In S. K. Clarren, A. Salmon & E. Jonsson (Eds.), *Prevention of fetal alcohol spectrum disorder (FASD): Who is responsible?* (pp. 1-21). Weinheim, Germany: Wiley-Blackwell.

DeVries, J. (2006, FASD: Diagnosting the spectrum - part 1. *FAS Times: Fetal Alcohol Syndrome/Family Resource Institute Newsletter.*, doi: Retrieved Oct 2, 2012 from http://www.fetalalcoholsyndrome.org/import/lop/FASDownloads/FAS-TimesWinter06.pdf

Evans, L. D. (2001). Interactional models of learning disabilities: Evidence from students entering foster care. *Psychology in the Schools, 38*(4), 381-390.

Famy, C., Streissguth, A. P., & Unis, A. S. (1998). Mental illness in adults with fetal alcohol syndrome or fetal alcohol effects. *American Journal of Psychiatry, 155*(4), 552-554.

First, M. B. (2000, March). Revising the diagnostic and statistical manual of mental disorders: Relevance to ARND. *Early Childhood Neurobehavioral Assessment for the Differential Diagnosis of Fetal Alcohol Syndrome and Alcohol-Related Neurodevelopmental Disorder.* Bethesda, MD.

Goldman, H. H., & Grob, G. N. (2006). Defining 'mental illness' in mental health policy. *Health Affairs, 25*(3), 737.

ICCFASD. (2011, Oct-Nov). Consensus statement on recognizing alcohol-related neurodevelopmental disorder (ARND) in primary health care children. Rockville, MD.

Jones, K. L., & Smith, D. W. (1973). Recognition of the fetal alcohol syndrome in early infancy. *Lancet, 2*(7836), 999-1001.

Mamah, D. (2006). Residents can help determine APA's future course. *Psychiatric News, 41*(3), 9-10.

May, P. A., Gossage, J. P., Kalberg, W. O., Robinson, L. K., Buckley, D., Manning, M., & Hoyme, H. E. (2009). Prevalence and epidemiologic characteristics of FASD from various research methods with an emphasis on recent in-school studies. *Developmental Disabilities Research Reviews, 15*(3), 176-192.

McLennan, J. D. (2011). Critical considerations for intervention planning for children with FASD. In E. Riley, S. K. Clarren, J. Weinberg & E. Jonsson (Eds.), *Fetal alcohol spectrum disorder - management and policy perspectives of FASD* (p. 369-386). Weinheim, Germany: Wiley-Blackwell.

MOFAS. (n.d.). *FASD advocacy letter and dx criteria.* Retrieved June 14, 2010, from http://www.mofas.org/dsm-V.aspx

NIAAA. (2009, May). Scientific workshop on cognitive deficits, neurobehavioral problems, and psychopathology due to prenatal alcohol exposure. Rockville, MD.

NIAAA. (n.d.). *Interagency coordinating committee on fetal alcohol spectrum disorders.* Retrieved Oct 2, 2012, from http://www.niaaa.nih.gov/ICCFASD

NOFAS. (n.d.). *FASD in DSM-5.* Retrieved June 14, 2010, from http://www.nofas.org/news/FASDinDSM-V.aspx

O'Connor, M. J., & Paley, B. (2009). Psychiatric conditions associated with prenatal alcohol exposure. *Developmental Disabilities Research Reviews, 15*(3), 225-234.

Rappo, P. D. (2002). Coding for mental health and behavioral problems: The arcane elevated to the ranks of the scientific. *Pediatrics, 110*(1), 167.

Rich, S. (2005). Fetal alcohol syndrome: Preventable tragedy *Psychiatric News, 40*(9), 12.

Rich, S. (2008, June). Through the lens of a child psychiatrist: The need to distinguish FASD from other neurodevelopmental disorders in the DSM-V. *Iceberg Newsletter, 18*(2)

Riley, E. P., & McGee, C. L. (2005). Fetal alcohol spectrum disorders: An overview with emphasis on changes in brain and behavior *Experimental Biology and Medicine, 230*(6), 357-365.

Streissguth, A. P., & O'Malley, K. (2000). Neuropsychiatric implications and long-term consequences of fetal alcohol spectrum disorders. *Seminars in Clinical Neuropsychiatry, 5*(3), 177-190.

Streissguth, A. P., Bookstein, F. L., Barr, H. M., Sampson, P. D., O'Malley, K., & Young, J. K. (2004). Risk factors for adverse life outcomes in fetal alcohol syndrome and fetal alcohol effects. *Journal of Developmental & Behavioral Pediatrics, 25*(4), 228-238.

Sullivan, P. M., & Knutson, J. F. (2000). Maltreatment and disabilities: A population-based epidemiological study. *Child Abuse & Neglect, 24*(10), 1257-1273.

WHO. (2004). *International classification of diseases* (10th rev., 2nd. ed.). Geneva, Switzerland: World Health Organization.

Discussion and recommendations: Clinical implications of historical development of DSM through examining two main disorders

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Discussion and recommendations: Clinical implications of historical development of DSM through examining two main disorders

In this paper-format dissertation, I have critically explored the historical development of the Diagnostic and Statistical Manual of Mental Disorders (DSM), autism/Asperger's Disorder, and Fetal Alcohol Spectrum Disorders (FASD). Through these reviews, analyses, and interpretations, I encourage my readers in various professional capacities to undertake their own critical and novel analyses of the DSM, other diagnostic guidelines, and the epistemology of mental disorders on the whole. It is my intent that results and conclusions of this dissertation may assist clinicians and researchers to view the DSM and mental disorder diagnosis through a critical lens and to use diagnostic criteria and guidelines more judiciously in their psychodiagnostic practices.

Because my intent is to help influence more judicious use of the DSM, dissemination of this study to readers who may benefit is key. With this goal in mind, variations of the majority of chapters in this paper-format dissertation have already been published in peer-reviewed journals, including the historical development of the DSM (Sanders, 2011a), the historical development of Asperger's Disorder as distinct from autism (Sanders, 2009a), and the historical development of FASD (Sanders, 2009b), including analysis of the work of a key predecessor in prenatal alcohol research (Sanders, 2011b). Another chapter reviewing the development of efforts to include FASD in the DSM is in press, with anticipated publication in Summer, 2013. Through dissemination of these

studies the results, analyses, and interpretations will be made available to many relevant professionals who could benefit in their diagnostic practices.

In this concluding chapter, I will synthesize the findings and analyses of the previous chapters. In addition, specific recommendations emerging from the findings will be provided to promote more judicious use of the DSM and diagnostic guidelines.

Approach to Integration

The procedure of integrating the chapters in this dissertation is based on a unique approach outlined by Aveyard (2010) that emphasizes a pseudo-qualitative analysis as a way of organizing and integrating existing literature. Like a thematic analysis (Van Manen, 1990), Aveyard's approach involves the development of themes through analysis of the data, with the data, being in this case, the chapters in this dissertation. Although Aveyard's method is intended for use in developing literature reviews, the approach is well-suited to my goal in this dissertation, which is to use historical critical analyses to encourage readers to scrutinize of the DSM and to use it more judiciously.

A potential limitation to this approach is that all sources of "data" via individual chapters emerged from my writing, and that my knowledge, worldview, biases, and skills are enmeshed in both the data and the interpretive process. In this way, the resulting themes emerging from the analysis are subjective. As indicated in the methods section of this dissertation, however, my primary goal is to spur my readers into more critical and novel analyses of the

DSM and their own diagnostic practices, even if our interpretations of the information conflict.

In context of the goal of the study to promote more judicious use of the DSM and other diagnostic criteria, the following dominant themes emerged: 1) modern DSMs espouse a medical/disease model to diagnosis of mental disorders; 2) the processes of defining DSM disorders and their criteria are fallible; 3) for better or worse, use of the DSM is firmly entrenched in clinical practice; 4) the DSM and other diagnostic guidelines provide a reference point for mental disorder diagnosis; and 5) the DSM and other diagnostic guidelines provide means for interdisciplinary communication, which can promote improved outcomes. Descriptions and explanations of these themes are outlined below.

Discussion of Themes

Modern DSMs espouse a medical/disease model to diagnosis of mental disorders

The shift toward a medical model for mental disorder diagnosis has been well established in the literature (Grob, 1991), and is of central importance in modern clinical practice. Beginning with its 3rd edition (APA, 1980), use of descriptive criteria based on observable symptoms formed the basis for diagnosis of mental disorders in the DSM. While its intent was to improve the reliability and objectivity of psychodiagnosis, this approach presents a number of limitations in practice.

First, a descriptive approach to diagnosis draws minimal attention toward psychosocial correlates of mental disorders (Cosgrove, 2005). The multi-axial

format utilized since DSM-III provides a landing point for psychosocial factors in the diagnosis of a mental disorder, yet has no direct impact on a resulting diagnosis unless it is actively considered as such by the diagnostician. Based on my professional experience, however, the multi-axial system is not used often in practice.

Second, use of a medical model implies that mental disorders pre-exist because it implies a common biological basis amongst patients who are affected. While there has been substantial research evidence suggesting biological commonalities amongst patients diagnosed with the same conditions (e.g. schizophrenia and the dopamine hypothesis), the DSM does not account as well for lifetimes of experience, which in many cases can trigger mental conditions (e.g. stress-diathesis model). In addition, regardless of underlying biology, there is a risk that "problems of living" are pathologized, which can result in stigma and unneeded services (Szasz, 1960).

Third, given a medical basis for mental disorders, the reliability of diagnoses of mental disorders is considered fundamental in the DSM context. It was in part the lack of reliability of psychiatric diagnoses that spurred the development of the empirical, descriptive approach to diagnosis in DSM-III. On a case-by-case basis in practice, however, the comparability of mental disorders to like cases is much less important than the impact it has on functioning for the client. In addition, on a case-by-case basis mental disorder diagnosis may not be reliable. This is demonstrated in the Not-Otherwise Specified (NOS) category of many DSM disorders, where conditions that are marginally alike may be

classified within the same diagnostic category. Establishing the reliability of a diagnosis from a medical model can also be problematic because a "reliable" mental disorder implies a "reliable" treatment or cure. Although there are some common treatment practices for certain conditions (e.g. stimulant medication for Attention Deficit/Hyperactivity Disorder), the best treatment approach for a certain case may not be considered. On a case-by-case basis, individualized intervention in the treatment of mental disorders should be considered.

The processes of defining DSM disorders and their criteria are fallible

The DSM has often been called the "Bible" of mental disorder diagnosis (Anand & Malhi, 2011). Based on professional experience, I believe it is at times used with such confidence and deference as if it were a gift from deity. Rather, the DSM is a constitutive manual based on research that is often quite limited (Cooper, 2004). Disorders and criteria included in the DSM are often arbitrary (Jablensky, 2007), and changes in the DSM may or may not be progressive or evolutionary. Evaluation of criteria in research has not yet shown strong levels of reliability and validity of mental disorders in the DSM (Hyman, 2010). Changes in DSM editions and criteria are subject to internal and external influences, including political and corporate pressures (Pilecki, Clegg, & McKay, 2011). These and other potential factors should weigh-in on clinicians' levels of confidence when using the DSM in their diagnostic practices, so that the diagnostic process itself is continually under scrutiny.

Use of the DSM is firmly entrenched in clinical practice

Although the DSM is widely considered an authority in the diagnosis of mental disorders (Anand & Malhi, 2011), use of the manual in practice is often based on institutional, organizational, and corporate mandates. It is often an institutionally-relevant DSM code that opens the door to services; likewise, a lack of a diagnosis, or a diagnosis not deemed serviceable by an institution will often close the door to services (Goldman & Grob, 2006). Although the presence or absence of a DSM code creates a relatively straightforward decision-making process for service provision, it does little to benefit the host of clients in need of support but who would not qualify. This is particularly relevant for individuals diagnosed with FASD and who are in dire need of supports, but who do not qualify for a DSM code.

Because the type of diagnosis often opens the door to needed services, human services professionals, such as psychologists, social workers, and mental health counsellors, are often faced with an ethical quandary. Because of client needs, or for other purposes, professionals may be tempted to intentionally misdiagnose clients, a practice that has been established in the literature (Kirk & Kutchins, 1988). While ethical guidelines in these areas of practice typically place the well-being of clients as top priority, intentional misdiagnosis should not be practiced as it is a gross violation of professional conduct (Braun & Cox, 2005).

The DSM and other diagnostic guidelines provide a reference point for mental disorder diagnosis

Despite its limitations, the DSM plays an important role in standardizing mental disorder diagnosis (APA, 2000). Individual clinicians come with different knowledge and skill bases, worldviews, and biases that can emerge in the diagnostic process (Whaley & Geller, 2007). The DSM can act as a standard against personal and professional biases. Reflecting on my own practice, I have seen some of my biases emerge with regard to specific disorders in diagnostic practice. I have also seen other clinicians with a penchant for diagnosing certain conditions when I believe alternate diagnoses would be more suitable. These significant challenges exist even with the presence of a standard nosology in the DSM-IV-TR, which the other clinician and I both adhere to. Without a standardized nosology, the separation between different diagnoses would be far more drastic.

The DSM and other diagnostic guidelines provide means for intra/interdisciplinary communication

The creation of DSM-I emerged due to the need for a common diagnostic language amongst diagnosticians (APA, 1952). Regardless of arbitrariness or even the validity of diagnostic criteria and guidelines, clinicians and researchers need to speak the same diagnostic language in order to better understand the conditions they diagnose and treat. Use of a common language creates a starting point for communicating about clients on an individual basis and assists in the development of service provisions and meeting of client needs.

In summary, there appear to be significant limitations to this diagnostic manual, which has reached hegemonic status. The DSM seems to have fallen short of its ideal of establishing a reliable approach to mental disorder diagnosis. There may be flaws in both the processes of establishing criteria and in the underlying principles of mental disorder diagnosis. In many cases, the DSM may provide little direct benefit to clients apart from meeting institutional, organizational, and corporate mandates for enabling service provision. This benefit, however, should not be underestimated. Although it does not appear to have reached its intended level of reliability, the DSM does provide some degree of consistency in diagnosis, which can help to identify clients in need of support and which facilitates communication between professionals. The DSM is far from infallible, but from a clinical perspective, we (including our clients) are on the whole better with it than without it. If used judiciously, the DSM can be a helpful tool that can open doors to needed services and assist diagnosticians to better understand client needs.

Recommendations

Based on the themes that emerged above and my professional experiences thus far, a number of recommendations are provided for clinicians and researchers in diagnostic practice.

Looking at the big picture

In my professional experience, *assessment* is often addressed synonymously with *diagnosis*, whereas there are important distinctions between the two. Diagnosis involves following diagnostic criteria using appropriate

assessment methods. Assessment is described by Hunsley and Mash as "a decision-making task in which the psychologist must iteratively formulate and test hypotheses by integrating data" (2011, p. 7). As defined in this way, assessment is a broad activity that includes identification of client strengths and assets, weaknesses and drawbacks, potential resources for mitigating struggles, and specific recommendations to improve client outcomes. Such hypotheses explored through the assessment process can include but are not limited to diagnoses of mental disorders. Appropriate assessment procedures can help to identify appropriate case-managed interventions and existing strengths and can thus pave the way for improved outcomes for clients. In short, appropriate assessment can paint the big picture, and diagnosis may or may not be a central part of it.

As I often found in my professional practice, referral sources and clinicians most often consider diagnosis to be the central and core purpose for an assessment. This may present with a potentially weighty confounding factor; where diagnoses are sought, they are almost inevitably found (Cooper, 2004). Keeping focus on the big picture of the client's clinical profile and resulting functioning can help to defend against a hunt for a diagnosis by considering non-diagnostic factors that could contribute to the presenting problem(s).

Diagnoses should be sufficiently supported by data

Data sources should be critically evaluated for both credibility and suitability for assessing the presenting problem. Regardless of the validity and reliability of the assessment tool being used, any assessment procedure involves some degree of error, though error is not always statistically measurable as it is in

many psychometric tests. Critical evaluation of data sources includes reference to existing diagnoses and professional reports. In my professional practice, I have come across numerous situations where an individual is diagnosed with a disability, only to find no primary documentation supporting a diagnosis in available records. In one case in particular, it appeared that a secondary student's diagnosis of FAS (which resulted in specialized support and funding) emerged as a result of speculated prenatal alcohol exposure and observed behavioural challenges as noted by a teacher on an Individualized Program Plan (IPP). Subsequent professionals confirmed a diagnosis of FAS, based on previous reports. Yet, upon follow-up, we found that no diagnostic procedure had taken place with a FASD diagnostic clinic or with a medical professional. Clinicians must take caution when critically evaluating their sources, including professional reports.

Using several sources of data, including background history, current presentation, behavioural observations, client interviews, psychometric testing, file reviews, and other testing procedures can aid in the assessment process (Sattler, 2001). Using multiple data sources allows the assessor to triangulate data and to help establish or refute the credibility of the information. When using a limited repertoire of data, the assessor runs greater risk of misdiagnosis.

Transparency with the assessment and diagnostic process

When making a diagnosis, describe client characteristics or behaviours of concern and reference them directly to the relevant diagnostic criteria. An inherent trust is placed upon the clinician to practice with integrity, and transparency in the

assessment process, including statement of limitations, is important to maintain worthiness of such trust. Transparency in the assessment process also assists future professionals to better understand the client's clinical history and facilitates greater confidence in the diagnostic process undertaken.

I recall in my professional practice encountering many professional reports that included significant and life-changing diagnoses without reference to how the data related to the diagnosis. In these cases, I was left to postulate regarding the justification and even the validity of such diagnoses.

Developing broad knowledge base of mental disorders

Having a broad knowledge base of mental disorders helps to create alternate hypotheses and facilitates the process of differential diagnosis. In the assessment process, it is easier to "rule-in" than to "rule-out" a diagnosis. Yet, hypothesis testing is critical for accurate diagnosis as multiple diagnoses in the DSM could fit a given clinical profile.

For example, our FASD clinic saw a client whom was suspected of having FASD. Although the client's clinical profile indicated that such concerns were plausible and that a diagnosis could be warranted based on current criteria, a diagnosis of FASD was deferred as her profile more precisely fit that of an alternate DSM diagnosis. Without knowledge of other diagnoses, the process of differential diagnosis could not take place.

Developing a depth of knowledge of mental disorders being diagnosed

The DSM-IV-TR provides some helpful information regarding its disorders beyond the diagnostic criteria, including central features, associated

features, prevalence, course of onset, familial patterns, and potential avenues for differential diagnosis. Yet, more in-depth and up-to-date information regarding mental disorders that are diagnosed is important in evidence-based assessment. Such knowledge can be obtained by accessing relevant literature, such as peer-reviewed journals or authoritative books, or by attending relevant conferences.

Using up-to-date editions of the DSM and other diagnostic guidelines

When espousing an evidence-based approach to assessment, it seems self-evident that the most up-to-date guidelines should be used. Yet, I have come across several instances where outdated criteria were used in the diagnosis of mental disorders. Although I indicated that changes to DSM and the disorders therein are not necessarily progressive, using more recent information increases the likelihood that the assessment will be better-informed. If nothing else, using the most recent editions of diagnostic criteria helps facilitate communication between professionals.

Using the DSM multi-axial format

Use of the multi-axial format can cause the assessor to view the larger picture of the assessment process. Use of the multi-axial format is no more time consuming than following appropriate but less structured assessment procedures, but it can assist the assessor in actively considering medical, environmental, and cognitive/personality issues that may underlie the presenting problems. The multi-axial format also brings attention to client level of functioning and impairment, which can assist in the development of recommendations.

Do not be bound by diagnostic guidelines and criteria

Assessment of individuals with a myriad of genetic traits, biological processes, and life experiences is a complex endeavor. These innumerable variables cannot be sufficiently accounted for in a set of guidelines applicable to all people in all circumstances. Using a case-management approach, the clinician is the decision maker and guidelines are tools. A diagnosis can be deferred or a second opinion can be sought if the clinician is not sufficiently convinced of the presence of a mental disorder, regardless of which diagnostic guidelines are met.

With the DSM, or any other diagnostic guidelines, recognize and appreciate strengths and limitations

There will be limitations with any diagnostic classifications and criteria, and criteria should continually be scrutinized in diagnostic practice. Yet, while current guidelines may present with many limitations, they are tools that can assist clients in the identification of challenges and provisions for intervention. Like any other tool, they should be used for that purpose to which they are intended. Clients in need of services cannot wait for the establishment of a flawless set of diagnostic guidelines.

Concluding Thoughts

Though a journey in itself, I have found the writing of this dissertation a rewarding experience both professionally and personally. As a new clinician, I feel the weight of practicing competently and ethically, and I do believe that professional conduct is inherently tied to public trust placed in the profession as a whole. Through this exercise, I have come to better understand the strengths and

limitations of many of the diagnostic criteria I use frequently in my professional practice. It has also helped me better appreciate the potential pitfalls I face in my profession. Through self-awareness and continued professional and personal development I will conduct my practice competently, ethically, and with the best interest of my clients in mind. I hope that the knowledge portrayed in this dissertation will be of benefit to the addictions counselling students I will teach in their assessment course and to my colleagues in the field.

Bibliography

Anand, S., & Malhi, G. S. (2011). From manual to bible: The questionable hegemony of DSM IV *The Australian and New Zealand Journal of Psychiatry, 45*(5), 348-350. doi:10.3109/00048674.2011.576814

APA. (1952). *Diagnostic and statistical manual of mental disorders*. Washington, DC: American Psychiatric Association.

APA. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: American Psychiatric Association.

APA. (2000). *Diagnostic and statistical manual of mental disorders - text revision* (4th ed.). Washington, DC: American Psychiatric Association.

Aveyard, H. (2010). *Doing a literature review in health and social care: A practical guide* (2nd ed.). New York: McGraw-Hill.

Braun, S. A., & Cox, J. A. (2005). Managed mental health care: Intentional misdiagnosis of mental disorders. *Journal of Counseling and Development : JCD, 83*(4), 425.

Cooper, R. (2004). What is wrong with the DSM? *History of Psychiatry, 15*(57,Pt1), 005-025. doi:http://dx.doi.org/10.1177/0957154X04039343

Cosgrove, L. (2005). When labels mask oppression: Implications for teaching psychiatric taxonomy to mental health counselors. *Journal of Mental Health Counseling, 27*(4), 283-296.

Goldman, H. H., & Grob, G. N. (2006). Defining 'mental illness' in mental health policy. *Health Affairs, 25*(3), 737.

Grob, G. N. (1991). Origins of DSM-I: A study in appearance and reality. *American Journal of Psychiatry, 148*(4), 421-431.

Hunsley, J., & Mash, E. (2011). The role of assessment in evidence-based practice. In M. M. Antony, & D. H. Barlow (Eds.), *Handbook of assessment and treatment planning for psychological disorders.* (2nd ed., pp. 3-22). New York: Guilford.

Hyman, S. E. (2010). The diagnosis of mental disorders: The problem of reification *Annual Review of Clinical Psychology, 6,* 155-179. doi:10.1146/annurev.clinpsy.3.022806.091532

Jablensky, A. (2007). Living in a kraepelinian world: Kraepelin's impact on modern psychiatry. *History of Psychiatry, 18*(71 Pt 3), 381-388.

Kirk, S. A., & Kutchins, H. (1988). Deliberate misdiagnosis in mental health practice. *Social Service Review, 62*(2), 225-237.

Pilecki, B. C., Clegg, J. W., & McKay, D. (2011). The influence of corporate and political interests on models of illness in the evolution of the DSM. *European*

Psychiatry, 26(3), 194-200. doi:http://dx.doi.org/10.1016/j.eurpsy.2011.01.005

Sanders, J. L. (2009a). Qualitative or quantitative differences between asperger's disorder and autism? historical considerations. *Journal of Autism and Developmental Disorders, 39*(11), 1560-1567.

Sanders, J. L. (2009b). Were our forebears aware of prenatal alcohol exposure and its effects? A review of the history of fetal alcohol spectrum disorder. *Canadian Journal of Clinical Pharmacology/Journal Canadien De Pharmacologie Clinique, 16*(2), e288-95.

Sanders, J. L. (2011a). A distinct language and a historic pendulum: The evolution of the diagnostic and statistical manual of mental disorders. *Archives of Psychiatric Nursing, 25*(6), 394-403.

Sanders, J. L. (2011b). What might have been: Sullivan may have impacted modern prenatal alcohol research under different circumstances. *International Journal of Epidemiology, 40*, 283-285.

Sattler, J. M. (2001). *Assessment of children: Cognitive applications* (4th ed ed.). San Diego, CA: Jerome M. Sattler, Publisher, Inc.

Szasz, T. S. (1960). The myth of mental illness. *American Psychologist, 15*(2), 113-118.

Van Manen, M. (1990). *Researching lived experience: Human sciences for an action sensitive pedagogy*. London, ON: The Althouse Press.

Whaley, A. L., & Geller, P. A. (2007). Toward a cognitive process model of ethnic/racial biases in clinical judgment. *Review of General Psychology, 11*(1), 75-96.