Introduction

International research has demonstrated that assertive community treatment (ACT) is a well-established and effective model for providing intensive treatment and psychosocial rehabilitation services to people with severe and persistent mental illnesses (Allness & Knoedler, 1998). Various Canadian provinces including Ontario have implemented the version Programs of Assertive Community Treatment (PACT) originally developed in the United States in the 1970s. The model has been shown to lead to significant reductions in psychiatric admissions and hospital stays and to improve housing stability, symptoms, and quality of life (Mueser, Bond, Drake & Resnick, 1998). While we know that people who have intellectual disabilities and severe and persistent mental disorders represent one of the most challenging-to-serve populations, seldom has research examined the efficacy of this service model for adults with intellectual disabilities. While the Ontario government funded one team dedicated to serving only adults with intellectual disabilities informal reports to the author indicated that other teams were often serving these individuals as well. This is not surprising since Health Canada suggested in 1988 that at least 0.8 per cent of the Canadian general population had an intellectual disability (Health Canada, 1988), and Ontario studies have shown that people with intellectual disabilities experience mental disorders (referred to as a dual diagnosis) at about double the rate of other citizens (i.e., 38-39) per cent (Ouellette-Kuntz & Bielska, 2009; Yu & Atkinson, 1993). As well, research from Ontario reported that people with a dual diagnosis accounted for 2.5 per cent of inpatient admissions to a general hospital psychiatric unit over a four-year period (Burge et al, 2002) and 18 per cent of all inpatients at the nine regional provincial psychiatric hospitals (Lunsky et al, 2006). Inpatients with intellectual disabilities have been found to have longer lengths of stay in psychiatric settings than those without these disabilities (Saeed et al, 2003). A systematic review of international studies on ACT for people with dual diagnosis uncovered only two randomized controlled trials (Balogh et al, 2009). Both
Editor’s Note

With the arrival of spring we offer you another issue of Synergy. In this issue we present a range of articles that highlight research projects and their findings and, report on aspects of the changing mental health landscape.

Our lead article outlines information about a key mental health community resource, assertive community treatment teams. These teams are apparently serving a significant proportion of clients with a dual diagnosis and this proportion varies greatly across the province. Our second article outlines the important clinical association between persons with schizophrenia and complications due to experiences of polydipsia. While it raises key theoretical questions it also informs the reader about laboratory research occurring at Queen’s University. Research that is aimed at uncovering useful knowledge that will hopefully soon lead to treatments for persons with schizophrenia who suffer from polydipsia and its many serious complications.

For those readers who reside in or visit the City of Kingston you may have noticed the ongoing construction to a portion of Kingston General Hospital (KGH). Indeed, a significant amount of this activity has been directed, over the past many months, to the construction of the new acute care consolidated psychiatry unit. A number of advantages should soon be realized by those citizens who use the inpatient service. They will have access to a high quality unit which ideally will help in the promotion of their mental health.

Our third article notes the major support from the Kinsmen service club in support of the child and adolescent acute care psychiatry unit expected to be open soon at KGH.

Finally, I would like to take this opportunity to thank our many Board members and Reviewers (listed on page 3) for their timely attention to detail over the past issues. A special ‘thanks’ goes to both Karen Gagnon, our Assistant Editor, and Krista Robertson, our administrative support person, for all their help.

We hope you enjoy reading this issue of Synergy. As always we look forward to your comments at robertk4@providencecare.ca.
studies were conducted in the United Kingdom, experienced methodological challenges, had small samples, and drew only tentative conclusions about the relative efficacy of the service model tested, and were not directly comparable to the PACT model (Martin et al, 2005; Oliver et al, 2005)

No information has been reported to date concerning the proportion of the province’s 78 ACT team clients who have a dual diagnosis. Recognizing this dearth of information about dual diagnosis and ACT teams generally, and in Canada specifically, we launched a preliminary study in Ontario.

Method and Results

Our key aim was to determine the proportion of Ontario’s ACT teams’ current and wait listed clients who were believed to have a dual diagnosis. We created a brief questionnaire with input from the Technical Advisory Panel (TAP) to Ontario’s 78 ACT teams and following ethical approval (i.e., from Queen’s University) sent it in late June 2009 to every ACT team leader and manager for self-administration. Recipients were requested to identify the number of current clients and wait listed clients who were believed to meet criteria for a dual diagnosis. Inclusionary diagnostic criteria from the DSM IV were included in the survey preamble.

Of the 78 Ontario ACT teams 85.9 per cent participated. The teams served a total of about 4500 clients and the number of clients served per team ranged widely from 25 to 135 for an average of 67 clients per team. Participants estimated that overall 9.3 per cent of their clientele and ten per cent of wait-list clients were estimated to have a dual diagnosis. Considerable variation in proportions of clients with a dual diagnosis was found on teams, from 0 to 100 per cent. As well, the actual number of clients with a dual diagnosis served ranged widely across teams, from 0 to 38 clients. The average number of clients with a dual diagnosis per team was 6. When the one team which serves only individuals with a dual diagnosis (sponsored by a Champlain LHIN hospital) was excluded the range was 0 to 30 clients (mean=5.7). When team data was aggregated by each LHIN area, wide variation was noted across LHINs. The LHIN with the highest proportion of clients with a dual diagnosis was the South East at 19 per cent, and that with the lowest was the Central East at 5.2 per cent.

Discussion

Substantial unexplained wide variation existed across teams as to the proportion of clients and wait-list clients estimated to have a dual diagnosis. Given the high proportion of people with a dual diagnosis served by ACT teams and that people with dual diagnosis have high rates of mental disorders, high rates of inpatient psychiatry admission, high rates of bed occupancy in psychiatric hospitals, and long lengths of stay in psychiatric hospitals, most ACT teams’ clinicians can expect a continued high volume of requests to serve people with a dual diagnosis.

Given that several teams reported case-loads with none or very few clients with dual diagnosis obvious questions emerged. Are substantial numbers of people with a dual diagnosis in those catchment areas experiencing significant unmet needs for ACT with resultant personally detrimental effects? Are other services or service
models (e.g., Developmental Services Workers, Adult Protective Service Workers, Intensive Case Management Teams) in those areas adequately providing for the needs for intensive and assertive mental health supports?

As a result of our research findings we have recommended ACT team clinicians examine their service’s referral patterns and determine if they are ensuring equitable consideration of people referred with a dual diagnosis. Similar advice holds for developmental services agency staff. They should establish if they are experiencing difficulty having clients accepted by ACT teams or if they are inadvertently biased against making these referrals to ACT teams in the first place. More study is needed and recommendations for further research, advocacy and training has been noted in the full research report (Burge, 2009) available at http://psychiatry.queensu.ca/page.asp?id=10.

References


WEBSITE INFORMATION

Frontenac Community Mental Health Services
www.fcmhs.ca

Hotel Dieu Hospital
www.hoteldieu.com

Kingston General Hospital
www.kgh.on.ca

Ongwanada
www.ongwanada.com

Providence Care
www.providencecare.ca

Queen’s University Department of Psychiatry
psychiatry.queensu.ca

Southeastern Ontario Addictions and Mental Health Services & Information
www.recoveryconnections.ca
Polydipsia in Schizophrenia: What Laboratory Rats Can Tell Us

By Emily Hawken, MSc
Centre for Neuroscience Studies, Department of Psychiatry
Queen’s University

By Nicholas J. Delva, MD, FRCPC
Departments of Psychiatry
Queen’s University and Dalhousie University

By Richard J. Beninger, PhD
Centre for Neuroscience Studies
Departments of Psychiatry and Psychology
Queen’s University

Rats’ inability to describe delusions and hallucinations, cardinal symptoms of schizophrenia, might suggest that rats cannot tell us anything about the illness. However, schizophrenia also involves other changes such as impairments of working memory, enhanced sensitivity to the stimulant effects of amphetamine, and changes in brain neurotransmitter systems. These can be detected in rats and a large number of studies now show that several different developmental, pharmacological or genetic manipulations can lead to deficits in rats like those seen in schizophrenia. These animal models of schizophrenic symptoms are providing new insights into the causes and more effective treatment of schizophrenia. In the Centre for Neuroscience Studies at Queen’s University in Kingston, we have been using a pharmacological model to study polydipsia, a sometimes life-threatening disorder involving excessive drinking beyond homeostatic need and seemingly without physiologic cause seen in a subset of patients with chronic schizophrenia.

Within the general population, one percent of people are affected by schizophrenia. A diagnosis of schizophrenia has been shown to increase the odds of developing other illnesses, for example, heart disease. Factors like poor nutrition and self-care often seen in these individuals may contribute to a higher risk of secondary physical disease. Primary polydipsia has been reported in a surprisingly large subset of chronic psychiatric in-patients, up to 20 per cent (de Leon et al, 1994). Although compulsive over-drinking is not unique to schizophrenia (Illowsky & Kirch, 1988), the majority, approximately 80 per cent, of afflicted patients have a diagnosis of schizophrenia (de Leon et al, 1996).

Clinically, polydipsia generally develops in three stages, beginning with simple primary polydipsia, followed by water intoxication and finally, physical complications secondary to persistent excessive fluid ingestion (de Leon et al, 1994). Depending on the extent of drinking, symptoms can range from temporary hyperdipsia and mental state changes, to water intoxication causing permanent brain damage, and death (Illowsky & Kirch, 1988). The association of polydipsia with schizophrenia is provocative and raises the question: is polydipsia a feature of schizophrenia or is it an unrelated illness? By studying polydipsia within the framework of schizophrenia, it may be possible to gain insight into possible treatments.

Polydipsia has been observed in rats1. When normal rats were food-restricted and then exposed to intermittent (one per minute) presentations of small food pellets many were observed to drink excessively, sometimes consuming an amount of water in a one hour session equal to their entire daily intake. In studies funded by the Ontario Mental Health Foundation, we used a pharmacological model of schizophrenic symptoms in rats to test the hypothesis that treated rats would show even greater polydipsia than that observed in normal rats. The model involved a number of injections of the glutamate receptor antagonist, dizocilpine, also known as MK-801 (Beninger et al, 2009). Humans sometimes abuse a similar compound and develop schizophrenia-like symptoms further validating the use of dizocilpine to produce schizophrenia-like symptoms in rats. We found that dizocilpine-treated rats showed a reliable tendency to become polydipsic and drank significantly more than untreated control rats.

1 To assure the humane treatment of rats, studies are conducted according to the guidelines of the Canadian Council on Animal Care and experimental protocols have to be approved by the University Animal Care Committee.
The neurobiological mechanisms that cause an animal pre-exposed to dizocilpine to drink more water are unknown, as is the pathophysiology of primary polydipsia associated with schizophrenia. Some investigators have pointed to aberrant function of the hippocampus, a subcortical structure within the medial temporal lobe. Known for its involvement in memory and learning, the hippocampus also regulates neuroendocrine systems, a pathway that has been suggested to mediate polydipsic tendencies (Goldman, 2009; Goldman et al., 2007). The hippocampus is a brain region frequently implicated in schizophrenia (Harrison, 2004) and dysfunction of neurons in this structure is emerging as a signature pathological feature of the illness (Goldman & Mitchell, 2004). The involvement of the anterior hippocampus (Schoebel et al., 2009) figures prominently in both schizophrenia and polydipsia. It is plausible then that the neuroendocrine dysregulation associated with polydipsia is secondary to the pathological neurophysiology of schizophrenia.

How dizocilpine causes animals to ingest more water than those without previous drug exposure can be speculated to reside in the actions of the drug on hippocampal function. Dizocilpine has been shown to decrease GABA interneurons in the hippocampus (Braun et al. 2007) and dizocilpine-like drugs affect hippocampal GABA receptors (Beninger et al. 2010). GABAergic neurotransmission is largely responsible for inhibitory tone of the nervous system. Without normal inhibition in the projections from the hippocampus, other systems that receive these projections may be dysregulated. GABAergic dysfunction in the hippocampus caused by dizocilpine may be responsible for the observed polydipsia in rats by affecting neuroendocrine function. One test of this hypothesis would be to evaluate the possible therapeutic effect of treatment with pro-GABA drugs on polydipsia in dizocilpine-treated rats. More research using this model needs to be conducted to determine the mechanisms underlying polydipsia.

Understanding polydipsia in animal models has the potential to suggest novel therapeutics for the human condition. Current successful treatments are limited to therapies such as behaviour modification (including simply controlling access to water) and antipsychotic medications. Developing effective treatment strategies is important to the in-patient population; this is underscored by our finding that recurrent polydipsia is an illness that significantly decreases patient longevity (Hawken et al. 2009). Preliminary findings also suggest that patients with polydipsia and schizophrenia have higher Clinical Global Index scores indicating that they are more severely mentally ill (unpublished data). It is difficult to determine which comes first: does illness severity increase predisposition to polydipsia or vice versa? Or polydipsia may be caused by the same neuropathology as that underlying schizophrenia. Does the emergence of polydipsia represent significant neuronal deterioration?

Given that polydipsia is associated with chronicity of illness, it can be speculated that putative progressive neuronal deterioration due to schizophrenia may underlie this association. These questions are yet to be answered but studies with laboratory rats are beginning to provide clues.

References


Braun I, Genius J, Grunze H, Bender A, Möller HJ, Rujescu D. Alterations of hippocampal and prefrontal GABAergic interneurons in an animal model of psychosis induced by NMDA receptor antagonism. Schizophr Res. 2007;97:254-263.


Like many people, I often read about new medical treatments and discoveries in the newspaper, magazines and increasingly on the Internet. Often, patients will take information garnered from these sources to their health care practitioner, wondering if they should adopt the latest trend. Newspapers and the Internet, each powerful tools of mass information are not always the most reliable sources. They have contributed to emerging fads in medicine and confusion among consumers, as it can be difficult to tease out fact from fiction.

An interesting website that is relevant to this topic is Media Doctor (www.mediadoctor.ca). The website reports that the press plays a critical role in communicating health messages to the public, who then often base their opinions on what they have read or heard in the press. Coverage of new medical treatments in the lay press is regarded as poor and is prone to exaggeration of facts according to Media Doctor. Hence the team of doctors at Media Doctor review these news stories with the goal of improving Canadian media coverage of new medical drugs and treatments.

The British website Behind The Headlines: Your Guide To The Science That Makes The News (www.nhs.uk/News/Pages/NewsIndex.aspx) provides unbiased and evidence-based analysis of health stories that make the news in Britain. Their goal is to explain the facts behind the headlines and give the public a better understanding of the science that makes the news.

For my next column, I will compile a list of reputable health websites that review medical conditions and treatments that clinicians’ can guide their patients to. If you have a website that you think is worthy of inclusion, please send it to me at: gagnonk@providencecare.ca

In the meantime, I am going to curl up on the sofa with my cats and read the news story: “Cat Owners Live Longer” and learn how having a cat can add as much as 10 years to my life span. As a cat lover, I was overjoyed at my potential increased longevity!
KINSMEN SUPPORT NEW INPATIENT MENTAL HEALTH UNIT

By KGH staff

Kingston General Hospital has a vested interest in helping to ensure a successful 2010 Kinsmen Dream Home Lottery. Proceeds from the 2010 lottery will support the relocation and expansion of the child and adolescent mental health inpatient unit from Hotel Dieu Hospital to KGH. The new eight-bed unit designed for the unique needs of youth to ensure a supportive, secure environment during times of crisis will move into the fourth floor of the expanded Burr wing later this fall. The home-like environment will have its own dining room, recreational area and single rooms within the larger, but separate, 37-bed inpatient mental health unit. The 32,000 square feet of space will complete the consolidation of all acute care inpatients beds from HDH to KGH. The units will ensure patients have more privacy, an enhanced therapeutic environment and better access to comprehensive medical services at KGH, such as renal, cancer and cardiac care.

The Kinsmen Club of Kingston has pledged $225,000 over three years to support the new child and adolescent mental health inpatient unit that will be named in the Kinsmen’s honour.

“The Kinsmen have long been involved in helping vulnerable people in our community through our special projects,” says Mark Donovan, President of the Kinsmen Club of Kingston. “We are delighted to be part of this particular project, which allows children and youth receiving mental health care to have a care environment specifically designed with their needs in mind.”

“This is an exciting day for children’s mental health,” said Dr. Roumen Milev, Mental Health Program Medical Director at KGH and HDH. “The Kinsmen’s gift will help us transform the care environment for children in our community when they are in crisis, providing a more caring and comfortable space that promotes recovery and healing.”

For more information, contact Kingston hospitals’ Joint Planning Office at (613) 549-6666 ext. 3629.