

What's in a Name?

The psychiatric illness currently known as schizophrenia was originally named dementia praecox. This term, literally meaning "precocious madness," first used by the French physician Morel in 1853, was later popularized by the German psychiatrist Kraepelin in 1896 to represent a chronic, incurable psychotic disorder with rapid cognitive disintegration that typically strikes in the late teens to early adulthood. In 1908, the Swiss psychiatrist Bleuler renamed it schizophrenia, to refer to splitting of mental functions including personality, thinking, memory, and perception.¹ Unlike Kraepelin's vision, Bleuler's view of the condition was that its prognosis was not uniformly poor, and that it actually consisted of a group of disorders which he referred to as "the schizophrenias." Over the next century, a precise definition was established with formal diagnostic criteria and scientific validation.

In the last 25 years, a new movement to rename the disorder has emerged. This movement is motivated by concerns that the term "schizophrenia" is confusing and misleading, generating negative stigma surrounding the illness and those who suffer with it. This term has led to widespread misunderstanding of the disorder as a problem of "split personalities," which is an entirely different and unrelated disorder. The public is further confused by the media which has unfortunately perpetuated incorrect characterizations of the disorder as representing any incoherent, contradictory, or deviant behavior, depicting individuals with this disorder as criminal, violent, and dangerous.



Carol S. North, MD, MPE, DLFAPA

An alternative common usage of the word "schizophrenia" has found its way into secondary dictionary definitions referring to contradictory or antagonistic qualities or attitudes (e.g., "schizophrenic weather patterns"). Therefore, some experts have called for a new name, aiming to increase understanding, improve the public image of the disorder, reduce stigma, and lead to better care for the disorder.¹

Many alternative names for schizophrenia have been suggested.¹⁻³ Candidates include Bleuler's syndrome, Kraepelin-Bleuler disease,

Schneider's syndrome, dopamine dysregulation disorder, salience dysregulation syndrome, neuro-emotional integration disorder, psychotic spectrum disorder, psychosis susceptibility syndrome, dysfunctional perception syndrome, and Youth Onset Conative, Cognitive and Reality Distortion (CONCORD). In 2002, Japan became the first country to rename schizophrenia, formally calling it integration disorder. In the same decade, Taiwan and Hong Kong officially renamed it dysfunction of thought and perception; next, South Korea adopted the name attunement disorder.

To date, the scientific community has not come to agree on any alternative name and it remains conflicted about whether renaming the disorder is advisable at all. Some experts do not think that a name change would necessarily affect the stigma surrounding the disorder, because it is the negative public perception of the disorder based on ignorance and fear, rather than the name itself, that is the root cause of the stigma.¹ Further, concerns have been raised that changing the disorder's name might simply transfer existing stigma from the old name to a new name. Yet others think that the scientific definition of the disorder needs to be changed along with its name, and that agreement on a new name will require discovery of the underlying causes of the disorder – a scientific achievement not expected in the near future.

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SCHIZOPHRENIA SURVIVORS



Laurie Russell (*Pseudonym*)

When Patient Becomes Doctor

Laurie's first contact with mental health services took place in 2005 when she was still at secondary school, where she cut her hand with a craft knife for no apparent reason. At the beginning nobody suspected anything more than a 'teenager's mood disorder' or even a kind of 'typical' self-injurious behavior found in girls of her age, Laurie however did not carry out the act out of distress, impulsivity or sadness. Instead, she had always claimed that she was not the one who did it, that she was not in control when it happened – someone else told her to cut her hand. However, even her psychiatrist dismissed her claims as manipulative excuses indicative of an emerging borderline personality disorder, and this 'someone else' in her mind was a sign that she was 'communicating her distress clearly.'

Disillusioned and severely let down by her first adult psychiatrist, Laurie's condition deteriorated as she started her undergraduate studies in Pharmacology. The other person from outside her own mind told her to injure herself more and more severely, to the extent that it became life-threatening. All this while Laurie denied she wanted to do any of this. She developed the ideas that her blood was contaminated with messages 'from the air,' that her thoughts were not hers and were accessible from her handwriting. But none of these belonged to her own self, her own volition. She was a puppet under the control of some sinister force. As she continued to spiral downwards, she hit her lowest point – a serious suicide attempt in November 2008, where she was found by the police and forcibly committed (called 'sectioned' under British legislation) to a psychiatric hospital.

Sadly Laurie was handed back to the same psychiatrist who discharged her with no follow-up care at all. Still, even the suicide attempt was not her own behavior. Her future husband (then partner) arranged for her to seek a second opinion, where she was finally diagnosed with schizophrenia and put on an atypical antipsychotic medication. This, for Laurie, was absolutely life-saving. She also requested a new psychiatrist in the National Health Service. Despite a few more hospitalizations and changes in medication in 2009 and 2011, she has not been an involuntary patient for over 10 years.

Although she had to take a whole year away from her undergraduate course, she managed to gain the highest mark in her final year research project and a year later she was awarded a Distinction in her master's degree in Psychiatric Research Methods. When she reflects on this, she thinks her psychotic break in fact helped her decide her future career in psychiatric research. In 2014, she was able to secure a UK Medical Research Council-funded PhD studentship focusing on studying the early stages of schizophrenia and successfully passed her viva three years later with only minor corrections. Although she continues to take her medications, she is also no longer in need of psychiatric care.

“(Laurie) views herself not as a psychiatric patient but first and foremost as a researcher.”

Today she is a Postdoctoral Fellow in Mental Health Research at a prestigious British University. Whilst her personal experience, especially that of a fragmented self-consciousness, inevitably informed if not inspired her own research, she views herself not as a psychiatric patient but first and foremost as a researcher who stands on equal footing with everyone else at her stage of the career. She does tend to downplay her psychiatric history, not always because of potential stigma (despite being a sad yet relevant concern) but most importantly, because she does not wish to be defined by a label (either given by psychiatrists OR by other patients) and there is so, so much more to what she can offer than being a service user only. When a patient becomes a doctor, it means that nobody should be limited by their experiences of mental illness alone. It means that it is still possible to thrive despite a lifelong diagnosis such as schizophrenia. Laurie hopes she will continue to contribute to her beloved scientific field and to the wider society and engage in influential research that has real-life benefits, so that more patients, should they choose to, can become doctors.

CureSZ would like to thank the following for their generosity and support helping us to fulfill our mission of education and research.

The Thesing Family,
Machen Thomason
and Tom Fruth



Thank You!

TARDIVE DYSKINESIA

DAWN OF HOPE



Dr. Craig Chepke, Member, Board of Directors, The CURESZ Foundation, Private Practice Psychiatrist, and Adjunct Assistant Professor of Psychiatry, University of North Carolina School of Medicine

My last column discussed that the removal of the medication that causes Tardive Dyskinesia (TD) rarely results in remission, and therefore requires its own specific treatment. However, there were no FDA approved treatments prior to 2017. The few options for which the American Academy of Neurology (AAN) made even a moderate recommendation had significant problems: clonazepam has a serious risk of physical dependency, and certain over-the-counter supplements like ginkgo biloba have been found by the U.S. Government Accountability Office to contain much less than the amount advertised, or even none.¹

Worse yet, the medication that seems to have become the de facto standard-of-care in the U.S. for nearly all drug-induced movement disorders is benztropine (*"Cogentin"*). Not only does it carry a heavy side-effect burden, its use in TD has been categorized as *"unsubstantiated"* by the AAN because there have been no controlled clinical trials. Even the manufacturer's official prescribing information says benztropine does not help TD and can aggravate its symptoms.²

Tetrabenazine is a medication from the 1950s which was not originally intended to treat

TD but was found to have some benefit in other hyperkinetic movement disorders. Tetrabenazine inhibits a protein called VMAT2, which collects dopamine for release. People with TD have hypersensitive motor circuits, so inhibiting VMAT2 reduces the amount of dopamine released into those areas, alleviating some of the symptoms. Unfortunately, the active components of tetrabenazine are eliminated from the body so quickly, it must be taken 3 times a day and leads to greater side effects, which limited its use.

In 2017, the FDA brought new hope to those suffering with TD by approving two highly effective medications to treat it. Both valbenazine and deutetrabenazine were derived from the tetrabenazine prototype but delivered improvements in side effects and convenience by slowing its metabolism – each with a different twist. Valbenazine slows elimination by elegantly attaching the amino acid valine to the component of tetrabenazine that is the most potent and selective. Deutetrabenazine slows the elimination by cleverly swapping 8 of tetrabenazine's hydrogen atoms for those of a close variant of hydrogen called deuterium.

Valbenazine and deutetrabenazine are both usually well-tolerated and have roughly similar benefits on average. Depending on the person, one may be more favorable than the other, and each has a different trade-off. Valbenazine has two once a day dosing options, both of which can be therapeutic. Deutetrabenazine has more potential dose options, but must be taken twice a day and requires titration over several weeks to the effective doses.

VMAT2 inhibitors only treat the symptoms of TD and are not curative. If treatment with any of them is stopped, the symptoms return to where they were before treatment. Still, an increasing focus of psychiatric research involves circuits in the brain that utilize the neurotransmitter glutamate, which facilitates *"synaptic plasticity,"* essentially the brain's ability to rewire itself. It will take much more investigation, but it is theoretically possible that future medications that modulate glutamate signaling could be found to help the brain remodel itself from the damage of TD.

Perhaps the dawn of hope for TD has only begun!

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Brain Facts

by Henry A. Nasrallah, MD
CureSZ VP and Scientific Director
Brain Regions Involved in Schizophrenia
Part 3: The Cerebellum

Most people think that the main function of the cerebellum is motor coordination. However, the cerebellum has multiple non-motor functions including emotions, affect and higher cognitive functions that are relevant to many psychiatric disorders. Damage to the cerebellum can cause the following:

- Lack of coordination of posture, balance and visuo-spatial coordination
- Impaired cognition including planning, abstraction, verbal fluency, working memory, and attention
- Emotional dysfunction and affect dysregulation including blunted affect
- Language problems including dysprosodia (lack of appropriate intonation when speaking) agrammatism (speech with content words but no function words) and anomia (inability to recall the names of everyday objects)
- Personality changes, disinhibition and inappropriate behavior

It is also interesting to know that although the cerebellum looks much smaller than the cerebrum, it actually has a much higher number of brain cells (neurons and glia) than the cerebral cortex!



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What's in a Name?

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Experts advocating for a new name for schizophrenia have recommended adoption of an eponym that names a person.² For example, Down's syndrome is an eponym for the condition known as trisomy 21 (formerly "mongolism"), and Alzheimer's disease is an eponym for what was previously called "senile dementia." A recent example of an individual who lived a brilliant life with schizophrenia is the late John Nash; the motion picture *A Beautiful Mind* dramatizes the story of this acclaimed mathematician who won a Nobel Prize in 1994. An eponym based on his life with schizophrenia could transform the disorder's name to John Nash disorder, a term without negative connotations, reflecting a positive image.

Experts have cautioned that to achieve the desired ends, a new name must be accompanied by professional and public education, legislation, and availability and acceptability of effective mental health services. These efforts should involve patients and their families as well as psychiatric clinicians and researchers.² Finally, it will be important to study the medical, economic, legal, and social ramifications of a new name.³ Renaming schizophrenia can be expected to be a long process, and an even longer process to achieve the expected benefits of changing public understanding and improving life for the people who live with this illness.

VIDEO HIGHLIGHT

Why Not Use Clozapine? (Part 1 of 3)

Clozapine is the only FDA-approved medication for the treatment of hallucinations and delusions when all other antipsychotics fail to work. It is also the only FDA-approved medication for suicidal thoughts in people with schizophrenia, and has been called a "gold standard" medication for that reason. However, despite its efficacy, it is infrequently used. While 25-30% of persons with schizophrenia qualify for a trial of clozapine, only 5% actually received a trial of the drug. A weekly blood draw is required at first to test for a serious reduction in white blood cells, which may occur in 0.8% to 1.0% of patients. Side effects include sedation, weight gain, excessive salivation and hypotension. In this video series, Bethany Yeiser, CURESZ President, and Dr. Louis Cady, CURESZ Board Member, discuss clozapine.



Please consider making a donation to the CureSZ Foundation online at CURESZ.org

Your contribution will help provide education and referrals to persons with schizophrenia, their families, and those who work with the seriously mentally ill. CURESZ informs the general public to better understand this serious brain illness, and to provide scientific advances showing that there is hope for recovery, and a return to a fulfilling and normal life. The CURESZ Foundation is a 501(c)(3) nonprofit organization. All contributions are tax deductible.

"We are committed to helping individuals to cope with and recover from schizophrenia."

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