

TheScientist

EXPLORING LIFE, INSPIRING INNOVATION

[News](#) ▾ [Magazine](#) ▾ [Multimedia](#) ▾ [Subjects](#) ▾ [Surveys](#) ▾ [Careers](#) ▾

[The Scientist](#) » [Magazine](#) » [Uncategorized](#)

Supplement: Schizophrenia in Childhood

1 This diagnosis is often difficult to confirm, because it is quite common for children to have magical thinking or beliefs, and they often report auditory hallucinations that occur upon waking or sleeping (hypnagogia or hypnopompia, respectively). In addition, children may not be able to articulate their psychotic symptoms as clearly as adolescents or adults, thus making the diagnosis less clear-cut. So, too, can other symptoms mistakenly resemble psychosis, including idiosyncratic thi

By Gholson J. Lyon and Harold S. Koplewicz | December 1, 2007

 [Comment](#)  

 Like 0 

 0  [Link this](#)  [Stumble](#)  [Tweet this](#)

1 This diagnosis is often difficult to confirm, because it is quite common for children to have magical thinking or beliefs, and they often report auditory hallucinations that occur upon waking or sleeping (hypnagogia or hypnopompia, respectively). In addition, children may not be able to articulate their psychotic symptoms as clearly as adolescents or adults, thus making the diagnosis less clear-cut.

So, too, can other symptoms mistakenly resemble psychosis, including idiosyncratic thinking and perceptions associated with pervasive developmental disorder (PDD), psychotic-like symptoms arising in children with posttraumatic stress syndrome, or anxiety-related transient "phobic" hallucinations. The symptoms of psychosis in childhood differ qualitatively and quantitatively from the adult form, and these symptoms must be interpreted in relationship to the developmental age. Once you identify psychotic or quasi-psychotic symptoms in children, you look for the cause, which includes a number of disorders: autism, disintegrative disorder, affective psychoses, Asperger's syndrome, drug-induced psychosis and psychotic states caused by organic disorders, and, rarely, childhood-onset schizophrenia.

Occurring in fewer than 1 in 10,000 children, childhood-onset schizophrenia (COS) is characterized by the onset of psychosis before the age of 13 years. Most people show signs of the disease later in life: There is a dramatic increase in the frequency of schizophrenia between 13 and 18 years of age (known as adolescent-onset schizophrenia), followed by even more cases of adult-onset schizophrenia after age 18.

In fact, fewer than one percent of patients with schizophrenia receive this diagnosis in childhood. Children who do develop schizophrenia in childhood commonly show aberrant social and cognitive development early on, before the onset of psychosis. This may include significant delays in language and motor development, and social withdrawal, which can be found in many other disorders in childhood, including autism, disintegrative disorder, Asperger syndrome, and pervasive developmental disorder (PDD). Once schizophrenia is diagnosed in childhood or adolescence, the long-term outlook is dire. This is particularly true in patients who manifest the disease before age 13, about two-thirds of whom are destined to remain chronically ill.

One cohort study in New Zealand found that children who later met criteria for schizophreniform disorder or schizophrenia at age 26 years had exhibited significant impairments across a range of developmental domains (neuromotor, language, cognitive, emotional, and interpersonal development) from as young as age 3 years. Children who had reported psychotic symptoms at age 11 were also more likely to eventually develop these global pandedevelopmental difficulties.²

Environmental risk factors for schizophrenia have been well documented: for example, fetal growth retardation or hypoxia, and hazards nearer the onset of illness such as abuse of drugs, including cannabis, amphetamines, and cocaine.

In another cohort study,³ researchers associated the ages at which children learned to stand, walk, and become potty-trained to their subsequent risk of schizophrenia and other psychoses. Those children who attained milestones earlier had a lower risk of schizophrenia than the entire cohort, while those who attained milestones later had a higher risk. This relationship was not observed in nonpsychotic outcomes. Therefore, it is certainly the case that many signs and symptoms pointing to future risk of schizophrenia are likely being missed in this young population.

In terms of etiology, much evidence supports the view that environmental stressors act in combination with genetic vulnerability factors to increase the risk of developing schizophrenia. Environmental risk factors for schizophrenia have been well documented: for example, fetal growth retardation or hypoxia, and hazards nearer the onset of illness such as abuse of drugs, including cannabis, amphetamines, and cocaine.⁴ However, exposure to these environmental risks is not sufficient to cause persistent psychoses in all children and adolescents, but rather psychosis might occur in only that small subset of children and adolescents genetically predisposed to the development of such illnesses. Unlike the situation with autism,⁵ there is very little, if any, documented evidence that the number of cases of psychosis in children is increasing.

Clinicians and researchers are working on identifying at-risk children and adolescents, and developing prevention strategies including psychosocial and pharmacotherapeutic treatments.^{6,7} These at-risk youth have not yet developed psychosis (such as delusions or hallucinations), but they may have odd beliefs or other symptoms that place them in the category of "prodromal" - possibly headed toward their first psychotic break. Delusions are by definition fixed erroneous beliefs that usually involve a misinterpretation of perceptions or experiences, and their content may include a variety of themes (e.g., persecutory, referential, somatic, religious, or grandiose). Therefore, during the development of such delusions, interventions that include cognitive behavioral therapy focused on reality testing can be quite helpful.



In terms of drug treatment, prescribing antipsychotic medications for such prodromal children is quite controversial. These medications can be associated with many side effects, including weight gain and sedation, which must be considered against the possible but yet unproven benefits in terms of prevention of psychosis. Recent randomized trial with 60 patients did not show a significant difference between olanzapine and placebo in the conversion-to-psychosis rate, although there was a trend toward significance in favor of olanzapine, leading the authors to speculate that the olanzapine might reduce the conversion rate and delay the onset of psychosis. However, patients on olanzapine experienced significant weight gain, placing them at risk of obesity, with all of its attendant health problems.⁸ Further research will be needed on the use of this and other medications during the prodromal phase of schizophrenia.

Recent anatomical brain magnetic resonance imaging studies have shown a striking postpsychotic

progressive loss of cortical gray matter during adolescence in patients with COS that was greater than that seen in normal children or adolescents with atypical psychosis.⁹ Current treatments are based on a multimodal approach, including antipsychotic medication, psychotherapy, family counseling, and other specific measures of rehabilitation. Unfortunately, these treatments are usually not sufficient; hence, much more research on the etiology, treatment, and prevention of psychoses in childhood, including COS, is direly needed.

Gholson J. Lyon is a child and adolescent psychiatry resident at the NYU Child Study Center. Harold S. Koplewicz is the founder and director of NYU Child Study Center; Arnold and Debbie Simon Professor and Chair, Department of Child and Adolescent Psychiatry.

References

1. J. Biederman et al., "Phenomenology of childhood psychosis: findings from a large sample of psychiatrically referred youth," *J Nerv Ment Dis*, 192:607-14, 2004.
2. M. Cannon et al., "Evidence for early-childhood, pandevelopmental impairment specific to schizophreniform disorder: results from a longitudinal birth cohort," *Arch Gen Psychiat*, 59:449-56, 2002.
3. M. Isohanni et al., "Early developmental milestones in adult schizophrenia and other psychoses. A 31-year follow-up of the Northern Finland 1966 Birth Cohort," *Schizophr Res*, 52:1-19, 2001.
4. J.L. Rapoport et al., "The neurodevelopmental model of schizophrenia: update 2005," *Mol Psychiat*, 10:434-49, 2005.
5. S.C. Bello, "Autism and environmental influences: review and commentary," *Rev Environ Health*, 22:139-56, 2007
6. J. Addington et al., "North American Prodrome Longitudinal Study: a collaborative multisite approach to prodromal schizophrenia research," *Schizophr Bull*, 33:665-72, 2007.
7. T.H. McGlashan et al., "Recruitment and treatment practices for help-seeking 'prodromal' patients," *Schizophr Bull*, 33:715-26, 2007.
8. T.H. McGlashan et al., "Randomized, double-blind trial of olanzapine versus placebo in patients prodromally symptomatic for psychosis," *Am J Psychiat*, 163:790-9, 2006.
9. N. Gogtay et al., "Comparison of progressive cortical gray matter loss in childhood-onset schizophrenia with that in childhood-onset atypical psychoses," *Arch Gen Psychiat*, 61:17-22, 2004.

 Comment  

 Like  0 

 +1 0  Link this  Stumble  Tweet this

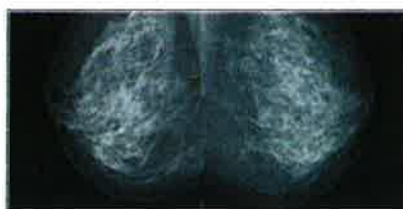
Related Articles



TS Picks: October 22, 2014

By Tracy Vence

Travel restrictions and Ebola aid; analyses of DTC genetic tests; Stanford's John Ioannidis on promoting "replication culture"



Reduced Risk?

By Molly Sharlach

A genome-wide association study identifies a SNP that could help explain the relatively low rates of breast cancer among Latina women.



Image of the Day: Novice Swimmers

By *The Scientist* Staff

Acoustic nano-tags on loggerhead turtle hatchlings have enabled researchers to closely track the animals' dispersal on their first day at sea.