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ON OR OFF THE “SPECTRUM”? 
The Complexity of Screening and Diagnosing Autism Spectrum Disorder (ASD) 

Scott Shaffer, MD, and Joaquin Fuentes, MD

Recent estimates by the Centers for Disease Control (CDC) have suggested that 1 in 68 children are diagnosed with an autism spectrum disorder (ASD). As the number of children diagnosed has risen steadily over the past decades, there has been an understandable surge of interest in the condition amongst health care professionals, teachers, the general public, and certainly amongst parents and advocates. Moreover, the DSM-5 issued a new set of diagnostic criteria that combined several DSM-IV-TR diagnoses, including autistic disorder, pervasive developmental disorder not otherwise specified (NOS), and Asperger’s Disorder into the new, singular diagnosis of autism spectrum disorder (ASD). The DSM-5 defines ASD as a neurodevelopmental condition characterized by impairments in social communication and interaction, as well as “restricted and repetitive patterns of behavior.”

Clinicians and researchers have known for a long time that earlier diagnosis generally leads to earlier identification of needs, more appropriate treatment for the child, better educational planning, and decreased family stress. Therefore, it is crucial that healthcare providers who see children (and adults) familiarize themselves with the typical signs and symptoms suggestive of ASD, as well as with screening and diagnostic tools used to help confirm this diagnosis. Clinicians often wonder how they should integrate their own clinical judgment with the results of a rating scale or diagnostic measure. This article will conclude with two diagnostic dilemmas which address this issue.

Developmental Signs of ASD

Research indicates that symptoms of ASD may be detectable in infancy, and by two years of age, a diagnosis by a professional is considered reliable. Children with ASD can present in different ways depending on their age.

The prospective follow up of babies-at-risk (that is, children that were born in the family after the diagnosis of ASD in an older sibling) constitutes an extremely useful research venture, as about 20% develope ASD themselves. This high-risk sample allows us to see the first nonspecific symptoms observable at around six months of age, notably not social aspects but motor delays, such as head lag (weak head and neck control). Then, in the following months, one can observe unusual and unexpected developmental aspects, like over-fixation on vision on objects, decreased interest in people, and slowed development of language. Characteristics of progressive social isolation, as well as non-functional interest in objects and poverty of verbal and non-verbal communication become manifest between 18 and 24 months (a one-hour video presentation of high-risk babies by expert Lonnie Zwaigenbaum is available online at http://sfari.org/sfari-community/community-blog/webinar-series/2013/webinar-lonnie-zwaigenbaum-discusses-high-risk-infant-studies).

Later in development, preschool-age children (age two to four years) typically demonstrate important milestones in their social development. For example, by two years of age, most children respond to their first name, copy the actions of other children and adults, become excited when with other children, and point to objects and pictures when they are named. Two year old children with ASD often exhibit a significant lack of these skills and are clearly less interested in others.

By the time typically developing children are three years old, they show affection for other children without prompting or concern for a child who is crying, and they can participate in conversations using two to three sentences. Three year old children with ASD often do not demonstrate these early signs of empathy, and they frequently have absent or delayed speech.

By four years of age typically developing children enjoy doing new things and their play with others becomes more creative and cooperative. Four year olds with ASD are resistant to change and start to demonstrate restricted interests and stereotyped movements.

Healthy school age children (age five to twelve years) further develop their social skills and are able to adapt to changing expectations and environments. Self-stimulatory behaviors that were present earlier typically are no longer present by this stage. School age children with ASD frequently have difficulty coping with change,
and they often continue to demonstrate self-stimulatory behaviors that can become more prominent. Adolescents and adults face increased social demands, and they typically have developed the skills to navigate their social worlds. In contrast, adolescents and adults with ASD often struggle to fit in socially with their peers.6

**Screening for ASD**

Pediatricians are often the first healthcare providers to identify concerns suggestive of ASD, although frequently the initial worries come from the parents. The American Academy of Pediatrics (AAP) recommends that all children be screened for general developmental progress by nine months of age.3 There are many general developmental screening tools used by clinicians. For example, the Denver-II Developmental Screening Test (http://denverii.com/denverii) is frequently used to screen expressive and receptive language, gross motor, fine motor, and personal-social skills. It is published by Denver Developmental Materials and is available for $40.7

AAP also recommends that all children be screened specifically for ASD at 9 months, 18 months and 24 or 30 months.1 The AAP recommends that additional screening be considered if a child is at high risk for ASD. Risk factors include having a sibling with ASD, preterm birth, and low birth weight.3 Additional indications for further evaluation include no babbling by 12 months, no gesturing (pointing, waving bye-bye) by 12 months, no single words by 16 months, no 2-word spontaneous phrases by 24 months, or loss of language or social skills at any age.2,3 While there are multiple screening measures available, the Modified Checklist for Autism in Toddlers (M-CHAT) and the Childhood Autism Spectrum Disorders Test (CAST) are commonly used as both are free, have been adapted cross culturally, can be applied for different ages, and have a wealth of research supporting them.2 The M-CHAT will be highlighted here. The CAST is applicable to older children, 4 to 11 years old, and was initially established to screen for Asperger’s disorder.

**ASD Screening with M-CHAT R/F**

The Modified Checklist for Autism in Toddlers (M-CHAT R/F, freely available at https://m-chat.org) is a screening tool created by Diana Robins which identifies children at risk for ASD.8 It was revised in 2009 in order to reduce the number of cases that screen as false positives.8 The M-CHAT R/F is based on parental report of children’s current skills and behaviors. It can be filled out by a parent, requires no special training to use, and is available for free in the public domain. It is validated for use in children between 16-30 months of age. It was developed to ensure high sensitivity, meaning it will pick up as many cases of ASD as possible. This ensures that more patients who truly have ASD will obtain a positive result.9 There is a high false positive rate associated with it, meaning that a significant number of these patients will not eventually meet criteria for ASD, but they may have other developmental problems meriting intervention. This high false positive rate has been decreased with a new two stage process.

The parent questionnaire takes less than two minutes to score. Children are scored either as low-risk, medium-risk, or high-risk based on the parents’ answers to 20 questions. Children scored as low-risk do not need further evaluation unless they are less than two years of age and will require subsequent standard developmental screens or if there is clinical concern. Children scored as medium-risk should receive the “second stage” questions of the M-CHAT R/F. If the child continues to screen positive, the child should be referred for a diagnostic evaluation by a specialist with expertise in ASD as well as be referred to early intervention programs. If a child is scored as high risk, he or she should be referred for a diagnostic evaluation and the second stage questions are unnecessary.8

If a clinician is concerned about a child with a negative M-CHAT, a referral to a specialist should still be made, but the use of the M-CHAT may help clarify the nature of the referral (e.g. “although the child screened negative, I have noticed certain behaviors that merit further evaluation”). A positive M-CHAT R/F and follow-up interview does not necessarily mean a diagnosis of ASD, as many patients will not ultimately meet criteria for the diagnosis. However, the patients who screen positive but subsequently do not have ASD should be evaluated for other developmental delays.

**Diagnostic Evaluations**

Specialists will typically employ a diagnostic measure to assist with the diagnosis of ASD. Two such measures, the Childhood Autism Rating Scale (CARS-2) and the Autism Diagnostic Observation Schedule (ADOS) will be highlighted here.
Childhood Autism Rating Scale (CARS-2)
The CARS-2, which is a diagnostic observation instrument first developed by Schopler et al., is a 15-item structured interview and observation instrument which can be used for children over two years of age. The CARS-2 is published by Western Psychological Services and is available for $175 (http://www.wpspublish.com/store/p/2646/childhood-autism-rating-scale-second-edition-cars-2). The CARS-2 can be administered by a range of professionals with knowledge of normal child development. It takes about 30-45 minutes to administer. The CARS-2 is not a standardized measure and should not be used to diagnose a child with ASD independent of clinical judgment. It can provide useful data to help support a diagnosis of ASD, or facilitate necessary referrals.

With the CARS-2, fourteen domains assess behaviors associated with ASD, and the fifteenth domain rates general impressions of ASD. Each item is scored on a scale from 1 to 4. Total scores can range from a low of 15 to a high of 60. Scores below 25.5 indicate that the patient likely does not have an ASD, scores between 25.5-36.5 indicate mild to moderate symptoms of ASD, and scores between 37-60 are consistent with severe symptoms of ASD. Perry et al., demonstrated that in a sample of 274 preschool children, there was an agreement rate of 88% between classification made by the CARS and the DSM-IV.

While the CARS has high sensitivity, it appears to over diagnose young children with intellectual disability as having ASD. Consequently, children can be incorrectly diagnosed and unnecessary referrals for expensive intervention services can occur, as is the case for all screening instruments. Filipek states that instruments used to diagnose ASD should have moderate sensitivity and good specificity. The CARS demonstrates strong internal consistency and inter-rater reliability. In 2010, a second edition for high functioning individuals, CARS2-HF, was developed including a version meant for children who have greater intellectual and verbal abilities.

Autism Diagnostic Observation Schedule (ADOS)
The ADOS is a semi-structured observational standardized assessment that includes investigator-led activities to assess communication, reciprocal social interaction, play, stereotypic behavior, restricted interests, and other abnormal behaviors. The ADOS is published by Western Psychological Services and is currently at a price of $1,995 for each kit (http://www.wpspublish.com/store/p/2647/autism-diagnostic-observation-schedule-ados).

Training is required to administer the ADOS. The interview takes about 30-45 minutes. It consists of four modules designed to be administered to individuals according to their level of expressive language. During the assessment, social interactions referred to as “presses” occur in which a range of social initiations and responses is likely to appear. The main goal of the ADOS is to provide interactions that elicit spontaneous behaviors in standardized contexts.

The ADOS was first introduced in the 1980s as a method of standardizing direct observations of social behavior, communication, and play in children suspected of having autism. It was originally proposed as a complementary instrument to the Autism Diagnostic Interview, (ADI) which later became the Autism Diagnostic Interview-Revised (ADI-R). The ADI-R consists of 93 items and is meant to be administered by an experienced clinician who has received training to use the instrument. It takes between 2-3 hours, and it is published by Western Psychological Services and is available for $85 (http://www.wpspublish.com/store/p/2645/autism-diagnostic-interview-revised-adi-r).

The ADOS-2 was published in 2012 and can be given to patients from age one year old through adulthood. There are five modules. The toddler module is used for children ages 12-30 months who have nonverbal mental ages of at least 12 months and do not consistently use phrase speech. Module 1 is used for children ages 31 months and older who have little or no phrase speech. Module 2 is used for children who have phrase speech but are not verbally fluent. Module 3 is used for children and young adolescents who are verbally fluent, and Module 4 is used for adolescents and adults who are verbally fluent.

Part of the testing is play-based and part of it is more structured. The presses are coded using a standardized rubric and then the scores are tallied using a diagnostic algorithm. Each press is given a score between 0 and 3, with 3 representing a more significant impairment. The individual items are grouped by domain including communication, reciprocal social interaction, and restricted and repetitive behaviors. The diagnostic algorithm score is then calculated which is compared to predetermined cutoff values, corresponding to diagnostic classifications of autism, autism spectrum, or non-spectrum. The toddler module uses different classifications which include little to no concern, mild to moderate concern, and moderate to severe concern. In addition, a comparison score is included which is meant to compare autism severity between individuals and over time. The score is on a scale of 1-10, with 10 being the highest severity score.

The use of the ADOS and ADI-R together provide more diagnostic clarity than when they are used alone. Using these two measures, combined with clinical judgment,
can ensure the highest levels of sensitivity and specificity.13

**Diagnostic Dilemma #1**

Jill is a 7 year old girl who presents with her parents complaining that she “drives them crazy.” She is always in her own world and she is extremely distractible. She is very oppositional and only interested in what she wants to do at any given moment. She is emotionally labile. She is so hyperactive that other children do not want to be around her. She becomes upset when routines change. Yesterday, she had a tantrum in school when there was a fire drill because the teacher would not let her play on the swings. She is described as fearless. She has some sensitivity to texture as she does not like the feeling of the tags on her shirt.

During her evaluation, she engages in reciprocal conversation with you about her interests and school. You decide to administer a CARS as Jill’s parents are anxious for answers regarding her diagnosis and you are not trained to administer an ADOS, nor is it available in the clinic. After interviewing the parents, she receives a score of 31 on the CARS-2, which is consistent with “mild to moderate symptoms of autism spectrum disorder.” You were surprised as your clinical judgment did not suggest ASD.

This case is an example of when a clinician’s judgment that a child is not “on the spectrum” differs from a diagnostic instrument which produces a score consistent with an ASD diagnosis. This patient has many symptoms which may be consistent with diagnoses such as attention-deficit/hyperactivity disorder (ADHD) and oppositional defiant disorder (ODD). Some categories on the CARS, such as Emotional Response and Activity Level, have significant overlap with these diagnoses, which consequently inflated this patient’s score. The clinician is also dependent on the parent’s reporting, which can sometimes result in over- or under-reporting of symptoms. A referral to a clinician who specializes in ASD is recommended for a complex case such as this.

**Diagnostic Dilemma #2**

Johnny is a 7 year old boy who has been having a difficult time making friends. When he is interacting with his peers, he wants to talk about different models of airplanes and other children quickly lose interest. He has difficulty reading their body language when they clearly are not interested in what he is saying. He has a difficult time understanding sarcasm and humor. Johnny is very sensitive to loud sounds. He has trouble adjusting to changes in routine. He had a terrible day in school yesterday when there was an unannounced fire drill. He had a tantrum that lasted hours. Johnny repeatedly watches documentaries about airplanes, and he can recite the dialogues by memory. You suspect that Johnny may have an ASD and you decide to refer him to your colleague who is trained to administer the ADOS. During the ADOS, Johnny is friendly and cooperative with testing. The ADOS score is below the cutoff for ASD. You are surprised as your clinical judgment suggested that Johnny had ASD.

This case is an example of when a clinician’s judgment that a child has an ASD is not supported by a diagnostic instrument. Johnny is quite intelligent and verbal. If he has already received interventions such as speech therapy, he has likely made progress through the years. A careful developmental history is essential as the clinician needs to establish whether there is a history of symptoms that the patient may have “grown out of” thanks to interventions he has received. As the ADOS is observational in nature, important information that can be obtained in the clinical history is not necessarily reflected in the score.

**Conclusion**

ASD can often be challenging and complex to diagnose as they can have vastly different presentations in patients of different ages and abilities. While there are several screening and diagnostic instruments that are valid and reliable, as in other conditions in child and adolescent psychiatry, they must be used in combination with solid clinical reasoning based on an effective interview and comprehensive history.
References:

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For more information visit [www.aacap.org/advocacyday](http://www.aacap.org/advocacyday) or contact Zach Kahan, Legislative Coordinator, at [zkahan@aacap.org](mailto:zkahan@aacap.org) or 202-587-9669.
Multiple decisions go into weighing the risks and benefits of starting a medication with a child, and one of the questions is how the medication will affect QT length. Most of the time it is not something we have to worry about because the medication is being prescribed to a medically healthy child with no significant family history. In such lower risk cases, QT prolongation may occur, but the amount is usually not considered dangerous.

The problem develops when there are existing risk factors for prolonging the QT interval, such as family history of sudden death or cardiac illness, using multiple medications that may affect cardiac conduction, or a history of long QT syndrome (LQTS). LQTS is rare, but may lead to torsades de pointes, a potentially fatal arrhythmia.

In this article we discuss why QT length matters to us, how psychiatric medications can prolong QT length, how to calculate the corrected QT (QTc), the risk factors that can cause a patient to have a baseline prolonged QT length, and when you need to do more, such as get an EKG or refer to a specialist.

Understanding the QT and QTc

We worry about the QT length because of its strong association with torsades de pointes, often referred to clinically as “torsades.” Torsades is seen on an EKG as “twisting of the points” (see Figure 1), and it is associated with syncope and sudden death. For adults, the overall mortality rate is 10-17%. Due to the strong association between torsades and QT length, the FDA has withdrawn or denied drug licenses based on QT prolongation more than any other reason in the last 10 years.

“QT” is measured on an EKG from the initial deflection, reflecting the beginning of the QRS complex to the end of the T wave. The QT interval represents the onset of ventricular depolarization to repolarization, beginning with the rapid influx of sodium ions into the cardiomyocytes, associated with the outflow of potassium ions through the rapid and slow potassium channels. The problem with looking at only the QT length is that it shortens as the heart rate increases, and the length needs corrected before clinical interpretation can begin.

There are standard methods used to deal with the dynamic nature of the QT interval and adjusting for the heart rate, allowing determination of a corrected QT interval, abbreviated “QTc”. QTc is the standard measure when assessing the risk of QT prolongation.

Calculating the QTc is an important skill for prescribers. When approaching an EKG, it may be tempting to rely on the automated interpretation when available, but this can be inaccurate, particularly in special populations like children, or given variations in the beat-to-beat interval, such as with sinus arrhythmia. The general steps for reviewing the QT on an EKG are outlined below.

The first step is to measure the QT from the beginning of the QRS complex to the end of the T wave (see Figure 2). The standard lead utilized is Lead II, as long as the T wave is clearly defined in this lead. Although there are different formulas, by far the most common approach is to calculate the QTc by using the Bazett’s formula, which requires measuring the R-to-R interval of the beat before

Figure 1. Torsades de Pointes on an EKG.
the one used for QT assessment, and then dividing the QT by the square root of the R-to-R, measured in seconds (QTc = QT/(R-to-R)^{1/2}). To improve reliability, it is good to average multiple intervals. The upper limit of normal of the QTc does not vary with age and is 450ms for females and 440 for males.

**Risk Factors for Torsades de Pointes**

Though there is an association between QTc and torsades, it is thought that there are many other factors that could increase the risk of developing torsades. Table 1 reviews risk factors for torsades that are associated with common medical conditions. Some of these risk factors are acute and need to be taken into consideration when someone is sick or in the hospital. For example, certain electrolyte disturbances including hypokalemia, hypocalcaemia, and hypomagnesemia can influence the way the heart depolarizes and repolarizes, which will influence the length of the QT.

Two of the most significant risk factors that increase the risk for QT prolongation and torsades when starting a medication are 1) another QT-prolonging medication, and 2) known history of LQTS. Nearly 3% of all medications are found to prolong the QT in some way, so it is common for a patient to be placed on medications that prolong the QT (Table 2).

Most of the medications that influence QT block one of the repolarizing potassium channels, thus prolonging the repolarization phase. The exact relationship between degree of QT prolongation and risk of torsades is not always known. For some medications the relationship is straightforward and the risk of torsades is directly associated with how much the QT is prolonged. For other medications, the risk of torsades can be high even with relatively small QT prolongation. Thus, simply knowing the relative increase in QT is not enough to know a specific medication’s risk for inducing torsades.

Medical history of QT prolongation is a risk of future QT prolongation. The extent to which it is a risk may vary depending on the individual. There is considerable individual variability. For example, a healthy child who was started on a medication known to prolong the QT may have a significant change, and another child with a history of LQTS who may have only a small change in QT. As such, it is difficult to judge the relative importance of family history and prior QT prolongation history when prescribing. Nonetheless, identifying the influence of heritable factors, mostly through family history, is considered essential in determining associated risk of torsades. Clinicians must carefully record and consider family history, particularly a history of sudden death or unexplained syncope in young individuals under 40 years of age.

Additional information in labeling of medications that increase risk for torsades based on their prolongation of the QT is available, but there is little evidence that these labels alter prescribing practices. To help people identify some of these medications, there are multiple websites, such as www.crediblemeds.org.

| Table 1: Risk Factors for Prolonged QT and Their Associated Conditions¹,³ |
|-----------------|---------------------------------------------------------------|
| **Risk Factor** | **Association**                                               |
| Electrolyte disturbances | Acute hypokalemia, chronic hypocalcemia, chronic hypokalemia, chronic hypomagnesemia |
| Medical history (endocrine) | Hyper/hypothyroidism, pheochromocytoma |
| Medical history (cardiac) | Congestive heart failure, myocarditis, complete atrioventricular block, severe bradycardia, sick sinus syndrome |
| Medical history (neurologic) | Stroke, head trauma, encephalitis, subarachnoid hemorrhage |
| Medical history (nutritional) | Alcoholism, liquid protein diet, starvation |
| Medical history (psychiatric) | Anorexia nervosa |
| Family history | Unexplained fainting spells or sudden death, epilepsy, sudden infant death syndrome (SIDS), unexplained auto accidents and drowning, Jervell Lange-Nielsen syndrome (autosomal recessive, rare), Romano-Ward syndrome (autosomal dominant, more common) |
**LQTS: A Heritable Risk**

The heritable forms of LQTS can be due to mutations on several genes. LQTS type 1 (LQTS1) is due to a KCNQ1 (potassium voltage-gated channel) mutation and is the most common type of inherited LQTS. LQTS1 has two phenotypic presentations: Romano-Ward and Jervell Lange-Nielsen syndromes. Romano-Ward is by far the most common LQTS1 and is a heterozygous condition associated with one of the 13 genes and over 500 mutations that have been identified to cause this abnormality of repolarization. The Jervell Lange-Nielsen Syndrome is very rare and is the homozygous form, associated with sensorineural hearing loss and a much more severe clinical course, including high rates of death if left untreated.

LQTS1 is the most common type of inherited LQTS. It responds well to beta blockers, and if those who have this diagnosis avoid all QT prolonging medications, their risk of sudden death is very small. As with other individuals who have long QT, individuals with KCNQ1 mutation may be at increased risk for torsades when starting a QT prolonging medication.

**Psychiatric Medications and QT**

Not all psychiatric medications are the same in their effect on QT prolongation. Citalopram, escitalopram, tricyclic antidepressants, and mirtazapine are associated with prolonged QT and the influence is often related to dosage and/or method of administration. For example with citalopram, the greatest risk is thought to occur above 40 mg/day, and with haloperidol, there is an increased risk when given intravenously.

The data unfortunately are not always consistent, and controversy exists around the Food and Drug Administration (FDA) black box warning related to prolonged QT for citalopram doses greater than 40mg. The FDA advised not using citalopram above 40 mg based on a study showing an increase in the QT interval by 18.5 ms. Subsequent studies have shown mixed results, reproducing or failing to reproduce the association. However, most studies seem to support citalopram and escitalopram increasing the QT in a dose-related fashion. On the other hand, most studies on fluvoxamine, fluoxetine, paroxetine, and sertraline have shown very small or no increase in the QT or an association with torsades (Table 3). For antipsychotics, there has been a more consistent, significant association with QT prolongation and torsades (Table 4). Phenothiazines and other low-potency typical antipsychotics, such as thioridazine, pose the greatest risk.

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### Table 2: Non-Psychiatric Medications That Prolong QT

<table>
<thead>
<tr>
<th>Type of Medication</th>
<th>Medication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antibiotics/antifungals</td>
<td>Macrolides, quinolones, pentamidine, chloroquine, halofantrine, fluconazole, ketoconazole, voriconazole</td>
</tr>
<tr>
<td>Antiarrhythmic agents</td>
<td>Quinidine, procainamide, disopyramide, sotalol, amiodarone, dofetilide</td>
</tr>
<tr>
<td>Diuretic</td>
<td>Furosemide</td>
</tr>
<tr>
<td>Estrogen antagonist</td>
<td>Tamoxifen</td>
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<tr>
<td>Opioid antagonist</td>
<td>Methadone</td>
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</table>

### Table 3: Antidepressant Medications and Risks for QT Prolongation and Torsades de Pointes

<table>
<thead>
<tr>
<th>Medication</th>
<th>Risk of Prolonged QT</th>
<th>Risk</th>
</tr>
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<tbody>
<tr>
<td>Tricyclic antidepressants</td>
<td>Longest prolongation</td>
<td>Conditional risk</td>
</tr>
<tr>
<td>Citalopram</td>
<td>Longest prolongation</td>
<td>Known risk</td>
</tr>
<tr>
<td>Escitalopram</td>
<td>Longest prolongation</td>
<td>Known risk</td>
</tr>
<tr>
<td>Mirtazapine</td>
<td>Moderate prolongation</td>
<td>Possible risk</td>
</tr>
<tr>
<td>Venlafaxine</td>
<td>Moderate prolongation</td>
<td>Possible risk</td>
</tr>
<tr>
<td>Sertraline</td>
<td>Least prolongation</td>
<td>Conditional risk</td>
</tr>
<tr>
<td>Fluoxetine</td>
<td>Least prolongation</td>
<td>Conditional risk</td>
</tr>
<tr>
<td>Paroxetine</td>
<td>Least prolongation</td>
<td>Conditional risk</td>
</tr>
</tbody>
</table>

Note: Black box warning for doses >40mg; specifically seen in elderly

---

### Table 4: Antipsychotic Medications and Risks for QT Prolongation and Torsades de Pointes

<table>
<thead>
<tr>
<th>Medication</th>
<th>Risk of Prolonged QT</th>
<th>Torsades Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haloperidol (IV)</td>
<td>Longest prolongation</td>
<td>Known risk</td>
</tr>
<tr>
<td>Haloperidol (PO/IM)</td>
<td>Medium prolongation</td>
<td>Known risk</td>
</tr>
<tr>
<td>Thioridazine (phenothiazines)</td>
<td>Longest prolongation</td>
<td>Known risk</td>
</tr>
<tr>
<td>Pimozide</td>
<td>Longest prolongation</td>
<td>Known risk</td>
</tr>
<tr>
<td>Ziprasidone*</td>
<td>Longest prolongation</td>
<td>Possible risk</td>
</tr>
<tr>
<td>Risperidone</td>
<td>Least prolongation</td>
<td>Possible risk</td>
</tr>
<tr>
<td>Olanzapine</td>
<td>Least prolongation</td>
<td>Possible risk</td>
</tr>
<tr>
<td>Quetiapine</td>
<td>Least prolongation</td>
<td>Possible risk</td>
</tr>
<tr>
<td>Paliperidone</td>
<td>Least prolongation</td>
<td>Possible risk</td>
</tr>
<tr>
<td>Iloperidone</td>
<td>Medium prolongation</td>
<td>Possible risk</td>
</tr>
<tr>
<td>Aripiprazole</td>
<td>Least prolongation</td>
<td>Possible risk</td>
</tr>
</tbody>
</table>

Note: IM = intramuscular; IV = intravenous; PO = oral

*Risk of torsades not found to be dose dependent
for QT prolongation and torsades.² As mentioned above, haloperidol has a higher risk when given intravenously, compared to orally or intramuscularly. Ziprasidone has an effect on repolarization, though this is not considered to be dose dependent.²

**Putting It Together: Assessing Risk for Long QT**

The American Heart Association provides recommendations for evaluating the risk of prolonged QT and what steps to take when considering a medication that may prolong the QT (Table 5). Other organizations also have similar recommendations, such as when to request a consultation (Table 5).

The guidelines emphasize that one of the most important parts of an assessment is a thorough past personal and family medical history, including current and past medications. One should find out about any known previous EKGs showing prolonged QT, history of unexplained syncopal or light-headed events, and any other current or past medical concerns, such as symptoms suggesting electrolyte problems or eating disorders.¹,³,¹²

When asking about family history, it is important to ask about LQTS, or syndromes known to have associated long QT, such as Jervell Lange-Nielsen Syndrome or Romano-Ward Syndrome, also anyone known to have had syncope, epilepsy, sudden infant death syndrome (SIDS), sudden death, unexplained accidents, such as fatal car accidents or drowning.¹,³,¹²

It is also essential to ask about other medications that can prolong the QT (as mentioned above) or can either inhibit or activate the cytochrome P450 enzymes, thus influencing risk (Table 5). Medications known to influence cytochrome P450 enzymes may slow down the metabolism of the medication being prescribed, leading to a larger amount of active medication in the system, and a higher rate of side effects, including prolonging the QT.

It is also important to do a physical exam and assess for abnormalities, such as tachycardia or irregular heart rhythm. If abnormalities are present, the recommendation is either to get an EKG or refer to a cardiologist for an evaluation and EKG.¹,¹²

**Referral to Cardiology**

Obtaining history from a primary care physician is indicated, especially if any cardiac risk factors are noted. This will allow the prescriber to understand the history better and may facilitate coordination of care with specialists. In addition, there are many common medications that influence the QT or the cytochrome P450 or other important enzymes, and communication with other providers is important. The primary care doctor should be able to help with medical history or other concerns that could influence making the decision to start a medication and could help with coordinating care with the pediatric cardiologist.

A referral to a cardiologist prior to initiation of medication is indicated with the QTc>460 ms.¹,³,¹² Certainly, cardiology consultation should be recommended if the child has a known congenital heart defect or other factors suggesting heart disease (especially because this may affect how the EKG will be read). It is also important to monitor the patient closely, including follow-up EKGs and referrals once steady state of the drug is reached and with each change in dosing.¹,¹²

**Conclusion**

Going back to the 16 year old with depression, there are clear risk factors that influence the decision to start a medication and what type of medication. Based on her family history and medications, discuss the risks and benefits of pharmacotherapy as well as alternatives. Next, if you decide a medication is warranted, she needs an EKG, and it would be advisable for the prescriber to consult with the pediatrician to confirm the history and discuss treatment. If the EKG or the history are concerning, then a referral to a cardiologist before starting the medication is indicated. In this case, with a relative with known LQTS, cardiology consultation is advised.

---

**Table 5: Recommendations for Monitoring for Prolonged QT**¹,¹²

<table>
<thead>
<tr>
<th>Table 5: Recommendations for Monitoring for Prolonged QT¹,¹²</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Before starting a medication</strong></td>
</tr>
<tr>
<td>Evaluate for family hx of LQTS or sudden unexplained deaths. Medical hx of palpitations, syncope, or near syncope. Medication history. Tell family about P450 affecting medications</td>
</tr>
<tr>
<td><strong>If something on first evaluation is concerning</strong></td>
</tr>
<tr>
<td>Refer to a pediatric cardiologist before starting medication</td>
</tr>
<tr>
<td><strong>Follow-up visit</strong></td>
</tr>
<tr>
<td>Evaluate for new meds and physical exam of HR and BP</td>
</tr>
<tr>
<td><strong>When to get an EKG</strong></td>
</tr>
<tr>
<td>If starting a TCA or phenothiazine and when at steady state</td>
</tr>
</tbody>
</table>

Note: Hx = history; LQTS = long QT syndrome
**Take-Home Summary:**

Numerous medications prolong the QT interval. Prescribers must know how to assess risk for QT prolongation and torsades de pointes, monitor and alter treatment accordingly, and refer for consultation in higher risk cases.

References

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Justin Schreiber, DO, MPH, is a post-graduate year-4 triple-board resident at the University of Pittsburgh Medical School, Western Psychiatric Institute and Clinic, Pittsburgh, PA. He is interested in the integration of care of child psychiatry and pediatrics, especially for chronically ill children, and increasing access to quality mental health services for all children.

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Psychiatric disorders are complex in etiology and prognosis, and most are thought to be a result of both genetics and environmental factors. As researchers delve deeper into uncovering causes and improving treatments for patients, the long-overlooked role of nutrition is entering the spotlight as a contributing factor in both cause and course of many mental illnesses.

Improper eating has been observed to exacerbate or worsen symptoms in several mental illnesses. These relationships are not yet well understood but have sparked curiosity about the interaction between nutrition and mental illness. The scientific community has been looking more closely into how what we eat nourishes not only the body but also the brain. This article will review four psychiatric illnesses that may be influenced by diet and nutrition and provide tips for asking about nutrition in the clinical setting.

Attention-Deficit Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder characterized by significant impairment in attention and/or hyperactivity and impulsivity. The causes of this disorder are largely unknown, despite being one of the most studied and diagnosed psychiatric disorders in children and adolescents.

There has been longtime speculation that food additives are to blame for increasing or worsening ADHD symptoms. Dr. Ben Feingold began the Feingold diet in 1976 for ADHD management, advising patients to eliminate certain artificial colors, dyes, preservatives, and chemicals from their diets. Many subsequent studies have found this regimen’s effects to be non-significant, but this diet nevertheless brought the relationship between food sensitivities and ADHD into the spotlight. Some subsequent studies have shown symptom improvement in ADHD patients when a food they are either allergic or sensitive to is completely removed from their diet.¹

People with ADHD have also been observed to have lower levels of omega-3 fatty acids, docosahexaenoic acid (DHA), and arachidonic acid (AA) omega-6 fatty acids in their bodies, as well as altered fatty acid metabolism. Supplementation of fatty acids has produced small reductions in ADHD symptoms in some studies, but clinical significance remains uncertain.²

When it comes to children being hyperactive, some people point to sugar consumption as a potential trigger. Although parents anecdotally report that increased sugar consumption is related to hyperactive behavior in their children, most studies have not found evidence of an association. Although high sugar consumption may lead to a spike in glucose level and provide a boost of energy, the effects on classifiable hyperactive and inattentive behavior such as attention, learning abilities, memory, and cognitive tasks have been found to be non-significant.¹

There is, however, evidence that dramatic spikes and troughs in glucose are associated with increased irritability, inattentiveness, and distractibility. For those who already have difficulties with inattention, unstable blood sugar levels may worsen these symptoms or make them more difficult to manage. Additionally, it has been documented that those with ADHD may have abnormal glucose tolerance levels. During hypoglycemia-like episodes, these individuals may suffer from headaches, difficulty concentrating, irritability, and “brain fog,” all of which may exacerbate already-present ADHD symptoms.²

Autism spectrum disorder (ASD) is characterized by communication deficits, inappropriate responses in conversations, difficulty building relationships, and misreading of nonverbal cues. ASD diagnoses have increased dramatically in recent history for unknown reasons. Treatment can be challenging, particularly in geographical areas without specialists trained in ASD.

Parents of children with ASD often try a variety of diet modifications in an attempt to improve their children’s symptoms. Gluten- and casein-free diets have recently become popular. Supporters of these diets claim that excessive opioid activity seen in autism is related to improper breakdown of gluten and casein in the body, and that the peptides from such proteins cross the blood brain barrier, delaying social and language skills. Some studies have
found such diets to be somewhat effective in areas of cognition, behavior, and social functioning, although they have received criticism for being small and/or uncontrolled. Several other double-blind controlled studies have found the diet to have no significant effect. More well-conducted, randomized trials with significant participant numbers and well-controlled diets will be needed to determine if there are, in fact, clinically significant effects or improvements in autism symptoms to warrant recommendation of such diets.

Much like in ADHD, there has been speculation that food additives, dyes, and artificial colors, flavors, and sweeteners are responsible for worsening symptomatology. More certain, it seems, is the link between autism spectrum disorders and increased incidence of GI problems. Elimination of known irritants, which may include artificial food additives and flavors, may improve mood and behavior. Additionally, those with autism often have imbalanced levels of omega-3 and omega-6 fatty acids, and so a dietary increase of these fatty acids may be beneficial.

Researchers are looking at gestation as a possible starting point for uncovering causes of autism. The impact of diet during pregnancy and of breastfeeding on incidence of ASD is currently being investigated. Autism Speaks (http://www.autismspeaks.org) has a list of guidelines for pregnant women to avoid environmental factors and certain toxic substances they believe may be linked to developmental disorders. In addition to a recommended focus on thoroughly washed produce and reduction of packaged food consumption, they advise reduced consumption of oily fish and tuna. It is their logic that several dyes and mercury from fish consumed during pregnancy are correlated with increased risk of autism.

Gestational folic acid deficiency and premature discontinuation of breastfeeding have also both been linked to increased risk of autism. While these are currently correlational studies and may potentially be linked to confounding issues (i.e., infants with neurodevelopmental issues may have trouble breastfeeding), these findings have focused more attention on the influence of maternal behavior and early nutrition on autism risk. Conclusive studies on the issue could have benefits for expectant mothers and children already diagnosed with ASD, in terms of both reducing risk and improving prognosis.

**Depressive disorders** are characterized by persistent low moods and/or irritability that affect a person’s thoughts, behaviors, and feelings. Depression is often accompanied by loss of interest or enjoyment of normal activities and daily functioning. In addition to feeling sad, empty, hopeless, anxious, and irritable, some symptoms, such as changes in appetite and weight loss or gain, are directly related to diet. Poor diets are frequently reported by depressed patients and prospective epidemiological studies have shown links between diet patterns and depressive symptoms. Processed foods, including sweets and fast foods, are associated with increased risk of depression in adolescents. Moreover, consumption of sweets has been shown to be positively correlated with depressive symptoms.

When it comes to healthy sources of fats, polyunsaturated fatty acids (PUFAs) and omega-3 fatty acids have both been observed to be significantly lower in depressed patients than in nondepressed patients. Olive oil and fish may provide the essential PUFAs and omega-3s individuals with depression often lack. These are both common in the Mediterranean diet, which has been found in studies to have protective effects against depression. This diet is comprised largely of whole-foods and plants. Core tenets of the Mediterranean diet may be beneficial to some; studies negatively correlate the Mediterranean eating style and depressive symptoms. When it comes to diet, poor diets are frequently reported by depressed patients and prospective epidemiological studies have shown links between diet patterns and depressive symptoms. Processed foods, including sweets and fast foods, are associated with increased risk of depression in adolescents.

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But how might food affect mood? Metabolism of neurotransmitters is one speculation. It is known that depressed patients often do not process serotonin and dopamine in the same way as nondepressed individuals. Selective serotonin reuptake inhibitors (SSRIs) are often prescribed, in theory, to correct these imbalances, but there may be a link between neurotransmitters and diet. Animal studies have shown that carbohydrate consumption at either extreme can harshly impact the rate at which serotonin is turned over in the brain, and such fluctuations can negatively impact mood. L-tryptophan is a precursor to serotonin and is directly linked to diet. Starvation may deplete the brain of means to build these important mood neurotransmitters, and diets too rich with excessive sugar may overwhelm turnover. It has even been observed that consumption of dietary fat and sugar
in excess can reduce dopamine D2 receptor sensitivity (an important receptor of the brain’s reward pathway), and diets too high in fat have been shown to alter dopamine gene expression. Eliminating processed foods may reduce sources of these potentially harmful substances and improve depressive symptoms. While more research needs to be done, it is evident that an appropriately balanced diet may be beneficial to those suffering from depression.

**Alzheimer’s Disease** is a degenerative form of dementia, which worsens as it progresses. Today it affects an estimated 1 in 85 people worldwide, and the scientific community is devoting a great deal of effort to finding the etiology and effective treatments. Oxidative stress has been considered as a cause of worsening symptoms, and some researchers are looking to diet in hopes of reducing or minimizing these effects. While it has been documented that antioxidant therapy reduced oxidative modifications in mice, long-term antioxidant therapy within tolerable guidelines may have limited benefits.

The Alzheimer’s Association suggests that high intake of saturated fat and imbalanced cholesterol levels increase risk for Alzheimer’s disease. Higher LDL cholesterol, lower HDL values, and diets high in saturated fat and have been speculated to increase risk for neuron dysfunction that could hasten the progression of dementia. These unhealthy patterns often start early in life, during childhood.

Several studies show that those following a Mediterranean diet have a lower risk of developing Alzheimer’s disease and improved prognosis by lowering the likelihood of cognitive decline. Consumption of omega-3 fatty acids is speculated by some to be protective against risk. Additionally, those following a diet high in simple carbohydrates (such as refined sugar) and saturated fat have an increased risk.

Other studies are looking to a few trace nutrients, specifically vitamin B12 and folate, as risk factors for development of Alzheimer’s disease. In one study, researchers found that low serum levels of vitamin B12 and folate more than doubled the risk of developing Alzheimer’s disease, and associations were even larger in participants with good baseline cognition.

**Nutrition and Child and Adolescent Psychiatry**

A nutritious diet and appropriate exercise are important to overall health, and encouraging awareness is well within the purview of the child and adolescent psychiatrist. Asking about basic diet, nutrition, and exercise is therefore an important component of an intake evaluation. However, some families may be sensitive to these questions due to cultural and social differences. Thus, it is important to start with open-ended questions and avoid seeming judgmental. Specific questions for youth include: “tell me about your favorite foods” and “which foods do you like so much you eat them several times a week?” These early questions can evolve to more elaborate, diagnosis-specific discussions over time and segue into opportunities to improve health through nutrition.

**Conclusion**

As researchers try to better understand cause and prognosis of complex mental illnesses including depression, ADHD, ASD, and Alzheimer’s disease, the role of nutrition is receiving more attention as an influential risk or protective factor. Although it is accepted that diet is indeed an important component of mental health, at this time little is deeply understood about the extent of impact of nutrition on mental illnesses. Current information is limited but deserving of more clinical attention and research in the future. At the end of the day, we all must eat, and we know that what we put in our bodies matters. It is becoming increasingly evident that nutrition plays a role in nourishing not only our physiological wellbeing, but our psychological wellbeing, as well.

**References:**


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Katherine Baker, BS, is a graduate of New York University with a bachelor's degree in psychology. She plans to pursue a Master's Degree in clinical nutrition before attending medical school for psychiatry, and is very interested in the role of nutrition in both physical and mental health.

Sara Weekly, MD, is a Clinical Assistant Professor of Psychiatry at the State University of New York Downstate where she works as an emergency psychiatrist. She is also a clinical instructor with the New York University Department of Child and Adolescent Psychiatry, and she leads an advanced seminar in eating disorders for New York University undergraduate students. She has a passion for learning and teaching about both healthful and disordered eating, as well as the ways in which nutrition interacts with mental illness.

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Severe psychotic illnesses like schizophrenia produce significant psychosocial and economic disability, particularly when they first present during youth. In the United States, it is estimated that the total cost of schizophrenia is over 60 billion dollars, which includes both direct costs from patient care and housing and indirect costs from unemployment, reduced workplace productivity, and premature mortality. As with many psychiatric disorders, where treatment begins after the onset of illness, psychotic disorders are often treated after the first psychotic episode. However, there has been increasing neurobiological and clinical research indicating the existence of a prodromal period prior to the first psychotic episode, which could be detected by clinical monitoring.

**The Prodrome**

It is estimated that some psychotic symptoms are present in 7.5% of adolescents and 5% of the general adult population, which may represent a subgroup at genetic risk of psychosis. A large majority of individuals with psychotic illnesses display subthreshold or “prodromal” signs of psychosis during adolescence and transition to adulthood.

The “prodromal period” is the time during which a disease process has begun but has not yet clinically manifested with sufficient severity or compromise that it meets criteria for full-blown illness. In the case of psychotic disorders, this is often described as a prolonged period of attenuated and nonspecific thought, mood, and perceptual disturbances accompanied by poor psychosocial functioning, which are typically identified retrospectively. Clinical high-risk (CHR), at-risk mental state (ARMS), or ultra-high-risk (UHR) are terms to identify prospectively individuals who are potentially in the prodromal phase of psychosis. The DSM has included attenuated psychosis syndrome as a category for further study in section 3.

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**Table 1: Examples of Symptoms During the Prodromal Period**

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive prodromal thought content</td>
<td>Perplexity, delusional mood, non-persecutory ideas of reference, over-valued beliefs, distrustful, exaggerated</td>
</tr>
<tr>
<td>Negative prodromal thought content</td>
<td>Social isolation or withdrawal, avolition, diminished emotional responsiveness,</td>
</tr>
<tr>
<td>Disorganization symptoms</td>
<td>Odd behavior, mannerisms, poor focus and attention, social inattentiveness,</td>
</tr>
<tr>
<td>Other symptoms</td>
<td>Sleep disturbances, dysphoric mood,</td>
</tr>
</tbody>
</table>

Note: Adapted from Structured Interview for Prodromal Symptoms (SIPS).
reflects signs and symptoms that increase risk for transition to clinical psychosis.

**Assessment Tools**
The Comprehensive Assessment of At-Risk Mental States (CAARMS) and Structural Interview for Prodromal Symptoms (SIPS) are tools used to identify and classify the ARMS or UHR as falling under 1) Attenuated Psychotic Symptoms (APS) group, which describes subthreshold attenuated positive psychotic symptoms in the last 12 months; 2) Brief Limited Intermittent Psychotic Symptoms (BLIPS) group, consisting of psychotic symptoms lasting for less than one week and spontaneously remitted; or 3) Trait and State Risk Factor Group, describing an individual with a first degree relative who has a psychotic disorder or schizotypal personality disorder and who has experienced psychosocial decline over the last one year.7, 12 The Prodromal Questionnaire is another frequently used screening tool.13 Of these tools, CAARMS and SIPS require specialized training, whereas the Prodromal Questionnaire-Brief Version is available freely.

**Psychotherapeutic Interventions**
Meta-analysis of five cognitive-behavioral therapy (CBT) trials reported that CBT reduced the risk of transition to psychosis at the end point by 48% when compared to controls (pooled risk ratio = 0.52, 95% confidence interval 0.79).9 A study examining the effects of an integrated psychological intervention, combining individual CBT, group skills training, cognitive remediation, and multifamily psychoeducation versus supportive counseling showed that the integrative psychological intervention was superior to supportive counseling in preventing progression to psychosis at 12 month follow-up (3.2% vs. 16.9%; p=.008) and at 24 month follow up (6.3% vs 20.0%; p=.019).14

**Psychopharmacological Interventions**
There have been a few trials evaluating the efficacy of antipsychotic agents in preventing transition to a psychotic episode. Transition rates at 12 months in a study comparing CBT and risperidone, CBT and placebo, and supportive therapy and placebo, were 10.7%, 9.6%, and 21.8%, respectively, and the differences were not statistically significant. The dropout rate was 37% for the risperidone arm.15 Another double-blind, placebo-controlled study did not find statistical differences in one-year conversion rates between olanzapine (5-15 mg/day) and placebo. The participants on olanzapine gained a statistically significant amount of weight compared to placebo, with an average weight gain of 8.7kg over 12 months (SD=9.05). The dropout rate was 54% for the olanzapine arm, and 34% for the placebo arm.16 Open label trials with amisulpride and aripiprazole have shown some efficacy but placebo-controlled trials are needed.17, 18 Long-chain omega-3 fatty acids have been shown to be superior to placebo in a 12-week randomized control trial. The trial needs to be replicated.19

**Conclusion**
Youth who present with prodromal symptoms (as listed in Table 1) may be at increased risk for developing clinical psychosis, which can be a chronic, debilitating condition. Clinicians should be diligent for screening for prodromal symptoms by monitoring for evolving symptoms on a regular basis (see Box 2 for practical tips). Several screening measures exist to aid clinicians. Preventative efforts are under investigation. Currently, psychotherapeutic modalities and some pharmacological interventions hold promise.

Furthermore, early detection and intervention in people at risk of developing psychosis can be successful to pre-
vent or delay a first psychotic episode. Additional research is needed to determine the benefit of antipsychotics at this stage, as current studies are limited by high drop-out rates.

**Other Resources:**
International Early Psychosis Association (IEPA)  
http://iepa.org.au/

North American Prodromal Longitudinal study (NAPLS)  
http://napls.psych.ucla.edu/

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**References**


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**About the Authors**

Tushita Mayanil, MD, pursued the Substance Abuse and Mental Health Administration (SAMHSA) – American Academy of Child and Adolescent Psychiatry (AACAP) Systems of Care Fellowship during the second year of her child and adolescent fellowship at the Children’s National Medical Center, Washington, DC. She worked collaboratively with the staff at SAMHSA and AACAP to envision and explore a community-based systems-of-care approach towards evidence-based early identification and possible intervention for individuals at risk for psychosis.

Steve Adelsheim, MD, is a Clinical Professor in Psychiatry at the Stanford University School of Medicine, Palo Alto, CA. Dr. Adelsheim has partnered in developing statewide mental health policy and community systems, including those focused on school mental health, telebehavioral health, early intervention for psychosis programs, and suicide prevention. He is the recipient of many awards including the 2012 AACAP Sidney Berman Award for School-Based Study and Treatment of Learning Disorders and Mental Illness and the 2006 APA Agnes Purcell McGavin Award for Prevention.

Gary M. Blau, PhD, is a Clinical Psychologist and is currently the Chief of the Child, Adolescent and Family Branch of the Center for Mental Health Services at the Substance Abuse and Mental Health Services Administration (SAMHSA), Rockville, MD. In this role he provides national leadership for children's mental health policy and block grant funding and promotes the “systems of care” across the country. He has published widely and has received many state and national level awards including the 2009 HHS Secretary’s Award for Meritorious Service for his national leadership in children’s mental health.

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**Take-Home Summary:**
Evidence is lacking on how to treat prodromal symptoms, although psychopharmacological and psychotherapeutic interventions are showing some preliminary potential benefits. More research is needed before these can be recommended to prevent the onset of psychosis.
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You are not alone in coming to the realization that writing is hard work. Don’t believe anyone who says otherwise, but also don’t let it dissuade you from giving it a shot. You may not know it, and someone early in his or her career may not believe it, but the fact is that someone wants to read your work, and there is a home for it. Only one thing is for certain: if you don’t get started and give it a real try, then your work will never be published. Writing is hard work, but it is also a skill you can learn and one that gets better with practice and time. I hope to persuade you that there are steps you can take to make the process more efficient, more hopeful, more likely to succeed, and, if nothing else, more bearable.

Have a Good Story to Tell
Whether you are writing up completed research, sharing an interesting clinical observation, or reviewing a book for the first time, there is no substitute for having good content and a compelling story. But what makes a story worth reading? As it pertains to the scientific literature, there are four main considerations: Is it new, true, clear, and ethical?

Producing something truly new may seem foreboding. Novelty may lie in a fresh hypothesis or a singular idea, but keep in mind that the novelty of a manuscript may also be that it is a good replication: nothing to be ashamed of there – science is based on the reproducibility of findings. How a submission is new will vary, but what makes the novelty important, as opposed to merely novel, is that it must pass the ultimate litmus test: it must push us forward.

In science, much of what is true comes from models (statistical models, models based on population samples, etc.), which are always approximations of truth, and are best at relegating untrue ideas to the dustbin. One model for truth in science is “validity,” both internal (Do the methods hold up and are they able to do what they claim?), and external (Do findings apply outside of the sample they are based on? Are they generalizable?). But even if you are not conducting a study where conventional models of validity are applicable, you can still ask a similar question: How do you know that what you are saying is true and that it is true outside of the story you are telling? This can be a matter of the integrity of your citations (How well are you basing your claims on empirical evidence, and how strong are the empirical claims themselves?), or of understanding the limits of your story (as with, for example, a case study).

Clarity is straightforward: Can your story, no matter how good, be conveyed in a manner that will be understood by your reader? Strive to be straightforward, simple, and direct; don’t “eschew obfuscation” when you can “avoid confusion” instead. Become a peer reviewer: editors will get to know you, and you will become a better critic of your own work. As a better critic, you will preempt and beat others to finding the weaknesses in your manuscripts and correcting them. Indeed, explicitly pointing out the limitations of your study will only strengthen your submission, and few things will prepare you for the task as well as serving as a reviewer.

Ethical principles are important enough to deserve separate mention.

Always Adhere to Ethical Principles
Ethical principles are not cautionary tales intended for unsavory and disreputable characters, and, as such, easily dismissed. They are core values that we must adhere to at all times, but which can be easily overlooked and infringed upon. Prevention is worth more than cure, which is why institutional review boards (IRBs) are so important. Make sure that you are well versed in the ethical principles of research and publication. You are responsible for knowing, understanding, and abiding by these standards. There are far too many aspects and nuances to numerate and attend to here, but they are practically summarized elsewhere (see resources listed below). A few core principles are key: treat patients and family as you would like to be treated (this includes using thoughtful and sensitive language when referring to them. Always use person-first language: ‘the child with autism,’ rather than ‘the autistic child’); don’t do or say anything that feels uncomfortable and that you cannot own up to with
pride; be mindful of potential conflicts of interest (and not only financial ones); don’t copy without attribution (even from your own past work); and attend to authorship order and attribution early on.

**Never Worry Alone**
Academic writing is rarely a solo act. It is usually a fruitful collaboration. If you are not part of a team, identify one that suits your needs. This could mean becoming part of a larger research group and taking the lead in writing a specific project, or writing something on your own and asking for input from a respected colleague. When approaching someone to look over your work, always “play up” – ask for the advice of someone more senior or experienced than you, someone who will take the time to engage with you and your writing and critique it thoughtfully, and someone who will help push your work forward and not just rubberstamp it. Ask for help, and not just in areas where you consider yourself inadequately prepared (statistics is a common weakness), but also where you may already feel confident. Writing in collaboration or under close supervision is where the mentorship rubber meets the road. Embrace the challenge posed by your mentor’s red ink.

**Think Like an Editor**
Most early rejections are based on a summary assessment of an article’s abstract. With some luck, the initial evaluation gets as far as the Method and Results. At least early in the peer review process, it is rare for an editor to spend much time going over the Introduction or Discussion sections. And yet, most authors spend the bulk of their writing time refining and making their arguments in these areas. The practical corollary is simple: don’t let your abstract be an afterthought. From the beginning, pay close attention to the 250 words of your abstract, polish them as you go along, and make sure they do justice to the overall article. An effective abstract should open the door for your paper to be sent out for peer review. It is your ‘elevator pitch,’ and with so much of your submission’s fate riding on it, it cannot be overlooked. You should not underestimate the inherent difficulty in writing so succinctly. Blaise Pascal alerted us to the challenge back in 1656: “I would have written a shorter letter, but I did not have the time.”

**Present Your Work in an Engaging Way**
Scholarly writing need not be sleep-inducing. This statement is not intended to encourage frivolous prose or cute-sy affectations. Rather, it is an invitation to be proactive, direct, and bold in approaching academic journals.

For starters, make good use of your salutation. Cover letters are generally underappreciated or overlooked altogether – a bureaucratic box to be checked – but prospective authors should not pass up the opportunity to make a good first impression. A brief letter that makes the case for your submission belonging in this particular journal can go a long way. If a submission is not a periodical’s ‘usual fare,’ consider approaching the editor in advance and making an inquiry – or a pitch. Editors are human, and as such, approachable. You may find that your idea is in fact of interest and welcome, or you may be steered in a more promising direction and avoid wasted effort.

Don’t irritate the editor or dismiss the obvious: read the instructions for authors and prepare your submission accordingly. When in doubt, ask the editorial office for clarification. Simple oversights and typos may be inconsequential to the overall science in question, but superficial sloppiness raises concerns about a study’s underlying integrity and attention to detail. Missing pieces or overlooked requirements can delay processing of your manuscript. Be wary of spell-check and rely instead on a careful read by a keen-eyed colleague (or three) before pressing the ‘submit’ button. Don’t let simple formalities doom your hard-earned efforts.

**Do Not Despair if English Is Not Your Native Language**
Practically speaking, English has become the universal language of scientific writing. This should not dissuade would-be authors from contributing to periodicals in their native languages, nor pose a significant hurdle to most speakers of English as a second language. The charge is not to write great prose so much as to write understandably and clearly. Can a reader outside of your specific field of interest understand what you are trying to convey? Is there a fluent English speaker on your team, or someone else you can approach to go over your draft? There are a number of professional ‘polishing’ services available online to improve the written quality of scientific submissions. These may be a useful resource, but one that should be used sparingly, and perhaps are most helpful late in the submission process, after the scientific bar has been cleared.
Learn From Rejections – Especially Quick Ones
Rejection is an inevitable byproduct of the submission process. Having no rejections is no badge of honor. In fact, having few rejections is likely to mean one of two things (neither one to be emulated): submitting insufficiently or submitting to less desirable outlets.

If you receive a decision letter inviting you to revise and resubmit, celebrate the occasion, for you are well on your way. There is an art to responding to reviewers’ comments, and since you are already halfway to publication, you don’t want to miss out on the opportunity by responding inadequately. Most comments from peer reviewers are likely to be helpful, and if properly incorporated, will strengthen your work. Responding to a reviewer does not mean that you always have to concur; you can respectfully disagree with a given point, so long as you can make an objective case to justify your decision. Be polite, civil, and transparent. Don’t become argumentative or defensive – stick to substance and facts.

If your manuscript is rejected following peer review, resist the temptation to resubmit the paper, unchanged, to another journal. It is all too easy to do so in the electronic age, but you are missing out on an opportunity for growth. Your initial impulse may be to argue with the decision, to state that your work was not properly understood or appreciated. Even if that is the case, it is still best to give yourself some distance and a few days before going back to the decision letter, at which time you can start objectively considering the feedback and incorporating pertinent aspects of it to improve your work before moving on to the next journal. Bear in mind that ours is a relatively small field and your manuscript may end up being assessed by the same reviewer at another journal. For this reason, and for the opportunity to improve on your submission, don’t dismiss potentially valuable feedback just because it is part of a rejection letter.

A quick rejection letter should also be welcome: an indication that you are engaged in the process and looking for the right venue for your submission. When a manuscript is rejected outright, before being sent out for peer review, the reason is likely one of fit rather than content. We are fortunate to have thousands of medical journals available, you can narrow your list down to relevant journals based on topic and scope, and ranked according to impact factor (IF), turnaround time, and other metrics that may be relevant to your goals. Senior colleagues can then help you refine that list, especially if they are experienced and successful in their own submission efforts. As a rule of thumb, try to submit slightly ‘above’ your target: you may gain access to a more visible or prestigious venue, or come away with useful feedback to incorporate. It is true that aiming for the sure target may yield fewer rejection slips, but it will not challenge and advance your efforts sufficiently.

Balance Ambition With Realism
IF and similar metrics should not be the main guiding principle in deciding where to submit your work. Determine your optimal target readership and pitch accordingly. This may lead to a more modest publication, but one better suited to your goals. Think globally and set high expectations for your work, but don’t dismiss more proximal venues that may be more appropriate. Regional publications may not have the cachet you had hoped for but may be better conduits to put the information into the hands of those who can most benefit from it. Particularly if you are early in your career trajectory, it is important to determine how long your curriculum, your promotion, or you yourself can wait to be published. A modest publication early in your career can prove pivotal, insofar as it can imbue you with a sense of possibility: seeing your name in print can validate your efforts and spur you on to the next level.

Never Take Rejection Personally
As authors, we place our work – and a certain amount of personal vulnerability – in the hands of an editor and the ‘black box’ of the editorial process. At some fundamental level, it is hard not to take rejection personally. And yet soldier on we must. Good editorial practice aims for objective and supportive feedback with no personal attacks or ad hominem comments slipping through to decision letters. That having been said, as an editor, I have been humbled on more than one occasion when an author has brought to my attention comments that did not adhere to these guidelines. As a result, I have redoubled my efforts to ensure editorial etiquette and minimize these unfortunate occurrences. In the process I gained respect for those authors and their direct, constructive, and proactive approach to the values of the peer review process itself.

There Are Alternative Doors Into a Journal
Keep writing: writing begets writing. Not every submission failure is rejected outright, before being sent out for peer review, the reason is likely one of fit rather than content. We are fortunate to have thousands of medical journals available, you can narrow your list down to relevant journals based on topic and scope, and ranked according to impact factor (IF), turnaround time, and other metrics that may be relevant to your goals. Senior colleagues can then help you refine that list, especially if they are experienced and successful in their own submission efforts. As a rule of thumb, try to submit slightly ‘above’ your target: you may gain access to a more visible or prestigious venue, or come away with useful feedback to incorporate. It is true that aiming for the sure target may yield fewer rejection slips, but it will not challenge and advance your efforts sufficiently.

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There Are Alternative Doors Into a Journal
Keep writing: writing begets writing. Not every submis-
tion needs to fit the mold of a classic scientific paper. In fact, there are often many pathways into a journal, some of which are especially relevant to novice authors. Letters to the editor can give voice to a new observation or engage with the substance of an earlier publication in a thoughtful way. Book reviews distill the essence of a new resource and place it into a larger intellectual context. There may be venues like this, JAACAP Connect, where you can hone ideas and writing skills, as well as clinical essays (such as those in JAACAP's Clinical Perspectives), and sometimes editorials and reviews. If you have read this far, this piece serves to exemplify that writing for a newsletter or bulletin can serve an educational function – and help its author remain limber in the writing domain. And do not forget that becoming a peer reviewer not only allows you to participate in one of the most important facets in scientific publications and to practice critical skills, but introduces you to journal editors, journal styles, and often excellent writing.

Just Do It!
Life is filled with compelling reasons to procrastinate. I know of what I speak: guilty as charged. Seek to be polished, not perfect. The perfect is the enemy of the good. You have a story to tell and someone out there wants to hear it. So get started and down to business. Roll up your sleeves. Just do it!

Selected Resources for Would-Be Authors, Reviewers, and Editors
1. This five-part series of short, practical articles published in the Archives of Pediatrics and Adolescent Medicine covers the critical aspects necessary to get a scholarly article into print.
2. Resources addressing ethical aspects of scholarly publication.
3. General resources.
4. For would-be editors.

About the Author:
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