AUTISM:
THEORY OF MIND

Mary ET Boyle, Ph.D.
Department of Cognitive Science
UCSD
Autism is...

Defined by behavioral criteria

Some biological markers

Clinical presentation is varied:

education, temperament, ability

dynamic changes over development

Co-morbid with other disorders
Autism is a developmental disorder characterized by impaired social interaction and communication as well as repetitive behaviours and restricted interests. The consequences of this disorder for everyday life adaptation are extremely variable. The general public is now more aware of the high prevalence of this lifelong disorder, with ca. 0.6% of the population being affected. However, the signs and symptoms of autism are still puzzling. Since a biological basis of autism was accepted, approaches from developmental cognitive neuroscience have been applied to further our understanding of the autism spectrum. The study of the behavioural and underlying cognitive deficits in autism has advanced ahead of the study of the underlying brain abnormalities and of the putative genetic mechanisms. However, advances in these fields are expected as methodological difficulties are overcome. In this paper, recent developments in the field of autism are outlined. In particular, we review the findings of the three main neuro-cognitive theories of autism: theory-of-mind deficit, weak central coherence and executive dysfunction.
**Autism:**
Is the most severe childhood neuropsychiatric condition diagnosed today.

**Abnormalities**
- Speech and communication
- Social functioning
- Imagination

**Behaviors**
- Repetitive
- Restricted interests
- Complex behavioral disability

**Demographics**
- M:F ratio: 4:1
- 0.6% population
- Appears during the first three years of life

American Psychiatric Association, *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.),
<table>
<thead>
<tr>
<th>PDD-Not Otherwise Specified (PDD-NOS)</th>
<th>Rett's Disorder</th>
<th>Childhood Disintegrative Disorder (CDD)</th>
<th>Asperger's Syndrome</th>
<th>Autistic Disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal neurological syndrome seen in girls. Arrest of normal brain development that occurs during infancy.</td>
<td>Normal intellectual and social development then 2-10 years show severe regression into autism.</td>
<td>Deficient or absent social interactions</td>
<td>Less severe</td>
<td>No language delay</td>
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</table>
“...many critics overlook a surprising fact about the new D.S.M.: how little attention practicing psychiatrists will give to it.

There are dozens of revisions in the D.S.M. — among them, the elimination of a “bereavement exclusion” from major depressive disorder and the creation of binge eating disorder — but they won’t alter clinical practice much, if at all. 

This is because psychiatrists tend to treat according to symptoms.”
“This is a problem because the D.S.M. is an imperfect guide to predicting what treatments will benefit patients most — a reality tied to the fact that psychiatric diagnoses are based on clinical appearances that tend to cluster, not on the mechanism behind the illness, as is the case with, say, bacterial pneumonia.”
So, after being pushed back, the DSM-5 came out in May. Prolly the biggest change (to me) was the term "Asperger's", along with other High Functioning labels, disappearing and replaced with "Autism Spectrum Disorder".

At first... I thought it was a terrible idea, cause I thought some folks might get mis-diagnosed if there's no "high functioning" term.

Then, I actually had a good look, and I saw the main point they made, and I hadta agree: The only difference in the diagnosis between HFA and Autism is that the criteria for Autism has an extra section that folks diagnosed with Asperger's should prolly still be able to meet.

So, good idea! Specially since now, mebbe the media can't shout "OMG, Asperger's!" every time there's a mass shooting.

The biggest issue was in the pdf form, where they decided to destroy our retinas by having all this info in white lettering on a bright blue background.
Purpose: To evaluate prevalence rates of autism and autism symptomatology in toddlers using DSM-IV vs DSM-5 criteria.

Method: Two thousand seven hundred and twenty-one toddlers at risk for a developmental disability participated. DSM-IV and DSM-5 criteria were applied and overall prevalence using each set of criteria was established. Groups were also compared on BISCUIT-Part 1 scores to determine if groups differed on autism symptomatology.

Results: DSM-5 resulted in 47.79% fewer toddlers being diagnosed with ASD compared to those on the DSM-IV. Toddlers diagnosed according to DSM-5 exhibited greater levels of autism symptomatology than those diagnosed with DSM-IV, but the latter group still exhibited significant levels of autism symptomatology.

Conclusion: The proposed DSM-5 will result in far fewer persons being diagnosed with ASD. These results replicate findings from two previous studies, with older children/adolescents and adults. As a result of these new criteria, far fewer people will qualify for needed autism services.

Characteristics of Autism

“Brent spent countless hours learning the meaning of ambiguous words and phrases. His Mother, co-author, Linda Gund Anderson, recognized that Brent’s language challenges were not unique and the idea for Unintentional Humor was born.”
Characteristics of Autism

**activities**
- sustained odd play
- spins objects or self
- noticeable extremes: over or under activity

**emotions**
- no real fear of danger
- pain extremes: over or under
- unresponsive to normal teaching methods

**motor skills**
- uneven gross/fine skills
- no cuddling
- little or no eye contact
Language Development

delayed & deviant

Peculiar use of sounds and words

echolalia
## Social Development

| Physical and emotional distance from others. | Failure to develop social attachments  
Difficulties in reacting to or recognizing other people’s feelings. | Lack of cooperative group play |
|--------------------------------------------|-------------------------------------------------------------------------------------------------|--------------------------------|

## Intellectual Development

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<th>Poor on verbal ability</th>
<th>May perform above average on memory or spatial tasks</th>
<th>May be talented in music or drawing</th>
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<tbody>
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<td>25-35% have IQ &gt; 70</td>
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## Diagnosing Autism

<table>
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<th>New tests:</th>
<th>5 behaviors:</th>
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</thead>
<tbody>
<tr>
<td>Childhood Autism rating scale (CARS)</td>
<td>• babbling (1 yr)</td>
</tr>
<tr>
<td>Questionnaire</td>
<td>• gesturing (1 yr)</td>
</tr>
<tr>
<td>Two year old screening</td>
<td>• Single words (16 mo)</td>
</tr>
<tr>
<td></td>
<td>• Two-word phrases (24 mo)</td>
</tr>
<tr>
<td></td>
<td>• Any loss of social skill (any age)</td>
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</table>
The savant is an individual with an islet of outstanding skill in one area, which can include calendar calculation, musical or artistic competence, often in the presence of modest or even low general intellectual ability.

Common reports of sensory abnormalities, which suggest heightened sensitivity to minute differences between stimuli, be they in sound, sight, taste or touch.
The term ‘autism’ is used to describe all individuals on the autistic spectrum.

- Behavioral findings are based on high-functioning individuals.
- Anatomical studies of the brain in autism are based on low-functioning individuals.
1st described

- Leo Kanner (1943)
- Hans Asperger (1944)

Explanation:

- “Refrigerator mother”
- Genetic
- Environment
10% of all cases of autism have definable biological causes – e.g. Rubella, prenatal thalidomide and encephalitis. Interference with a particular stage of prenatal development can cause autism.

Brain pathology


Herbert et al. (2004) show white matter abnormalities in short-range v. long range axons.
Soma

no myelin

<time

3.5 msec

Axon

Hebbian Learning

Saltatory Conduction

Learn
The most consistent finding about the autistic brain to have emerged in recent years is that it is on average larger and heavier than the normal brain.
Autistic brain is, on average slightly smaller at birth, it begins to grow abnormally quickly, and by two to three years of age it is about 10 percent larger than a normal brain.

“Importantly, the increased size is not evident from birth, but from ca. 2–4 years.

A reason for this increase could be a failure of the normal pruning process that occurs several times during development after an initial wave of proliferation of synapses.”

Courchesne et al. 2001
Differential growth pattern:

- The frontal cortex and temporal cortex of the autistic brain grow quickly during the first two years of life but then show little or not increase in size during the next four years.
- The amygdala has an abnormal growth pattern:
  - At 4 years of age – it is larger
  - At adulthood – it is normal size – BUT fewer neurons

Growth pattern of lower order regions of the cerebral cortex – primary visual cortex and extra-striate cortex are relatively normal in the autistic brain.
White matter problems, too.

Volume of white matter containing short-range axons was increased.

Volume of white matter containing long-range axons lower.

Social cognition

Empathy

The ability to understand each other as conscious beings with internal mental states

“Feeling the feelings” of others

Theory of Mind

The metacognitive understandings of our own minds as well as the minds of others
## Social Cognition Terms

<table>
<thead>
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<th>Mentalizing</th>
<th>Mind Reading</th>
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<td>- We understand ourselves and others not just as sensory objects but also as subjective beings with mental states</td>
<td>- Identifies our ability to attune our own behaviors to the minds and anticipated actions of others</td>
</tr>
</tbody>
</table>
Theory of Mind deficit: A fault in one component of the social brain can lead to the inability to understand certain basic aspects of communication.

Autism? mindblindness or mentalizing failure
Children were shown that Sally had a basket and Ann a box.

Sally puts a marble in her basket and goes outside.

While she is outside, naughty Ann moves Sally’s marble to her own basket.

Sally then comes back in and wants to play with her marble.

Children were asked, ‘where will Sally look for her marble?’
To a normally developing 4-year-old child, the answer is clear: **Sally will look for her marble where she thinks it is and not where it really is now.** Furthermore, the normally developing child can reason that Sally will look in her basket because this is where she put it and she does not know that it has been moved.

80% of children with autism, with a mental age equivalent to a 4 year-old or above, failed to answer this question correctly.

Individuals with autism do not activate the face area of the fusiform gyrus that is reliably activated by normal individuals when looking at faces as opposed to objects.
Brain imaging studies consistently find that the fusiform region of the temporal lobe becomes active when people look at faces.

The patient sits on the bed, his head wrapped in thick gauze bandages. He looks his doctor in the eye and says, “You just turned into somebody else... You almost look like somebody I’ve seen before, but somebody different. That was a trip.”
This study compared individuals with autism with normal individuals on mentalizing tasks while their brains were being imaged.
The goal: to judge a person’s emotional states from photographs of the eye region.

The subject must decide which two words best describe the mental state of the individual in the photograph.

The results: individuals with autism, in contrast to the control group, showed less extensive activation in frontal regions and no activation in the amygdala.
fMRI activation of the fusiform gyrus and amygdala to cartoon characters but not to faces in a boy with autism

David J. Grelotti\textsuperscript{a,1}, Ami J. Klin\textsuperscript{a}, Isabel Gauthier\textsuperscript{b}, Pawel Skudlarski\textsuperscript{c}, Donald J. Cohen\textsuperscript{a,2}, John C. Gore\textsuperscript{d}, Fred R. Volkmar\textsuperscript{a}, Robert T. Schultz\textsuperscript{a,c,*}

\textsuperscript{a} Child Study Center, Yale University School of Medicine, P.O. Box 207900, New Haven, CT 06520-7900, USA
\textsuperscript{b} Department of Psychology, Vanderbilt University, Wilson Hall, Nashville, TN 37203, USA
\textsuperscript{c} Department of Diagnostic Radiology, Yale University School of Medicine, Magnetic Resonance Research Center, 300 Cedar Street, New Haven, CT 06510, USA
\textsuperscript{d} Institute of Imaging Sciences, Vanderbilt University, Nashville, TN 37203, USA

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Evidence the U.S. autism epidemic initiated by acetaminophen (Tylenol) is aggravated by oral antibiotic amoxicillin/clavulanate (Augmentin) and now exponentially by herbicide glyphosate (Roundup)

Peter Good

Autism Studies, PO Box 1983, La Pine, OR 97739, USA

SUMMARY

Because certain hereditary diseases show autistic behavior, and autism often runs in families, researchers seek genes underlying the pathophysiology of autism, thus core behaviors. Other researchers argue environmental factors are decisive, citing compelling evidence of an autism epidemic in the United States beginning about 1980. Recognition that environmental factors influence gene expression led to synthesis of these views—an "epigenetic epidemic" provoked by pervasive environmental agents altering expression of vulnerable genes, inducing characteristic autistic biochemistries in many mothers and infants. Two toxins most implicated in the U.S. autism epidemic are analgesic/antipyretic acetaminophen (Tylenol) and oral antibiotic amoxicillin/clavulanate (Augmentin). Recently herbicide glyphosate (Roundup) was exponentially implicated. What do these toxins have in common? Acetaminophen depletes sulfate and glutathione required to detoxify it. Oral antibiotics kill and glyphosate inhibits intestinal bacteria that synthesize methionine (precursor of sulfate and glutathione, and required to methylate DNA), bacteria that synthesize tryptophan (sole precursor of neuro-inhibitor serotonin), and bacteria that restrain ammonia generating anaerobes. Sulfate plus glutathione normally stabilize fetal adrenal androgen dehydroepiandrosterone to DHEAS—major precursor of placental/postnatal estrogens. Glyphosate and heavy metals also inhibit aromatase that turns androgens to estrogens. Placental/postnatal estrogens dehydrate/mature brain myelic sheaths, mature corpus callosum and left hemisphere preferentially, dilute brain blood vessels, and elevate brain serotonin and oxytocin. Stress-induced weak androgens and estrogen depletion coherently explain white matter asymmetry and disconnection in autism, extreme male brain, low brain blood flow, hyperreactibility, social anxiety, and insufficient maternal oxytocin at birth to limit fetal brain chloride water and mature GABA.
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