The Autism Debate...

1st Global Congress for Consensus in Pediatrics & Child Health
February 17-20, 2011 Paris, France

Autism: The Emergence of a New Epidemic

Professor Virginia Wong
Division of Child Neurology/Development Paediatrics/NeuroHabilitation
Department of Paediatrics & Adolescent Medicine,
The University of Hong Kong
Autism Epidemic... or not?
INSIDE THE WORLD OF AUTISM

More than one million Americans may have it, and the number of new cases is exploding.
What scientists have discovered. What families should know.
Number of autism related articles published up to 2009

- **DSM III**
  - 1980

- **DSM IIIR**
  - 1987

- **DSM IV**
  - 1994

- **DSM IV-TR**
  - 2000

- **ADI-R**
  - 1987

- **ADOS**
  - 1984


0 200 400 600 800 1000 1200 1400

Year

Number of published articles
High Functioning Autistic Radio Boy
Dear Prof. Wong,

My wife and me are your students (Class 97'). We have a baby "Hei" who is 7 months old. We noticed he made little sound, and seldom vocalized. We believed his vision and hearing ability was normal. His motor development was within normal range. He would smile to familiar people, showed strange anxiety.

He would not look at you nor give any verbal response (e.g. sound; he only laugh at you) when we call his name. What we worry is whether something abnormal and we do not understand.

We may be too anxious, but is it possible to have an appointment with you for assessing our baby?

Thank you.

Yours sincerely,

Dr. Kong
Dr. Lam
7 months old Hei-Hei 2007.12.5
Asperger Baby ? (Hei Hei) 2008.1.4
Protoconversation between mother and infant

Syrhythmic regulation of mutual psychological engagement by exchange of expressions of interest and emotional feelings.

Eye contact

Joint Attention

01/03/2011 13:43 1st CIP Paris VW

Protoconversation between mother and infant

Synrhythmic regulation of mutual psychological engagement by exchange of expressions of interest and emotional feelings.

Joint Attention

No Eye contact
Early Warning Signs of Autism

The following list of symptoms represents the broad range of the disorder and is not meant to be a checklist to determine whether or not a child has ASD. Some children love hugs and are very affectionate; others are extremely touch sensitive to the point of it being painful. Some kids hit their developmental milestones, but they still seem to be “missing” something in the way they interact with others. Trust your gut instincts. If your child manifests several of these symptoms and your intuition suggests “something is just not right,” discuss your concerns with your pediatrician and ask for an evaluation. Get a second opinion if warranted. The earlier services are started with a child, the better is the opportunity for optimal positive learning and change to occur. Be proactive.

At 6 months
• Not making eye contact with parents during interaction.
• Not cooing or babbling.
• Not smiling when parents smile.
• Not participating in vocal turn-taking (baby makes a sound, adult makes a sound, and so forth).
• Not responding to peek-a-boo game.

At 12 months
• No attempts to speak.
• Not pointing, waving or grasping.
• No response when name is called.
• Indifferent to others.
• Repetitive body motions such as rocking or hand flapping.
• Fixation on a single object.
• Oversensitivity to textures, smells, sounds.
• Strong resistance to change in routine.
• Any loss of language.

At 24 months
• Does not initiate two-word phrases (that is, doesn’t just echo words).
• Any loss of words or developmental skill.

Source for Early Warning Signs: Dr. Rebecca Landa, Center for Autism and Related Disorders at the Kennedy Krieger Institute, Baltimore.
What is Autism?
It is an Umbrella diagnosis for many different biological processes with “Similar”
and yet Overlapping Phenotype

Prevalence = 1 in 150
Male to Female ratio: 4 :1
Autism Spectrum Disorder

Categorical vs Dimensional

A continuum of Disability, with quantitative differences in severity; probable categorical distinctions

Communication Impairment

Social Impairment

Restricted Interests & Compulsivity or RIBA

(Repetitive Interests, Behavior, Activities)

Autism

Asperger Disorder
Autism Prevalence
On The Rise*

There has been a 600% increase in prevalence over the last two decades.

*Recent research has indicated that changes in diagnostic practices may account for at least 25% of the increase in prevalence over time, however much of the increase is still unaccounted for and may be influenced by environmental factors.
Past & Present: Autism Prevalence Rate

(Estimation)

**Not Exhaustive Prevalence Studies**
Estimated prevalence of Autism Children = 37-60 /10000 for <20y

Estimated Number of Children = 2.2 billion (WHO 2009)

Estimated Number of Autism Children: 1,540,000 - 13,200,000
What Autism Epidemic?

By Claudia Wallis
Thursday, Jan. 11, 2007

Epidemic is a powerful word. It generates bold headlines, congressional hearings, research dollars and dramatic, high-stakes hunts for culprits. It's a word that has lately been attached to autism. How else to account for the fact that a disorder that before 1990 was reported to affect just 4.7 out of every 10,000 American children now strikes 60 per 10,000, according to many estimates—the equivalent of 1 in 166 kids?

But what if there is no epidemic? What if the apparent explosion in autism numbers is simply the unforeseen result of shifting definitions, policy changes and increased awareness among parents, educators and doctors? That's what George Washington University anthropologist Roy Richard Grinker persuasively argues in a new book sure to generate controversy. In Unstrange Minds: Remapping the World of Autism, Grinker uses the lens of anthropology to show how shifting cultural conditions change the way medical scientists do their work and how we perceive mental health.
In addition to rising awareness of autism, Grinker points to these factors:

**BROADER DEFINITIONS** Each successive edition of the Diagnostic and Statistical Manual of Mental Disorders--the bible of mental health--has revised the criteria for identifying autism in ways that tend to include more people. Two conditions on the milder end of the autistic spectrum--Asperger’s syndrome and the awkwardly named PDD-NOS (pervasive developmental disorder, not otherwise specified)--were added to the DSM in 1994 and 1987, respectively. Grinker and others say 50% to 75% of the increase in diagnoses is coming in these milder categories.

**SCHOOL POLICY** U.S. schools are required to report data on kids who receive special-education services, but autism wasn't added as a category until the 1991-92 school year. No wonder the numbers exploded--from 22,445 receiving services for autism in 1995 to 140,254 in 2004. Grinker points out that "traumatic brain injury" also became one of the 13 reportable categories in 1992, and it had a similar spike.

**MORE HELP, LESS STIGMA** As services have become more available for kids with autism, more parents are seeking a diagnosis they would have shunned 30 years ago, when psychiatrists still blamed autism on chilly "refrigerator" mothers. Doctors are also more willing to apply the diagnosis to help a patient. "I'll call a kid a zebra if it will get him the educational services I think he needs," National Institute of Mental Health psychiatrist Judith Rapoport told Grinker.
Diagnostic and Statistical Manual of Mental Disorders

Published by the American Psychiatry Association (APA)

Goal: To provide a common language and standard criteria for the classification of Mental Disorders
## Evolution of Diagnostic and Statistical Manual of Mental Disorders (DSM)

<table>
<thead>
<tr>
<th>Year</th>
<th>DSM Edition</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1943</td>
<td>DSM-I</td>
<td>Autism not included</td>
</tr>
<tr>
<td>1952</td>
<td>DSM-II</td>
<td>Autism not included</td>
</tr>
<tr>
<td>1968</td>
<td>DSM-II</td>
<td>Autism not included</td>
</tr>
<tr>
<td>1980</td>
<td>DSM-III</td>
<td>Autism first included</td>
</tr>
<tr>
<td>1987</td>
<td>DSM-III-Revised</td>
<td>&quot;Infantile Autism&quot; first included</td>
</tr>
<tr>
<td>1994</td>
<td>DSM-IV</td>
<td>Autism as a separate diagnostic category</td>
</tr>
<tr>
<td>1994</td>
<td>DSM-IV-TR</td>
<td>Further refinement of diagnostic criteria for AS and AD</td>
</tr>
<tr>
<td>2013</td>
<td>DSM-V</td>
<td>Expected release date</td>
</tr>
</tbody>
</table>

**Leo Kanner**, Psychiatrist at Johns Hopkins University

Published "Autistic Disturbances of Affective Contact" in *Journal of Nervous Child*, describing the behaviour of 11 children with "autism" from Greek word "auto" meaning "self".

**Hans Asperger**, Austrian Pediatrician

During WWII, conducted studies on exceptionally gifted, yet withdrawn children. He published the first definition of Asperger's Syndrome in 1944. Defined "autistic psychopathy" meaning autism (self) and psychopathy (personality disease).
Evolution of Diagnostic and Statistical Manual of Mental Disorders (DSM)

- **1943**: Leo Kanner, Psychiatrist at Johns Hopkins University
- **1944**: Hans Asperger, Austrian Pediatrician
- **1952**: DSM-I
  - Autism not included
- **1968**: DSM-II
  - Autism not included
- **1980**: DSM-III
  - "Infantile Autism" first included
- **1987**: DSM-III-Revised
  - "Autistic Disorder" was used.
- **1994**: DSM-IV
  - ASD represents 3 of the PDD
- **2000**: DSM-IV-TR (current edition)
  - Refinement of AS, AD and PDD-NOS
- **2013**: May 2013
  - Expected Release date of DSM-V
  - 01/03/2011 13:43
  - 1st CIP Paris VW
DSM-V Autism Spectrum Disorder

1. Clinically significant, persistent deficits in social communication and interactions, as manifest by all of the following:
   a. Marked deficits in nonverbal and verbal communication used for social interaction;
   b. Lack of social reciprocity;
   c. Failure to develop and maintain peer relationships appropriate to developmental level

2. Restricted, repetitive patterns of behavior, interests, and activities, as manifested by at least TWO of the following:
   a. Stereotyped motor or verbal behaviors, or unusual sensory behaviors
   b. Excessive adherence to routines and ritualized patterns of behavior
   c. Restricted, fixated interests

3. Symptoms must be present in early childhood (but may not become fully manifest until social demands exceed limited capacities)

http://www.dsm5.org/ProposedRevisions/Pages/Default.aspx
Autism Spectrum Disorder

Social Communication Impairment

Fixated Interests & Repetitive Behaviour
### 299.00

**Autistic Disorder**

<table>
<thead>
<tr>
<th>Proposed Revision</th>
<th>Rationale</th>
<th>Severity</th>
<th>DSM-IV</th>
</tr>
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</table>

New name for category, autism spectrum disorder, which includes autistic disorder (autism), Asperger's disorder, childhood disintegrative disorder, and pervasive developmental disorder not otherwise specified.

- Differentiation of autism spectrum disorder from typical development and other "nonspectrum" disorders is done reliably and with validity; while distinctions among disorders have been found to be inconsistent over time, variable across sites and often associated with severity, language level or intelligence rather than features of the disorder.

- Because autism is defined by a common set of behaviors, it is best represented as a single diagnostic category that is adapted to the individual's clinical presentation by inclusion of clinical specifiers (e.g., severity, verbal abilities and others) and associated features (e.g., known genetic disorders, epilepsy, intellectual disability and others.) A single spectrum disorder is a better reflection of the state of knowledge about pathology and clinical presentation; previously, the criteria were equivalent to trying to “cleave meatloaf at the joints”.

http://www.dsm5.org/ProposedRevisions/Pages/proposedrevision.aspx?rid=94#
UPDATED FROM (NEWEST INFORMATION JAN 26 2011 ***
Deficits in Social Communication/Interaction

Restricted, Repetitive Patterns of Behaviors, Interest or Activities

Symptoms Together must limit and Impair everyday functioning

Symptoms must be Present in early childhood

Autism Spectrum Disorder
Proposed Revision for DSM V (last updated Jan 26 2011)
Autistic Disorder

Must meet criteria A, B, C, and D:

A. Persistent deficits in social communication and social interaction across contexts, not accounted for by general developmental delays, and manifest by all 3 of the following:
   1. Deficits in social-emotional reciprocity, ranging from abnormal social approach and failure of normal back and forth conversation through reduced sharing of interests, emotions, and affect and response to total lack of initiation of social interaction.
   2. Deficits in nonverbal communicative behaviors used for social interaction; ranging from poorly integrated-verbal and nonverbal communication, through abnormalities in eye contact and body-language, or deficits in understanding and use of nonverbal communication, to total lack of facial expression or gestures.
   3. Deficits in developing and maintaining relationships, appropriate to developmental level (beyond those with caregivers), ranging from difficulties adjusting behavior to suit different social contexts through difficulties in sharing imaginative play and in making friends to an apparent absence of interest in people.

B. Restricted, repetitive patterns of behavior, interests, or activities as manifested by at least two of the following:
   1. Stereotyped or repetitive speech, motor movements, or use of objects: (such as simple motor stereotypies, echelalia, repetitive use of objects, or idiosyncratic phrases).
   2. Excessive adherence to routines, ritualized patterns of verbal or nonverbal behavior, or excessive resistance to change: (such as motoric rituals, insistence on same route or food, repetitive questioning or extreme distress at small changes).
   3. Highly restricted, fixated interests that are abnormal in intensity or focus: (such as strong attachment to or preoccupation with unusual objects, excessively circumscribed or perseverative interests).
   4. Hyper-or hypo-reactivity to sensory input or unusual interest in sensory aspects of environment: (such as apparent indifference to pain/heat/cold, adverse response to specific sounds or textures, excessive smelling or touching of objects, fascination with lights or spinning objects).

C. Symptoms must be present in early childhood (but may not become fully manifest until social demands exceed limited capacities)

D. Symptoms together limit and impair everyday functioning.
<table>
<thead>
<tr>
<th>Severity Level for ASD</th>
<th>Social Communication</th>
<th>Restricted interests &amp; repetitive behaviors</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Level 3</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>‘Requiring very substantial support’</td>
<td>Severe deficits in verbal and nonverbal social communication skills cause severe impairments in functioning, very limited initiation of social interactions and minimal response to social overtures from others.</td>
<td>Preoccupations, fixated rituals and/or repetitive behaviors markedly interfere with functioning in all spheres. Marked distress when rituals or routines are interrupted; very difficult to redirect from fixated interest or returns to it quickly.</td>
</tr>
<tr>
<td><strong>Level 2</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>‘Requiring substantial support’</td>
<td>Marked deficits in verbal and nonverbal social communication skills; social impairments apparent even with supports in place; limited initiation of social interactions and reduced or abnormal response to social overtures from others.</td>
<td>RRBs and/or preoccupations or fixated interests appear frequently enough to be obvious to the casual observer and interfere with functioning in a variety of contexts. Distress or frustration is apparent when RRB’s are interrupted; difficult to redirect from fixated interest.</td>
</tr>
<tr>
<td><strong>Level 1</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>‘Requiring support’</td>
<td>Without supports in place, deficits in social communication cause noticeable impairments. Has difficulty initiating social interactions and demonstrates clear examples of atypical or unsuccessful responses to social overtures of others. May appear to have decreased interest in social interactions.</td>
<td>Rituals and repetitive behaviors (RRB’s) cause significant interference with functioning in one or more contexts. Resists attempts by others to interrupt RRB’s or to be redirected from fixated interest.</td>
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In addition to rising awareness of autism, Grinker points to these factors:

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MORE HELP, LESS STIGMA As services have become more available for kids with autism, more parents are seeking a diagnosis they would have shunned 30 years ago, when psychiatrists still blamed autism on chilly "refrigerator" mothers. Doctors are also more willing to apply the diagnosis to help a patient. "I'll call a kid a zebra if it will get him the educational services I think he needs," National Institute of Mental Health psychiatrist Judith Rapoport told Grinker.
FINANCIAL INCENTIVES In some states, parents of children with autism can apply for Medicaid even if they are not near the poverty line. A diagnosis of mental retardation doesn’t always offer this advantage.

RELABELING For all the reasons above, many kids previously given other diagnoses are now called autistic. University of Wisconsin researcher Paul Shattuck has found that the number of kids getting special-ed services for retardation and learning disabilities declined in 47 states between 1994 and 2003, just as those getting help for autism was rising. In 44 states, the drop exceeded the rise in autism.

As convincing as Grinker’s analysis seems, arguments about the apparent epidemic will probably continue. It’s simply impossible to accurately reconstruct the past incidence of the disorder, given how radically definitions have changed. Those who believe the increase is real often focus on the mysterious paucity of autistic adults. With their conspicuous symptoms like hand flapping and little or no language, "I think we would be recognizing them in institutions," says Dr. Robert Hendren, executive director of the M.I.N.D. Institute at the University of California, Davis.
“A Ticket for Service”

or

“Diagnostic Substitution” (Fombonne 2005)
The Contribution of Diagnostic Substitution to the Growing Administrative Prevalence of Autism in US Special Education

Paul T. Shattuck, PhD

Waisman Center, University of Wisconsin, Madison, Wisconsin

The author has indicated he has no financial relationships relevant to the article to disclose.

ABSTRACT

OBJECTIVE. Growing administrative prevalence of autism has stirred public controversy and concern. The extent to which increases in the administrative prevalence of autism have been associated with corresponding decreases in the use of other diagnostic categories is unknown. The main objective of this study was to examine the relationship between the rising administrative prevalence of autism in US special education and changes in the use of other classification categories.

METHODS. The main outcome measure was the administrative prevalence of autism among children ages 6 to 11 in US special education. Analysis involved estimating multilevel regression models of time-series data on the prevalence of disabilities among children in US special education from 1984 to 2003.

RESULTS. The average administrative prevalence of autism among children increased from 0.6 to 3.1 per 1000 from 1994 to 2003. By 2003, only 17 states had a special education prevalence of autism that was within the range of recent epidemiological estimates. During the same period, the prevalence of mental retardation and learning disabilities declined by 2.5 and 8.3 per 1000, respectively. Higher autism prevalence was significantly associated with corresponding declines in the prevalence of mental retardation and learning disabilities. The declining prevalence of mental retardation and learning disabilities from 1994 to 2003 represented a significant downward deflection in their preexisting trajectories of prevalence from 1984 to 1993. California was one of a handful of states that did not clearly follow this pattern.

CONCLUSIONS. Prevalence findings from special education data do not support the claim of an autism epidemic because the administrative prevalence figures for most states are well below epidemiological estimates. The growing administrative prevalence of autism from 1994 to 2003 was associated with corresponding declines in the usage of other diagnostic categories.

Grinker’s answer is that autistic adults are out there but wearing other labels. "Where are all the adults with fetal alcohol syndrome?" he asks. No one over 40 has the condition, thought to affect up to 1 in 500 kids today, because it was not recognized until the mid-'70s. "But no one would say alcoholism among pregnant women just started," says Grinker.

Grinker, whose 15-year-old daughter is autistic, concedes that there's something reassuring about the idea of an epidemic: "Thinking about any disorder as an epidemic is easier than thinking about it in terms of multiple causes, shifting definitions and a scientific reality we are only just beginning to understand." Besides, if a disease suddenly spikes, it seems more plausible that the increase could be reversed--if only we could find the mysterious environmental trigger. With autism, though, that hopeful scenario seems just too simple.
Autism and vaccines
The Truth About Vaccines

Worried about autism, many parents are opting out of immunizations. How they're putting the rest of us at risk

BY ALICE PARK
H Honda et al.
Vaccines and the changing epidemiology of autism

B. Taylor
Community Child Health, Royal Free and University College Medical School, UCL, Hampstead Campus, London, UK.
Accepted for publication 5 April 2006

Abstract
Background The epidemiology of autism has been rather confusing, with very variable published prevalence figures and no clear incidence data. The cause of autism is unclear; vaccines have been incriminated.
Methods Literature review and interpretation.
Results The recorded prevalence of autism has increased considerably in recent years. This reflects greater recognition, with changes in diagnostic practice associated with more trained diagnosticians; broadening of diagnostic criteria to include a spectrum of disorder; a greater willingness by parents and educationalists to accept the label (in part because of entitlement to services); and better recording systems, among other factors. The cause(s) of autism remains unclear. There is a strong genetic component which, along with post-natal 'cause', makes any post-natal 'cause' unlikely.

Conclusions There has (probably) been no real increase in the incidence of autism. There is no scientific evidence that the measles, mumps and rubella (MMR) vaccine or the mercury preservative used in some vaccines plays any part in the aetiology or triggering of autism, even in a subgroup of children with the condition.
U.S. Court Finds No Link Between Vaccines, Autism

By Shankar Vedantam
Washington Post Staff Writer
Friday, February 13, 2009

A special federal court ruled yesterday that vaccines do not cause autism and that thousands of families with autistic children are not entitled to compensation, delivering a major blow to an international movement that has tried for years to link childhood immunizations with the devastating disorder.
Wakefield’s article linking MMR vaccine and autism was fraudulent

Fiona Godlee, editor in chief, Jane Smith, deputy editor, Harvey Marcovitch, associate editor

Correspondence to: F Godlee fgodlee@bmj.com

Clear evidence of falsification of data should now close the door on this damaging vaccine scare

“Science is at once the most questioning and . . . sceptical of activities and also the most trusting,” said Arnold Reiman, former editor of the New England Journal of Medicine, in 1989. “It is intensely sceptical
Autism and MMR vaccine controversy

January 2010

The General Medical Council in Great Britain censures Dr. Andrew Wakefield and other doctors who conducted research in the late 1990s claiming links between a common children's vaccine for combined measles-mumps-rubella (MMR) and autism.

Wakefield said the allegations against him and his colleagues were unfounded and unjust.
Causes of Autism Spectrum Disorders

Genetic causes:
- Chromosomal rearrangements: ~7% cases
- Single gene mutations: up to 5% cases
- Sub-microscopic CNVs

Environmental causes:
- Chemicals
- Vaccines
- Pregnancy complications
- Viral infections
- Diet
- Assisted reproduction
- Advancing Paternal Age

Epigenetic causes
- Epigenetic modifications can change gene expression patterns without changing primary nucleotide sequence (i.e. no mutation)
- Epigenetic Mechanisms include: DNA methylation, chromatin conformation, histone modifications, RNA silencing
Causes of ASD?

Nutrition/ Digestive disorders 1990s-Present

Parental Practice 1980s-1990s

Vaccines 1998-2009

Viruses/ Impaired Immunity 2000-Present

Brain/ Neurological 1990s-Present

Environmental 1990s-Present

Pollution

Drugs/Toxin

Genetics 2002-Present

Medical 1990s-Present

Fragile X syndrome

Rett syndrome

Tuberous Sclerosis
Mirror Neuron Theory
Figure 1. Mirror regions of the human brain. The core of the mirror neuron system is located in the inferior frontal (a) and inferior parietal (b) regions of the human brain. These areas are the human equivalent of regions F5 and inferior parietal lobule in the macaque brain, where mirror neurons have been reported [1], and several fMRI studies have demonstrated that these areas respond during performance, observation and imitation of hand actions [1]. In the current paper discussion of the MNS refers to this core system. Some recent data suggest that our concept of a mirror system should be extended to encompass brain regions that respond to a wider range of stimuli for both self and other (blue regions). For example, the secondary somatosensory cortex (c) responds to performed and observed touch, whereas the anterior insula (d) and anterior cingulate (e) cortex respond when a person experiences, observes or imitates emotional facial expressions (reviewed in [49]). It is possible that future work will identify more brain regions with mirroring properties. However, it is unlikely that all these different brain regions can function as a single mirror neuron system. Rather, there might be several mirroring systems that could be differentially involved in different social processes and differentially impaired or intact in autism.
Unbroken mirrors: challenging a theory of Autism

Victoria Southgate\(^1\) and Antonia F. de C. Hamilton\(^2\)

\(^1\)Centre for Brain and Cognitive Development, School of Psychology, Birkbeck College, London, WC1E 7HX, UK
\(^2\)School of Psychology, University of Nottingham, Nottingham, NG7 2RD, UK

The ‘broken mirror’ theory of autism has received considerable attention far beyond the scientific community. This theory proposes that the varied social-cognitive difficulties characteristic of autism could be explained by dysfunction of the mirror neuron system, thought to play a role in imitation. We examine this theory and argue that explaining typical imitation behavior, and the failure to imitate in autism, requires much more than the mirror neuron system. Furthermore, evidence for the role of the mirror neuron system in autism is weak. We suggest the broken mirror theory of autism is premature and that better cognitive models of social behavior within and beyond the mirror neuron system are required to understand the causes of poor social interaction in autism.
Time for fatherhood: Aging affects sperm
EXCLUSIVE: Baby-faced father of little Maisie

DAD AT 13
Autism is a heterogeneous neurodevelopmental syndrome with a complex genetic etiology. It is still not clear whether autism comprises a vast collection of different disorders akin to intellectual disability or a few disorders sharing common aberrant pathways. Unifying principles among cases of autism are likely to be at the level of brain circuitry in addition to molecular pathways.
The Autistic Neuron: Troubled Translation?

Raymond J. Kelleher III* and Mark F. Bear* 

1Center for Human Genetic Research, Harvard-Partners Center for Genetics and Genomics, and the Autism Consortium, Massachusetts General Hospital, Program in Neuroscience and Department of Neurology, Harvard Medical School, Boston, MA 02115, USA 

2Howard Hughes Medical Institute, Picower Institute for Learning and Memory, Department of Brain and Cognitive Sciences, and the Autism Consortium, Massachusetts Institute of Technology, Cambridge, MA 02139, USA 

*Correspondence: kelleher@helix.mgh.harvard.edu (R.J.K.), mbear@mit.edu (M.F.B.)

DOI 10.1016/j.cell.2008.10.017

Autism is a complex genetic disorder, but single-gene disorders with a high prevalence of autism offer insight into its pathogenesis. Recent evidence suggests that some molecular defects in autism may interfere with the mechanisms of synaptic protein synthesis. We propose that aberrant synaptic protein synthesis may represent one possible pathway leading to autistic phenotypes, including cognitive impairment and savant abilities.
Battle of the sexes may set the brain

A tug-of-war between the mother’s and father’s genes in the developing brain could explain a spectrum of mental disorders from autism to schizophrenia, suggest Christopher Badcock and Bernard Crespi.
Screening tools for ASD
Age-Line for Screening Tests for ASD

0 3m 6m 9m 12m 18m 2y 3y 6y 18y

- ASSQ (6y-17y)
- PDDAS (6y-12y)
- ASAS (5y +)

Chat 23 (16m-86m)
- BITSEA (16m-36m)
- M-CHAT (16m-48m)

ASQ-SE (6m-16m)
- CSBS-DP (6m-24m)
- CAT/CLAMS (3m-36m)
- POINT (2m-36m)
- BINS-III (1-42m)
- ASQ-3 (1-66m)

Denver II (0-6m)
- IDI (0-18m)
- Brigance Screens – II (0-90m)
- BDI-ST (0-95m)
- Social-Emotional Growth Chart (0-42m)
- PEDS 0-8yrs
- DP-3 (0-12y)

Autism Research Institute's Form E-2 Checklist (3y-5y)
- SCQ (4y +)
- CAST (4y-11y)

DIAL – 3 (3y-7y)
- ESP (2y-6y)
- STATS (24m-35m)

SKOLD (30-48m)
- CHAT (18m - 3yr)
- CDR-PQ (18m-4y)
- LDS (18m-5y)
- ASIEP-3 (18m+)
- PDDST-II (18m +)
Age-Line for Diagnostic Tests for ASD

- ADOS (Toddler to Adult)
- CARS 2 (1y+)
- ADI-R (2y+)
- GADS (3yr – 22 yr)
- GARS-2 (3y-22y)
- CARS (3yr – 22 yr)
- ASDS (5y – 18y)
- KADI (6y – 21y)
Age-Line for Assessment Instruments for ASD

TABS (11m–17m)
ATEC (1y-12y)
CDI (15m-6y)
CBCL (18m-18y)
DIAL-3 (3y-<7y)
ABC (3y +)
SRS (4y-18y)

0          3          6          9          12          15          18          2y          3y          6y          18y
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<th>Instrument/Resource</th>
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<tr>
<td>Autism Diagnostic Interview (ADI-R)</td>
<td>Caregiver interview</td>
<td>Keyed to DSM-IV criteria, widely used in research, requires training</td>
</tr>
<tr>
<td>Autism Diagnostic Observation Schedule (ADOS)</td>
<td>Structured client interaction/interview</td>
<td>Widely used in research, social probe, requires training, complements ADI</td>
</tr>
<tr>
<td>Childhood Autism Rating Scale (CARS)</td>
<td>Rating scale based on history, report, and observation</td>
<td>Some training needed, targeted screening instrument</td>
</tr>
<tr>
<td>Autism Behavior Checklist (ABC)</td>
<td>Caregiver rating</td>
<td>Minimal training needed, scales quantify maladaptive behavior, screening instrument for teachers</td>
</tr>
<tr>
<td>Modified Checklist for Autism In Toddlers (M-CHAT)</td>
<td>Parental report</td>
<td>Screening instrument for toddlers</td>
</tr>
</tbody>
</table>

Autism Diagnostic Interview, Revised (ADI-R)
Autism Diagnostic Observation Schedule (ADOS)
Childhood Autism Rating Scale, Second Edition (CARS2)
Modified Checklist for Autism in Toddlers (M-CHAT) (1999)
M-CHAT-Language versions available as of 2011

Arabic 2; Bangla 1; Cambodia 1; Chinese 3; Czech 1; Dutch 2; English 17; French 3; German 2; Greek 1; Gujarati 1; Hmong 1; Hebrew 1; Hungarian 1; Icelandic 1; Japanese 2; Italian 2; Kannada 1; Korean 2; Kurdish 1; Malay 1; Lao 1; Malay 1; Portuguese 2; Polish 1; Russian 2; Sinhala 1; Somali 1; Spanish 13; Swedish 1; Tamil 1; Turkish 2; Tagalog 1; Vietnamese 2; Urdu 1; Yugoslavian 1
CHAT, M-CHAT and CHAT-23
Section A: Ask parent

1. Does your child enjoy being swung, bounced on your knee, etc.?  
2. Does your child take an interest in other children?  
3. Does your child like climbing on things, such as up stairs?  
4. Does your child enjoy playing peek-a-boo/hide-and-seek?  
5. Does your child ever PRETEND, for example, to make a cup of tea using a toy cup and teapot, or pretend other things?  
6. Does your child ever use his/her index finger to point, to ASK for something?  
7. Does your child ever use his/her index finger to point, to indicate INTEREST in something?  
8. Can your child play properly with small toys (e.g. cars or bricks) without just mouthing, fiddling or dropping them?  
9. Does your child ever bring objects over to you (parent) to SHOW you something?  

Section B: General Practitioner or Health Visitor Observation

i. During the appointment, has the child made eye contact with you?  
ii. Get child’s attention, then point across the room at an interesting object and say ‘Oh look! There’s a [name of toy]!’ Watch child’s face. Does the child look across to see what you are pointing at?  
iii. Get the child’s attention, then give child a miniature toy cup and teapot and say ‘Can you make a cup of tea?’ Does the child pretend to pour out tea, drink it, etc.?  
iv. Say to the child ‘Where’s the light?’, or ‘Show me the light’. Does the child POINT with his/her index finger?  
v. Can the child build a tower of bricks? (If so how many?) (No. of bricks: . . . . . . . )
CHAT
(18m)
Diagnostic criteria for autism

1. Failing all 5 critical items (i.e. A5, A7, B2, B3, B4) indicates high risk for autism group

2. Failing the 2 keys items (i.e. A7, B4) indicates medium risk for autism group
Section A: Ask parent

1. Does your child enjoy being swung, bounced on your knee, etc.? Yes No
2. Does your child take an interest in other children? Yes No
3. Does your child like climbing on things, such as upstairs? Yes No
4. Does your child enjoy playing peek-a-boo/hide-and-seek? Yes No
5. Does your child ever PRETEND, for example, to make a cup of tea using a toy cup and teapot, or pretend other things? Yes No
6. Does your child ever use his/her index finger to point to ASK for something? Yes No
7. Does your child ever use his/her index finger to point to indicate INTEREST in something? Yes No
8. Can your child play properly with small toys (e.g. cars or bricks) without just mouthing, fiddling or dropping them? Yes No
9. Does your child ever bring objects over to you (parent) to SHOW you something? Yes No

Section B: General practitioner or health visitor observation

i. During the appointment, has the child made eye contact with you? Yes No
ii. Get child’s attention, then point across the room at an interesting object and say ‘Oh look! There’s a (name of toy)!’ Watch child’s face. Does the child look across to see what you are pointing at? Yes No
iii. Get the child’s attention, then give child a miniature toy cup and teapot and say ‘Can you make a cup of tea?’ Does the child pretend to pour out tea, drink it, etc.? Yes No
iv. Say to the child ‘Where’s the light?’, or ‘Show me the light’. Does the child POINT with his/her index finger at the light? Yes No
v. Can the child build a tower of bricks? (If so how many?) (No. of bricks: . . . . . .) Yes No
# M-CHAT (23 questions)

1. Does your child enjoy being swung, bounced on your knee, etc.?  | Yes | No  
2. Does your child take an interest in other children?  | Yes | No  
3. Does your child like climbing on things, such as up stairs?  | Yes | No  
4. Does your child enjoy playing peek-a-boo/hide-and-seek?  | Yes | No  
5. Does your child ever pretend, for example, to talk on the phone or take care of dolls, or pretend other things?  | Yes | No  
6. Does your child ever use his/her index finger to point, to ask for something?  | Yes | No  
7. Does your child ever use his/her index finger to point, to indicate interest in something?  | Yes | No  
8. Can your child play properly with small toys (e.g., cars or bricks) without just mouthing, fiddling, or dropping them?  | Yes | No  
9. Does your child ever bring objects over to you (parent) to show you something?  | Yes | No  
10. Does your child look you in the eye for more than a second or two?  | Yes | No  
11. Does your child ever seem oversensitive to noise? (e.g., plugging ears)  | Yes | No  
12. Does your child smile in response to your face or your smile?  | Yes | No  
13. Does your child imitate you? (e.g., you make a face—will your child imitate it?)  | Yes | No  
14. Does your child respond to his/her name when you call?  | Yes | No  
15. If you point at a toy across the room, does your child look at it?  | Yes | No  
16. Does your child walk?  | Yes | No  
17. Does your child look at things you are looking at?  | Yes | No  
18. Does your child make unusual finger movements near his/her face?  | Yes | No  
19. Does your child try to attract your attention to his/her own activity?  | Yes | No  
20. Have you ever wondered if your child is deaf?  | Yes | No  
21. Does your child understand what people say?  | Yes | No  
22. Does your child sometimes stare at nothing or wander with no purpose?  | Yes | No  
23. Does your child look at your face to check your reaction when faced with something unfamiliar?  | Yes | No
Diagnostic criteria for autism
M-CHAT
18-24m

1. Failing any 3 out of all (23) questions in part A

OR

2. Failing any 2 out of the 6 discriminative items (2, 7, 9, 13, 14, 15)
1. Does your child enjoy being swung, bounced on your knee, etc.?  
2. Does your child take an interest in other children?  
   Yes  
   No  
3. Does your child like climbing on things, such as up stairs?  
   Yes  
   No  
4. Does your child enjoy playing peek-a-boo/hide-and-seek?  
   Yes  
   No  
5. Does your child ever pretend, for example, to talk on the phone or take care of dolls, or pretend other things?  
   Yes  
   No  
6. Does your child ever use his/her index finger to point, to ask for something?  
   Yes  
   No  
7. Does your child ever use his/her index finger to point, to indicate interest in something?  
   Yes  
   No  
8. Can your child play properly with small toys (e.g., cars or bricks) without just mouthing, fiddling, or dropping them?  
   Yes  
   No  
9. Does your child ever bring objects over to you (parent) to show you something?  
   Yes  
   No  
10. Does your child look you in the eye for more than a second or two?  
    Yes  
    No  
11. Does your child ever seem oversensitive to noise? (e.g., plugging ears)  
    Yes  
    No  
12. Does your child smile in response to your face or your smile?  
    Yes  
    No  
13. Does your child imitate you? (e.g., you make a face—will your child imitate it?)  
    Yes  
    No  
14. Does your child respond to his/her name when you call?  
    Yes  
    No  
15. If you point at a toy across the room, does your child look at it?  
    Yes  
    No  
16. Does your child walk?  
    Yes  
    No  
17. Does your child look at things you are looking at?  
    Yes  
    No  
18. Does your child make unusual finger movements near his/her face?  
    Yes  
    No  
19. Does your child try to attract your attention to his/her own activity?  
    Yes  
    No  
20. Have you ever wondered if your child is deaf?  
    Yes  
    No  
21. Does your child understand what people say?  
    Yes  
    No  
22. Does your child sometimes stare at nothing or wander with no purpose?  
    Yes  
    No  
23. Does your child look at your face to check your reaction when faced with something unfamiliar?  
    Yes  
    No
Most discriminative feature ...

- Question 7 is the most discriminative as it appeared in all three CHAT instrument as in screening toddlers for autism.

7.
Does your child ever use his/her index finger to point, to indicate interest in something?
<table>
<thead>
<tr>
<th>Behavioral characteristic</th>
<th>Disruptions first reported at 6–12 months</th>
<th>Disruptions first reported at 9–14 months</th>
<th>Disruptions first reported at 20–24 months</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social responsiveness</td>
<td>Infrequent look to others’ faces or gaze aversion\textsuperscript{14,101} Poor eye contact\textsuperscript{101}</td>
<td>Abnormal orienting to name or others’ voice\textsuperscript{16–19,102} Infrequent monitoring of others’ gaze\textsuperscript{35,103} Infrequent response to others’ nonverbal cues of shifted attention\textsuperscript{35,104} Lack of imitation\textsuperscript{105} Lack of interest in other children\textsuperscript{39} Infrequent social gaze in response to others’ distress\textsuperscript{105} Brief duration of gaze towards others\textsuperscript{103}</td>
<td></td>
</tr>
<tr>
<td>Social initiation</td>
<td>Poor social initiative\textsuperscript{15}</td>
<td>Infrequent initiation of joint attention through pointing or showing\textsuperscript{18,35} Infrequent directing of play acts towards others\textsuperscript{35} Infrequent initiation of communicative bids for social or regulatory purposes\textsuperscript{35}</td>
<td>Infrequent seeking to share\textsuperscript{39}</td>
</tr>
<tr>
<td>Social–emotional interaction</td>
<td>Absence of facial expression\textsuperscript{14,15} Decreased frequency of smiling\textsuperscript{101} Lack of emotional modulation\textsuperscript{15}</td>
<td>Infrequent sharing of positive affect\textsuperscript{35}</td>
<td>Limited range of facial expression\textsuperscript{39} Infrequent offering of comfort\textsuperscript{39}</td>
</tr>
<tr>
<td>Communication and play</td>
<td>Delay in babbling\textsuperscript{26} Decreased frequency of vocalization\textsuperscript{101}</td>
<td>Low diversity in consonants produced communicatively\textsuperscript{35} Infrequent and low variety of conventional gestures\textsuperscript{20,35,107} Delayed receptive and expressive language\textsuperscript{25} Reduced variety of play acts\textsuperscript{35} Reduced variety of action sequences in play\textsuperscript{35} Failure to integrate gaze with other communication behaviors\textsuperscript{106} Reduced inventory of words produced\textsuperscript{35,66} Abnormal prosody\textsuperscript{106}</td>
<td></td>
</tr>
<tr>
<td>Sensory, motor or attention behavior</td>
<td>Abnormal postural control\textsuperscript{24} Atypical movements\textsuperscript{24} Low diversity of movements\textsuperscript{24} Hypotonicity\textsuperscript{15,24} Poor motor coordination\textsuperscript{24,26} Hypoactivity and passivity, and decreased object exploration\textsuperscript{15,24} Abnormal pattern and focus of attention\textsuperscript{15}</td>
<td>Repetitive and perseverative actions\textsuperscript{107,108} Difficulty with attention disengagement\textsuperscript{16} Abnormalities in arousal or unusual sensory responses\textsuperscript{14,16}</td>
<td>Repetitive behaviors and restricted interests\textsuperscript{106}</td>
</tr>
</tbody>
</table>
Eye Contact

I am so cutie…

Oh my God, how come I am so ugly..& fluffy?
Non-autistic person
Imitation
Can you recognize ME?
Can you recognize ME?
Fig. 2. Functional regions of interest for faces (FFAs—red) and objects (PHGs—blue) defined by the localizer paradigm.

The Fusiform Face Area is tuned for curvilinear patterns with more high-contrasted elements in the upper part.

Reference
Caldara R
Comparison of the effect of facial expression on FFA activation in 3 healthy young adult males free of social disability.

(A) The fMRI task involved a block design with person identity discrimination of expressive faces (top) or neutral faces (bottom).

(B) Expressive faces elicited significantly greater (p=0.000002, fixed effects, uncorrected) FFA activation (shown in red/yellow) in the fusiform gyrus (y=-49).

(C) Left and right hemispheres are reversed by convention in this coronal orientation; note the greater right fusiform gyrus activation to faces.

Example of FFA activation (circled in red) in a typical young adult male compared to a young adult male with autism.

Maps threshold at $p < 0.01$, uncorrected.

Areas in red/yellow depict brain areas that are significantly more active during perception of faces;
Areas in blue show where brain was more active during perception of non-face objects.

The right side of the brain is shown on the left side of the image, as if you were looking at the person face on.

Schultz 2005;23:125-141
Figure 1. Brain areas that have been implicated in the mediation of the three core behaviors that are impaired in autism: social behavior, language and communication, and repetitive and stereotyped behaviors.
Neuropsychology
Theory of Mind (ToM)
Mind-blindness

Can be described as an inability to develop an awareness of what is in the mind of another human.

It is not necessarily caused by an inability to imagine an answer, but is often due to not being able to gather enough information to work out which of the many possible answers is correct.

Mind-blindness is the opposite of Empathy.

Simon Baron-Cohen was the first person to use the term 'mind-blindness' to help understand some of the problems encountered by people with Autism or Asperger Syndrome or other developmental disorders.
my fake smile is better than your fake smile
Figure 1. The Kanizsa Triangle
Animals do experience the illusion, although some species may be more inclined to attend to details, of the pac-man before extracting the overall triangular shape (see text).
Figure 2. Laterlization in the Avian Brain, Illustrated Using the Australian Magpie, *Gymnorhina tibicen*. Behaviour controlled by the left and right hemispheres is listed. As in humans, functions of the left hemisphere are consistent with those of autistic humans. (Photograph: G. Kaplan, Centre for Neuroscience and Animal Behaviour, University of New England, Australia).
The 2010 TIME 100

In our annual TIME 100 issue we name the people who most affect our world

Heroes

Temple Grandin

By MARC HAUSER  Thursday, Apr. 29, 2010

What do neurologists, cattle and McDonald’s have in common? They all owe a great deal to one woman, a renowned animal scientist born with autism, Temple Grandin. Though she didn’t utter a word until close to her fourth birthday, substituting screams for phonemes, she splashed onto the stage of public awareness in 1995, thanks to the vivid, sensitive writing of the famed neurologist Oliver Sacks. Little was known about autism at the time except that people so afflicted appeared socially isolated, emotionally fragile and difficult to engage. But as with many psychological disorders, autism is a spectrum, and Temple, 62, is on one edge. Living on this edge has allowed her to be an extraordinary source of inspiration for autistic children, their parents — and all people. She is also a source of hope for another mammal: the cow.

Using her unique window into the minds of animals, she has developed corrals for cattle that improve their quality of life by reducing stress. And though the fast-food industry continues to use cattle in its patties, it has come to appreciate the ethics and compassion of a Grandin burger.
Dr Temple Grandin, Neurologist, Author, Top 100 Influential People in TIME 2010
Box 1. Response by Temple Grandin to the Essay “Are Animals Autistic Savants?”

Editors note: We asked Temple Grandin to respond to the essay “Are Animals Autistic Savants?”, which presents a critique, based on studies of comparative animal cognition, of Vallortigara’s premise that animals and autistic savants share similarities in cognition.

The Essay “Are Animals Autistic Savants?” by Giorgio Vallortigara et al. provides a fascinating overview of the most recent research on animal cognition. I think the basic disagreement between the authors and me arises from the concept of details—specifically how details are perceived by humans, who think in language, compared with animals, who think in sensory-based data. Since animals do not have verbal language, they have to store memories as pictures, sounds, or other sensory impressions. Sensory-based information by its very nature is more detailed than word-based memories. As a person with autism, all my thoughts are in photo-realistic pictures. I can search my own brain, like using Google, for images. As I read about the cognition experiments, I saw the birds performing in my imagination like a virtual reality computer system. The main similarity between animal thought and my thought is the lack of verbal language. Verbal language narrates the images when I “surf the Internet” inside my own brain. If you give me a “keyword,” such as “peanut,” I start to see images like a series of slides shown one after the other. The first image was the Planter’s Peanut logo, the second was a Western restaurant that serves peanuts, and the third was a bag of peanuts on a plane.

The Essay by Vallortigara et al. clearly showed that cognition is very real in animals. In normal humans, higher brain processes cover up the sensory-based processing that we share with animals. In Animals in Translation, I discussed the work of Bruce Miller, who studied patients with frontal-temporal lobe dementia [1,2]. As the disease destroyed higher brain function art and music talent emerged. The sensory-based, more-detailed thinking we share with animals was unmasked.

I was most interested to learn that the skills that taxi drivers had developed to navigate by using landmarks “had come at the price of acquiring new visual spatial information” according to the authors. Further experiments need to be done with birds to either confirm or disprove Vallortigara et al.’s hypothesis that birds such as the Clark’s nutcracker, which has savant-like memory for food storage, has retained good cognition in other domains. My hypothesis is that birds that have savant-like skills for food storage sites or remembering migration routes may be less flexible in their cognition. It is well known that people with autism do poorly on the Wisconsin card sorting task, where colors and shapes have to be sorted into different categories. The person with autism is slower to respond correctly when the category is switched from a shape to a color.

An operant conditioning task could be used to compare flexible problem solving in migratory and food-storing bird species to species that do not have savant-like skills. The task could be to peck a lever when a light comes on. After the bird had learned this, the stimulus would be switched to a sound. I predict that the more savant-like birds will require more trials to switch back and forth between the light and the sound cue.

I am pleased that my book has stimulated so much discussion, and I hope it stimulates more research on animal cognition.
The Hamburger Kid
漢堡飽小子

Toronto 2005 多倫多

01/03/2011 13:43
1st CIP Paris VW
102
METROPOLITAN HOTEL
TORONTO

"Hamburger" para-graph

1st sentence - thesis
- introduces topic

2nd sentence - 1st supporting sentence and analysis
- first impression of

3rd sentence - 2nd supporting sentence and analysis

4th sentence - 3rd supporting sentence and analysis

5th sentence - concluding sentence
- stay on topic

Do not
- use pronouns

Use "yes" or "no"
The Boy with the “Eagle-Eye”

“鷹眼”小子
A Boy with William Syndrome
Evidence is reviewed suggesting that, in the general population, empathizing and systemizing show strong sex differences. The function of systemizing is to predict lawful events, including lawful change, or patterns in data. Also reviewed is the evidence that individuals on the autistic spectrum have degrees of empathizing difficulties alongside hypersystemizing. The hypersystemizing theory of autism spectrum conditions (ASC) proposes that people with ASC have an unusually strong drive to systemize. This can explain their preference for systems that change in highly lawful or predictable ways; why they become disabled when faced with systems characterized by less lawful change; and their “need for sameness” or “resistance to change”. If “truth” is defined as lawful patterns in data then, according to the hypersystemizing theory, people with ASC are strongly driven to discover the “truth”.
<table>
<thead>
<tr>
<th>Profile of individuals</th>
<th>Shorthand notation</th>
<th>Type of brain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Empathizing more developed than systemizing</td>
<td>$E &gt; S$</td>
<td>“female” (or Type E)</td>
</tr>
<tr>
<td>Systemizing more developed than empathizing</td>
<td>$S &gt; E$</td>
<td>“male” (or Type S)</td>
</tr>
<tr>
<td>Systemizing and empathizing both equally developed</td>
<td>$S = E$</td>
<td>“balanced” (or Type B)</td>
</tr>
<tr>
<td>Systemizing hyperdeveloped and empathizing hypodeveloped (the autistic end of the spectrum)—may be talented systemizers, but at the same time may be “mind blind”</td>
<td>$S &gt;&gt; E$</td>
<td>extreme male brain</td>
</tr>
<tr>
<td>Hyperdeveloped empathizing skills and systemizing hypodeveloped—may be “system blind”</td>
<td>$E &gt;&gt; S$</td>
<td>extreme female brain (postulated)</td>
</tr>
</tbody>
</table>
April was slightly above average in temperature. It was the warmest April since 1950 and was very dry. It strongly implies the smallest rainfall of the year.

May was slightly drier than average and temperatures were normal.

June was dry with average temperature.

July was cool and very dry, it was the wettest July since 1930 and

August was warm but drier than average.

September was sunny and warm.

October was dry with average temperature. It was the driest October since 1997.

November was very mild and very dry. It was the mildest since 1997 and the drier since 1993.

December was milder than average and the driest December since 1995.

2002 was the driest year since 1992.
Figure 2. A family pedigree. The informant recorded occupational data where known and any suggestion of strong systemizing interests, again where known. She also indicated definite diagnoses of autism spectrum conditions (ASC; shaded blue) and strongly suspected cases of ASC (shaded yellow). Also indicated is one case of polycystic ovary syndrome (PCOS), which is testosterone related. Such one-off pedigrees are not very informative, but are suggestive of a genetic association between strong systemizing and ASC liability. Current research is investigating whether the rate of ASC is higher in such families and whether this reflects assortative mating between two strong systemizers. To view the figure in colour, visit the journal’s website (http://www.psyress.com/jjep) and navigate to the online version of the paper.
Asperger "Talents"
Albert Einstein ‘found genius through autism’

By Nic Fleming, science correspondent
Last Updated: 4:01pm GMT 21/02/2008

Many leading figures in the fields of science, politics and the arts have achieved success because they had autism, a leading psychiatrist has claimed.

Michael Fitzgerald, Professor of Psychiatry at Trinity College, Dublin, argues the characteristics linked to autism spectrum disorders (ASDs) were the same as those associated with creative genius.
Scientist

Isaac Newton (1643-1727)
Gravitation

Gregor Johann Mendal (1822-1884)
Father of genetics

Albert Einstein (1879-1955)
Theory of relativity

Nikola Tesla (1856-1943)
Generator

Charles Darwin (1809-1882)
The Origin of Species by Means of Natural Selection

Henry Cavendish (1731-1810)
Hydrogen
Mathematician

Archimedes (287 B.C. – 212 B.C.)

Norbert Wiener (1894-1964)

Cybernetics

David Hilbert (1862-1943)

Grundlagen der Geometrie

Charles Babbage (1791-1871)

Kurt Gödel (1906-1978)

Paul Erdős (1913-1996)
Writer
Gerard Manley Honkins (1844-1889)
H.G. Wells. 1866-1946

President
Thomas Jefferson (1743-1826)

Best known as the primary author of the Declaration of Independence, Thomas Jefferson was the third president of the United States. He was a man of many talents--an architect, an inventor, a scientist, and a collector of books and artifacts of American history. He could read more than five languages and was the U.S. minister to France for several years.
Composer

Mozart
01/03/2011 13:43 (1756-1791)

Beethoven
(1770-1827)

1st CIP Paris VW
Writer
Hans Christian Anderson (1805-1875)

Artist
Gogh, Vincent van (1853-1890)
Many of the features of high-functioning autism/Asperger Syndrome enhance creativity and general effectiveness (apart from social effectiveness); for example, the ability to focus intensely on a topic for very long periods – days at a time – without interruption even for meals, and to take endless pains.

Persons with Asperger Syndrome do not give up when they meet obstacles to their creativity. They are workaholics and show extraordinary capacity for perseverance.
Rain Man (1988)

- 1st movie about people with autism
- an Academy Award winning, 1988 drama film directed by Barry Levinson (US)
- Story between a man and his autistic brother
Suzanne and Bob Wright,
Co-founder of Autism Speaks

"This disorder has taken our children away. It's time to get them back."
SPECIAL REPORT

Autism Speaks: the United States pays up

In recent years, autism has become the golden child of the fund-raising circuit. Meredith Wadman looks at a US public-relations success that is driving research funds and expertise towards this childhood condition, and asks who is missing out.

Toni Braxton, Matthew Broderick, Bill Cosby — perhaps not names you would associate with social or communication difficulties, and yet in one night, they helped raise $1.45 million at a fund-raiser for research into one of the least-understood disorders affecting children: autism.

Star-studded events such as that fund-raiser at New York’s swanky Lincoln Center in April are the latest front of a public-relations battle in 2003, has spent more than $40 million and plans to spend another $100 million over the next five years.

The commonly held belief that the prevalence of autism is rising may be partly responsible for the cash flow. Previously reported as affecting fewer than 1 in 2,000 children, studies over the past five years have found a dramatically increased prevalence: 1 in 150 eight-year-olds in the United States, according

Autism Epidemic...or not?

- Huge rise in prevalence, rate
- Globally affected
- Rising figures
- Environmental?
- Result of unidentified risk factors

- Changes in diagnostic criteria
- Improved detection
- Rise in awareness
- Better record keeping
- More media attention

The term “epidemic” must be used with caution. We must avoid unnecessary panic and be mindful that labels can be misunderstanding and can be misleading.
TIME

Autism Epidemic... or not?
EPIDEMIC OF AWARENESS

Epidemic of Information
Read my mind now?
Guess my Smile....

Happy??

Wicked Smile??

Scared

Can you identify my artificial tooth? Heehee....

Embarrassed ??

01/03/2011 13:43
Animal Smiles to Connect…
Autism “Communicates” -

We need a Consensus on Dx - ------->>