Mental Health Services for Students with Autism Spectrum Disorders

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Disclosures

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National Institutes of Health

Bernier, R. & Gerdts, J. (2010). Autism Spectrum Disorders, ABC-CLIO.

Overview

- Mile High Overview of Autism Spectrum Disorders
- Diagnosis and Characterization
- Etiology
- Neuroscience
- Mental Health Services/Intervention

What do we know about ASD?

- Autism is a neurobiological disorder characterized by impairments in social communication and restricted/repetitive behaviors.
- Is diagnosed in males 3-4 more times than females.
- Autism is found in all social class levels and in all racial/ethnic groups.
- Behaviorally based interventions are effective at improving outcomes.
- There are no genetic or biological tests to diagnose autism.
- Autism has a strong genetic component.

State of the Science on Diagnosis of ASD

ASD per the DSM-5

1. Eliminated the separate diagnostic sub-categories within the autism spectrum and subsuming them under a single category of Autism Spectrum Disorder (ASD)
2. Reduced the number of ASD symptom domains from three to two
3. Added specifiers regarding intellectual functioning, language, and associated conditions
4. Added a rating for level of ASD severity
   Requiring support, requiring substantial support, requiring very substantial support

ASD in the DSM-5

1. **Eliminated the separate diagnostic subcategories within the autism spectrum and subsuming them under a single category of Autism Spectrum Disorder (ASD)**

DSM-5 "Autism Spectrum Disorders"

Includes, *but not as distinct subcategories*:

- Autistic Disorder
- Asperger's Disorder
- Pervasive Developmental Disorder, Not Otherwise Specified
- Childhood Disintegrative Disorder

ASD in the DSM-5

1. Eliminated the separate diagnostic sub-categories within the autism spectrum and subsuming them under a single category of Autism Spectrum Disorder (ASD)

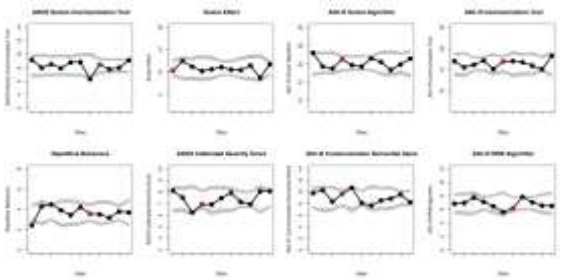
WHY?

- ***Clinicians don't agree on current sub-categories***
- ***Etiological research doesn't support the current distinctions***
- ***Treatment rec are not based on sub-categories***

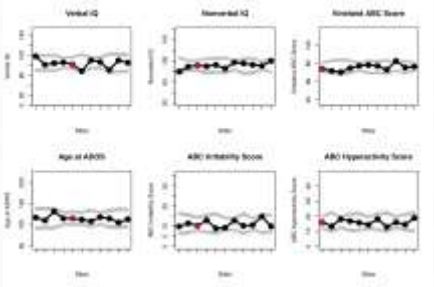
Simons Simplex Collection

- History of including clinical diagnosis in studies of autism within sites (even when blind, reliable diagnoses).
- Examine clinical diagnosis across sites where clinicians reliably use all measures, but haven't established "clinical diagnosis" prototypes.
- Strict guidelines and reliability measures for evaluative tools:
  - ADI
  - ADOS

Expert clinicians don't agree



Expert clinicians don't agree



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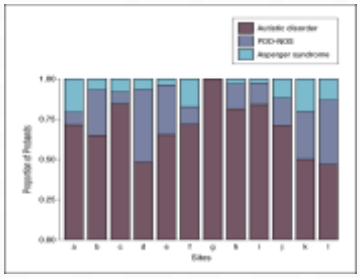


ASD diagnoses assigned by 12 university-based sites *(n = 2,102)*

Lord, C. et al. Arch Gen Psychiatry 2012;69:306-313.

ASD in the DSM-5

2. Reduced the number of ASD symptom domains from three to two

WHY?

- ***Communication and social behaviors are overlapping and hard to distinguish***
- ***Reduces likelihood of some criteria carrying excessive weight***
- ***Secondary data analysis supports the more streamlined approach in terms of sensitivity and specificity***

Diagnostic Process

- Interview of Developmental History
- Intake interview focusing on primary domains of ASD with caregiver (ADI)
- Interaction with individual (ADOS)
- Assessment of language (broadly speaking), cognitive (IQ), and adaptive ability (ABAS, Vineland)

State of the Science on Diagnosis of ASD

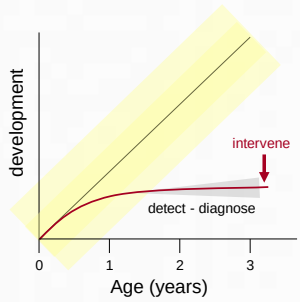
Questions?

State of the Science on Etiology of ASD

- Why do we care about etiology?
- How do we know genetics is involved in ASD?
- Advances in the genetics of ASD
- Current thinking on etiology

Why do we care about etiology?

Early Detection

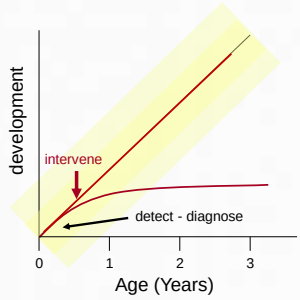


Early Detection

early detection

early intervention

better outcome?



Genetic insights inform mechanism and provide treatment options

genes

proteins

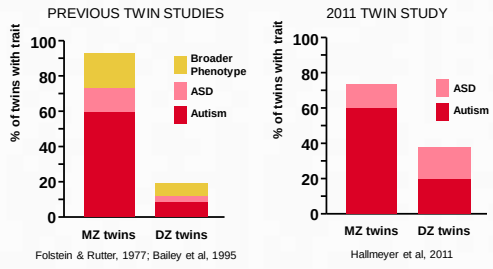
molecules

mechanism

pharmacological interventions?

How do we know genetics is involved in ASD?

Twin studies provide powerful tools for examining etiology



Folstein & Rutter, 1977; Bailey et al, 1995

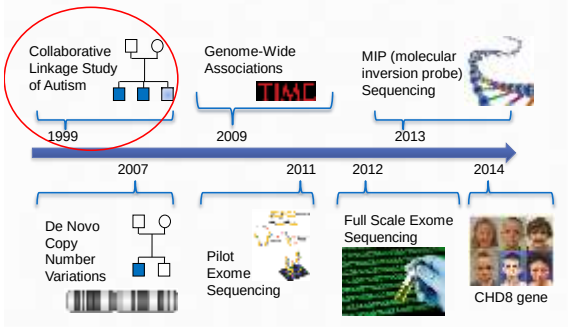
Hallmeyer et al, 2011

Infant siblings inform ASD risk

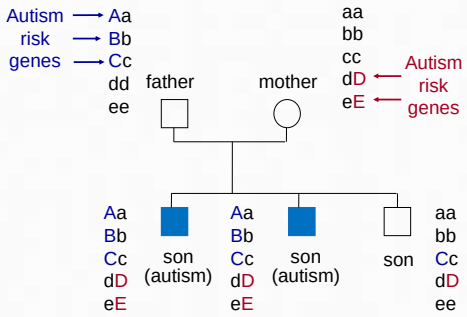
Ozonoff et al, 2011

Advances in the genetics of ASD

A decade and a half of studies



Collaborative Linkage Study of Autism

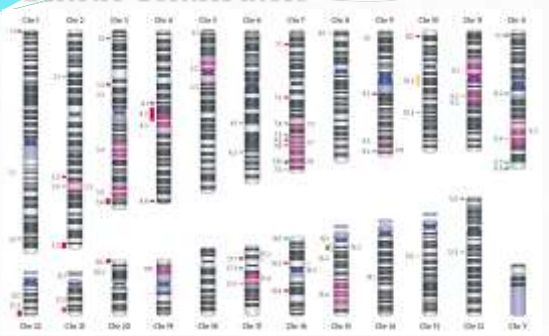


Collaborative Linkage Study of Autism

- Linkage to chromosome 13
- Linkage to chromosome 7

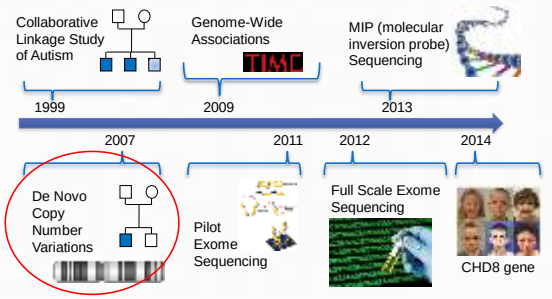
Barrett, et al, 1999

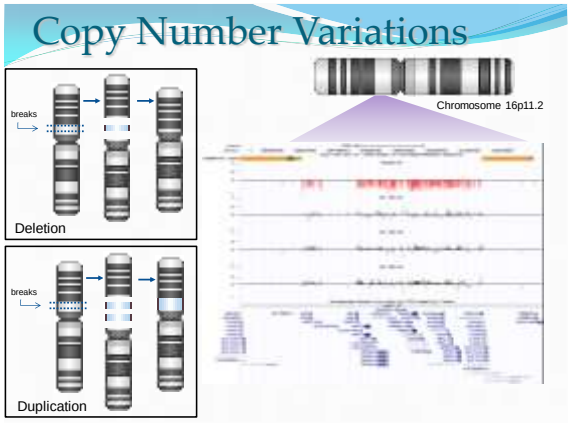
Genetic Contributors



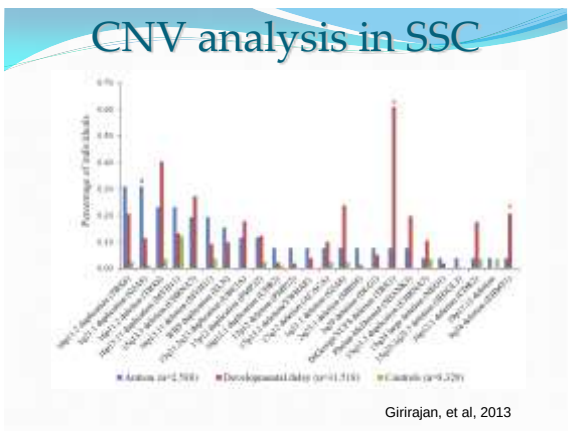
From Abrahams & Geschwind, 2008

A decade and a half of studies

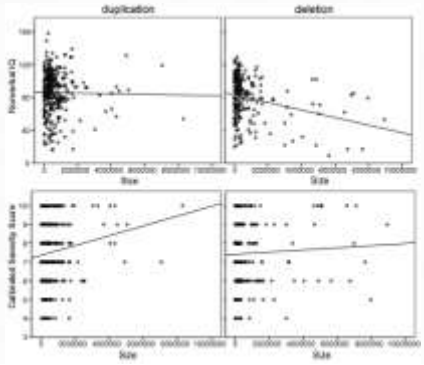



Copy Number Variations
breaks
Deletion
breaks
Duplication
Chromosome 16p11.2

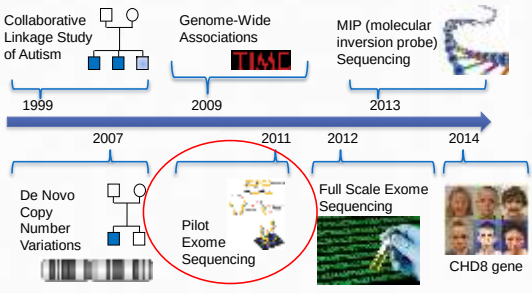

The Simons Simplex Collection
innovation, courage, connection
leading an international investigation into the mysteries of AUTISM
2000 families, one mission


CNV analysis in SSC
Girirajan, et al, 2013

CNV analysis in SSC

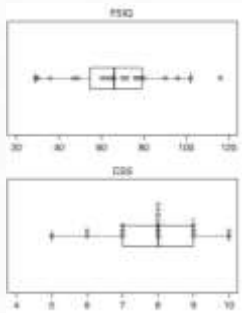


A decade and a half of studies



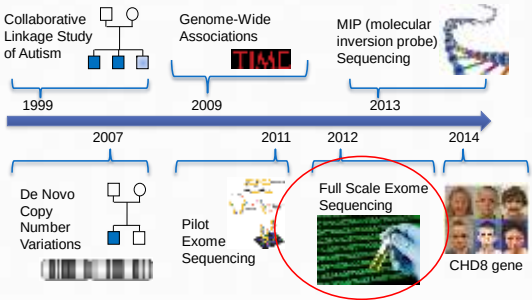
Exome Sequencing in Autism

- Selected 20 individuals
  - No family history of mental illness
  - No BAP in family members
  - Significantly impacted by ASD symptoms
- Identified neurologically expressed gene-disrupting mutations in 4 individuals
- These 4 individuals were more impaired



Oroak et al, 2011, *Nature Genetics*

A decade and a half of studies

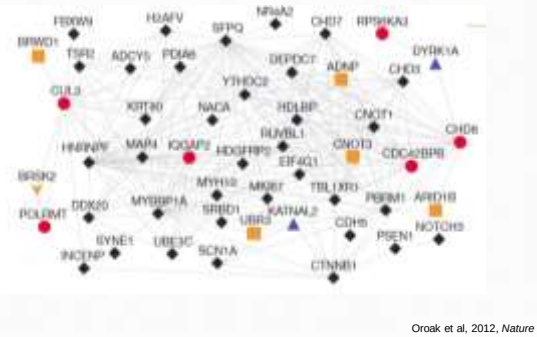


Exome Sequencing in Autism

- Replicated exome study in 209 individuals with ASD
- Identified 248 neurologically expressed gene-disrupting mutations in 25% of the sample
- Only 2 genes with recurrence: *CHD8* (2 individuals) & *NTNG1* (2 individuals)
- Based on this number of mutations, we can estimate there are 384-821 autism risk loci

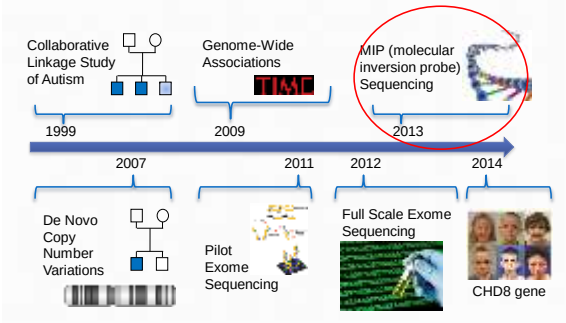
Oroak et al, 2012, *Nature*

Exome Sequencing in Autism



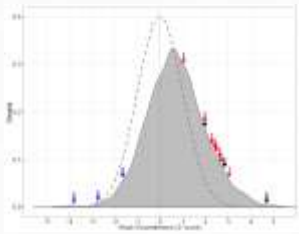
Oroak et al, 2012, *Nature*

A decade and a half of studies



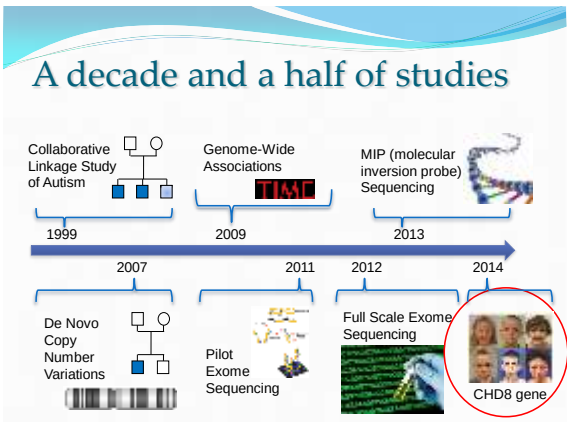
MIP Sequencing

- Tested for disruption of the 44 network genes in over 2000 individuals with ASD.
- Identified recurrent mutations to these genes.
- **The most common 6 genes in this network account for 1% of ASD.**



Oroak et al, 2012, *Science*

A decade and a half of studies



Clinical Characterization

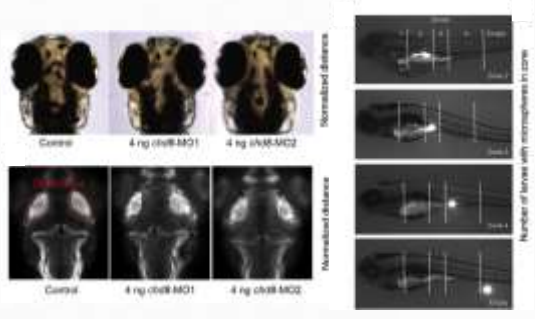
Clinical Characterization

- 87% (13/15) meet strict diagnostic criteria for ASD
- 60% have Intellectual Disability

- 80% have macrocephaly (rapid, early growth)
- 80% tall stature
- Hypertelorism, down-slanted palpebral fissures, prominent forehead, pointed chin

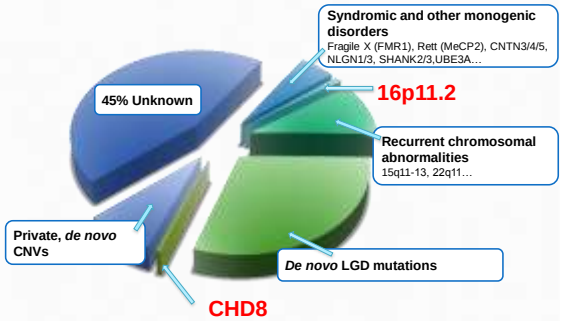
- 80% have gastrointestinal problems (constipation)*
- 67% have significant sleep problems
- 3 of 3 females with precocious puberty (following observation by Talkowski et al, 2012)

Recapitulation in zebrafish



Current thinking on etiology?

Genetic Risk Factors



What about the environment?

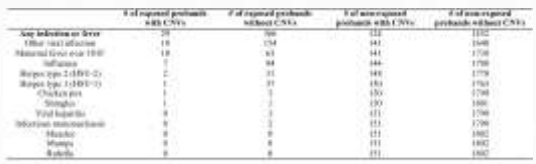
- Genes don't always act alone

Potential Environmental Risk Factors

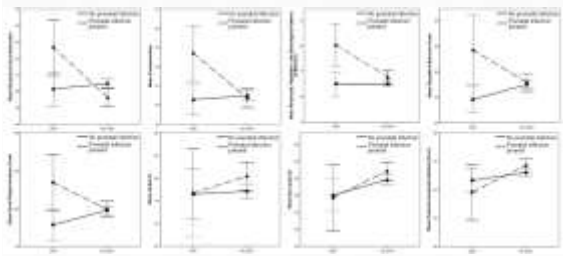
- Toxic exposures during pregnancy:
  - valproic acid (seizure/mood stabilizer)
  - prenatal rubella
  - misoprostol (ulcer treatment)
  - Chlorpyrifos (insecticide)
  - Pollution (proximity to freeways, amt of traffic)
  - Agricultural pesticides
- Increased paternal age
- Interaction between exposures and genetic background

Maternal infection in utero

Reported infectious illnesses and observed copy number variants (CNVs) in probands (N=1971)



Infections and CNVs



Conclusion

- Tremendous behavioral and causal heterogeneity in ASD.
- Genetically defined individuals reveal subtle but real behavioral subtypes in ASD.
- Interactions between genotype and exposures early in development likely contributing.
- By identifying causal mechanisms we can detect autism earlier, develop targeted treatments (at multiple levels), inform course and prognosis, identify individualized interventions.

State of the Science on Etiology of ASD

Questions?

State of the Science on Neuroscience of ASD

- Current theories on neurophysiology of ASD
- Imitation, Mirror Neurons, and ASD

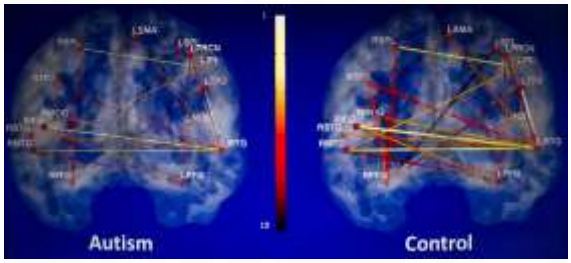
The brain in ASD

- Autopsy, neuropsych testing, imaging, EEG, MEG, TMS, etc all show differential activity in the brain in ASD.
- 3 current theories:
  - Connectivity hypothesis
  - Social information processing hypothesis
  - Social Motivation hypothesis

Connectivity Hypothesis

- Due to poor long range connectivity, simple, local processing is intact while complex, distributed information processing is impaired.
- The nature of the information processed is relevant only insofar as it requires distributed brain function.
- Because social interaction tends to be complex, these theories suggest that it is particularly vulnerable to disruption due to underconnectivity.

Underconnectivity



Kana et al, 2014

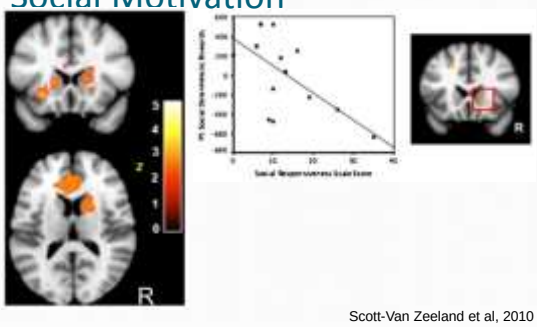
Support for Connectivity

- Imaging study results vary: showing overconnectivity, underconnectivity, and typical connectivity in ASD.
- Movement artifact in MRI actually causes increased close connectivity and decreased long range connectivity: blow to connectivity theories.
- Behavioral correlates are proposed: improved visual-spatial, splinter skills, etc.
- Connectivity unlikely to be universal.

Social Motivation Hypothesis

- Disruption of brain structures related to assignment of reward that result in the cascade of impairments observed in ASD
  - Amygdala, ventral striatum, orbitofrontal and ventromedial prefrontal cortex
- Reduced reward value placed on social stimuli.
- Reduced reward value leads to decreased attention (behaviorally/neurologically) to social, increased to non-social.
- Decreased attention results in decreased experience (and therefore decreased skill and ability) in social cognition.

Social Motivation



Scott-Van Zeeland et al, 2010

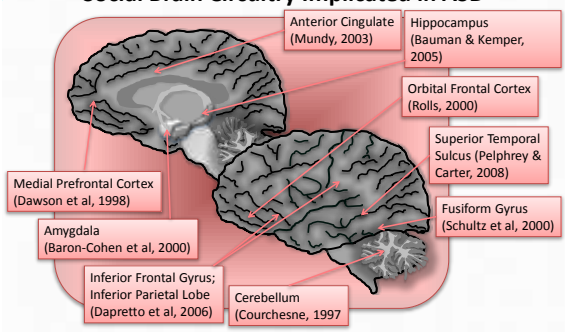
Support for Social Motivation

- Imaging study results suggests differential activation in response to social vs nonsocial rewards.
- Eye tracking studies provide strong support for differential attention to social stimuli
- Design limitations temper conclusions (e.g., faulty comparisons between smiling faces and money; how motivation defined).
- Promising avenue for future work.

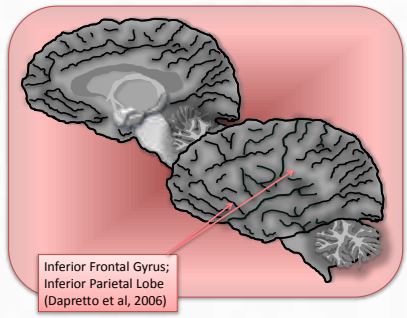
Social Information Processing Hypothesis

- Disruption of brain structures in the social brain that result in the impairments observed in ASD.
- Brothers (1990) proposed a collection of brain regions to be responsible for social information processing and interpretation.
- Non-human primate studies: lesions of brain regions impacted social functioning.
- In Humans, different approaches used.

Social Brain Circuitry Implicated in ASD



Social Brain Circuitry Implicated in ASD



Imitation Impairments in Autism

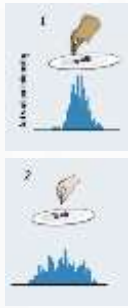
- Symbolic meaning hypothesis
  - (Baron-Cohen, 1988; Rogers et al, 1996)
- Executive functioning hypothesis
  - (Ozonoff, Pennington & Rogers, 1991; Rogers et al, 1996)
- Poor social motivation to attempt tasks
  - (Trevarthen & Aitken, 2001)
- Dyspraxia or motor dysfunction
  - (Jones & Pryor, 1985)
- Deficit in Self-Other mapping
  - (Rogers & Pennington, 1991; Williams et al, 2001)

Imitation and Self-Other Mapping

- Class of visuomotor neurons that activate when an individual is both performing an action and observing a similar action
- Potentially serve as neurological substrate for self other mapping

Mirror Neurons: non-human primates

- Mirror neurons activate during the execution AND observation of actions.
- First identified in area F5 of monkeys.
- Proposed to mediate action understanding.



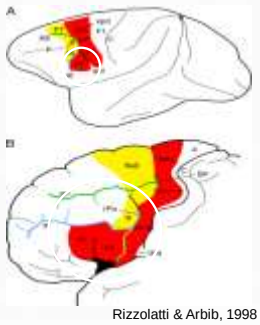
(from Ramachandran & Oberman, 2006)

Mirror Neurons: Humans

- In humans, also appears to be an execution/observation system.
- Proposed to be the mirror neuron system—limited direct evidence (e.g., fMRI and EEG).

Mirror Neuron Regions

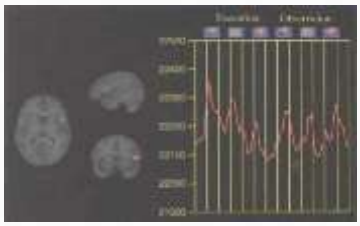
- Inferior Frontal Gyrus
- Inferior Parietal Lobe
- Related regions?
  - Superior Temporal Sulcus
  - Insula
  - Anterior Cingulate



Rizzolatti & Arbib, 1998

Mirror Neurons: Humans

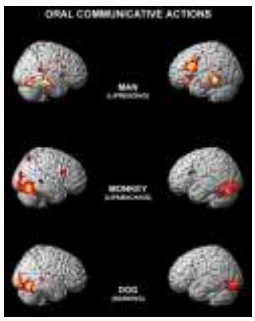
- In humans, area 44 (Inferior Frontal Gyrus) is activated when observing, executing, and imitating movement.



- Iacoboni et al, 1999

Mirror Neurons: Humans

- Only species specific actions result in MNS activation.



- Buccino et al, 2004

Mirror Neurons: Humans

- Activation occurs
  - With intransitive movements (emotional expressions)
  - Actions that are in behavioral repertoire (lip reading vs barking)
- Seems to serve a wider role.
- Provide the ability to understand others' actions and emotions through internal representation without reflection.
- Hypothesized to underlie imitation, empathy, theory of mind, metaphor, and evolution of language.
- How best to study non-invasively with individuals with autism?

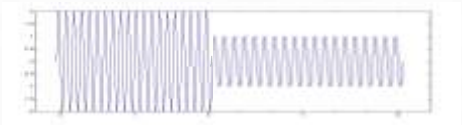
Electroencephalography and Mirror Neurons

EEG Mu rhythm reflects execution/observation matching system

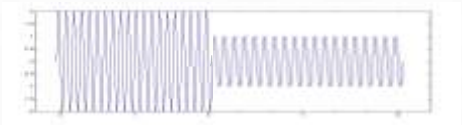
EEGs and Mirror Neurons

Mu rhythm is neuronal activity recorded from central channels over motor cortex in ~8-13 Hz frequency band

- At rest = synchronous
- Execution and observation of movement = asynchronous → reducing mu amplitude



EEGs and Mirror Neurons



Research Questions

- Observed deficits in social cognition in ASD.
- Proposed role of MNS in social cognition.

- Is there disruption of the MNS in autism?
- Is there a correlation between imitation ability and MNS functioning?

Procedure



Imitation battery

Tasks:

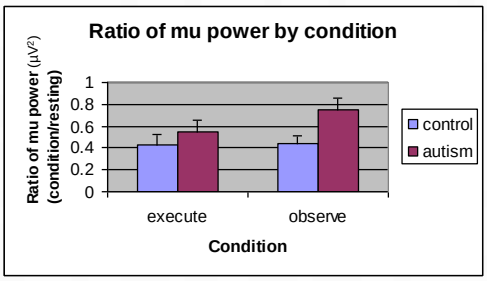
- Single step facial expressions
- Sequenced facial expressions
- Single step hand gestures
- Sequenced hand gestures
- Complex two hand gestures
- Meaningless hand movements
- Actions on objects (gentle or harsh style)

Imitation Task Models

Single Face Expression Example

Sequenced Hand Gesture Example

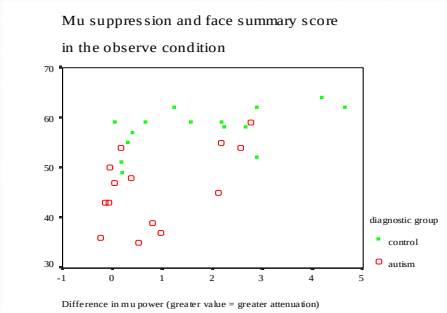
Results



Condition X Group: p<.05

Bernier et al 2007

Results

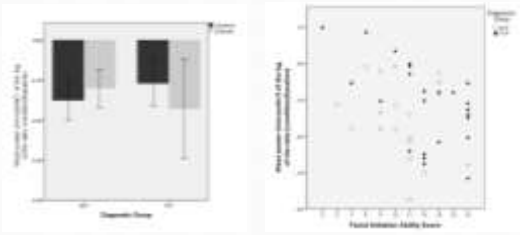


Bernier et al 2007

Conclusions

- Adults with ASD show less attenuation of the mu rhythm in response to the observation of actions. Suggests dysfunction of the mirror neuron system.
- Continued impairments in behavioral imitation skills.
- Mu wave attenuation when observing actions correlates with facial imitation skills. Suggests the EEG mu rhythm may reflect underlying neurological activity related to imitation ability.

Mu rhythm in children with ASD



Bernier et al, 2013

Conclusions

- Children with ASD on average do not show mu wave attenuation differences from typical children.
- Continued impairments in behavioral imitation skills.
- Mu wave attenuation when observing actions correlates with imitation skills. Suggests the EEG mu rhythm may reflect underlying neurological activity related to imitation ability.
- Variability in findings may be due to variability in imitation ability, not presence of ASD per se.

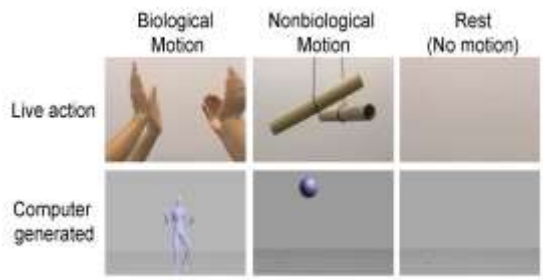
Mu rhythm in ASD

- Conflicting results in literature:
  - Between group differences:
    - Oberman et al, 2005
    - Bernier et al, 2007
    - Martineau et al, 2008
    - Oberman et al, 2008
    - Dumas et al, 2014
  - No differences between groups
    - Raymaekers et al, 2009
    - Fan et al, 2010
    - Bernier et al, 2013
    - Ruysschaert et al, 2014
- Due to Sample differences?
  - Diagnostic presentation, variability in ASD related abilities (eg, imitation), etiological contribution

Question: How does etiology contribute?

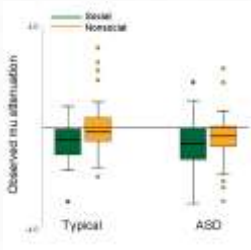
- Complete exome sequencing in ASD sample
  - All ASD individuals meet strict ADI, ADOS, clinical criteria
  - Participants in Simons Simplex Collection or SAGE Study
- Identify individuals with Likely Gene Disrupting (LGD) mutations in interactive protein network playing a contributory role in ASD
  - Re-contact and invite for visit for comprehensive follow up
  - LGD events: CHD8 (2), DSCAM, DYRK1A (3), GRIN2B, KDM6B, SCN2A, SETBP1
- Identify comparison groups: age & gender matched "idiopathic ASD" and typical group
- Hypothesis: ASD group is comprised of many etiologies each contributing distinctly to neural structure/function leading to variable findings.

Dynamic Stimuli



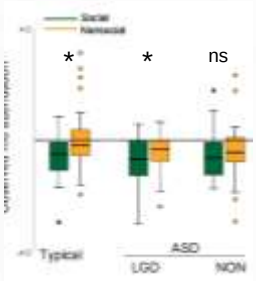
ASD vs TYP

- No group X condition interaction: $F(1,7023)=.94$, $p=.33$.
- Both exhibit more mu attenuation for social vs. nonsocial

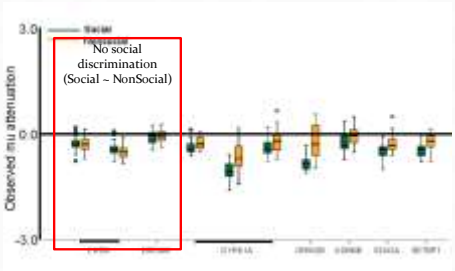


ASD vs LGD vs TYP

- Group X condition interaction: $F(1,7031)=30.93$, $p<.0001$
  - ASD-LGD and TYP groups exhibited more mu attenuation for social than nonsocial stimuli, respectively, $t=5.39$ and $t=6.09$, p's$<.0001$.
  - ASD-NON group did not exhibit mu attenuation condition differences, $t=2.49$, $p=.19$.



ASD subtypes



Conclusions

- Conflicting brain findings in ASD.
- Our results demonstrate that distinct etiologies may contribute differentially to imaging findings.
- Given, ASD is genetically heterogeneous, neglecting etiology introduces variability and obscures true relationships between genotype; protein expression; neural development, structure, function; and behavior.

State of the Science on Neuroscience of ASD

Questions?

State of the Science on Mental Health Services/Intervention Approaches

- Interventions for ASD
- What does science tell us about interventions for ASD
- How to think about interventions for ASD

Interventions for ASD

- No known cure. Focus is on educational and behavioral approaches to:
  - Improve skills in core deficit areas
  - Optimize functional outcomes:
    - Independence
    - Acceptance
    - Productivity
  - Treatments needs can be lifelong
    - Lifetime costs of caring for an individual with autism is estimated at $3.5 to 5 million.
  - Early Intervention is critical

Interventions for Autism Spectrum Disorder

Educational and Integrated Services
TEACCH
DAILY LIFE THERAPY
Technology based
VIDEO MODELING
Alternative and Augmentative Communication
PICTURE EXCHANGE COMMUNICATION SYSTEM (PECS)
COMPLEMENTARY
Animal based
CANINE COMPANION
DOLPHIN THERAPY
HIPPO THERAPY
THERAPEUTIC HORSEBACK RIDING
Spirituality based
PRAYER
ENERGY HEALING
Diets and Supplements
DIETARY INTERVENTION (E.G. GLUTEN-FREE, CASEIN-FREE DIET (GFCF DIET); YEAST FREE; KETOGENIC)
HOMEOPATHY
IMMUNOTHERAPY
VITAMIN THERAPY
SECRETIN
MELATONIN
Medical Procedures
CHELATION
HYPERBARIC OXYGEN THERAPY
ELECTROCONVULSIVE THERAPY
Relationship-based Interventions
FLOORTIME
SON-RISE TREATMENT
GENTLE TEACHING
HOLDING THERAPY
RELATIONSHIP DEVELOPMENT INTERVENTION
PEER MENTORING
Integrated Interventions
LINWOOD METHOD
SCERTS MODEL

Interventions for Autism Spectrum Disorders

PSYCHO-EDUCATIONAL
Standard
ART THERAPY
MUSIC THERAPY
PLAY THERAPY
OCCUPATIONAL THERAPY
SPEECH THERAPY
PROMPT SPEECH THERAPY
PHYSICAL THERAPY
Behavioral Interventions
EARLY START DENVER MODEL (ESDM)
PIVOTAL RESPONSE TRAINING (PRT)
DISCRETE TRIAL TRAINING (DTT)
SOCIAL STORIES
SOCIAL SKILLS TRAINING
SOCIAL GROUPS
INTEGRATED PLAY THERAPY
Educational and Integrated Services
TEACCH
DAILY LIFE THERAPY
Technology based
VIDEO MODELING
Alternative and Augmentative Communication
VISUAL SCHEDULES
PICTURE EXCHANGE COMMUNICATION SYSTEM (PECS)

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COMPLEMENTARY Continued
Physiological Interventions
CHIROPRACTIC
CRANIO-SACRAL THERAPY
ACUPUNCTURE AND ACUPRESSURE
YOGA
THERAPEUTIC MASSAGE
PATTERNING THERAPIES
MILLER METHOD
AUDITORY INTEGRATION THERAPY
RHYTHMIC ENTRAINMENT INTERVENTION
SAMONAS
SENSORY INTEGRATION THERAPY
TOMATIS METHOD
VISION THERAPY
IRLEN LENS SYSTEM
BIOFEEDBACK AND NEUROFEEDBACK
AQUATIC THERAPY
Technology based
ASSISTIVE TECHNOLOGY
TEACHTOWN
FAST FORWORD
ONLINE COMMUNITIES
Alternative and Augmentative Communication
FACILITATED COMMUNICATION
RAPID PROMPTING

PSYCHO-PHARMACOLOGICAL
PHARMACOLOGICAL INTERVENTIONS: Including: Anti-convulsants; Anti-depressants; Anti-fungals; Anti-hypertensives; Anti-psychotics; Anxiolytics; Mood stabilizers; Sedatives; Stimulants; among others.

Interventions in ASD:

- Many different models of autism intervention
- Many common terms represent multiple approaches
  - Applied Behavioral Analysis (ABA)
  - Discrete Trial Training (DTT) or Lovaas method
  - Early Intensive Behavioral Intervention (EIBI)
  - Early Start Denver Model (ESDM)
  - Pivotal Response Training (PRT)
  - Social skills training
  - Complementary and alternative medicine (CAM)
  - Pharmacological

Differences between ASD treatments

- **Scope** *(skill-focused vs. comprehensive)*
- **Targeted outcomes** *(language, behavior)*
- **Setting** *(clinic, home, school)*
- **Intensity** *(hours per week)*
- **Duration** *(weeks, months, years)*
- **Recipient** *(child, caregivers)*
- **Methodology** *(behavioral vs. developmental)*

Some comprehensive models

DTT

- Breaking a skill into smaller parts
- Teaching one subskill at a time
- Prompt fading and shaping of appropriate behaviors until mastered
- Data collection

ESDM

- Focuses on developmentally appropriate skill learning
- Particular focus on child's affect, attention, and arousal.
- Data collection

PRT

- Focus on two pivotal behaviors: motivation and responsivity to multiple cues
- The thinking is by changing these behaviors, improves everything else
- Data collection

TEACCH model

- TEACCH: Treatment and Education of Autistic and related Communication Handicapped Children
- Started by Eric Schopler at UNC in 1966.
- Physical modification of space, providing visual cues, schedules, and structure to help accommodate needs of those with ASD.

Social skills training

- Multiple settings: clinic, home, school
- Multiple formats: dyad, group
- Multiple approaches: therapist-led, peer-led
- In general:
  - Therapist/trained peer role:
    - Teach skill
    - Provide concrete reinforcement for all attempts
    - Highlight natural contingencies
  - Fun activities:
    - Developing rules that are clear, concrete and appropriate
    - Conversation Pong and Conversation Bridge
    - Emotional Charades

Complementary and Alternative Medicines (CAM)

Animal based
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AQUATIC THERAPY

Pharmacological

- Antipsychotics (risperidone): aggressive behavior
- SSRI's: anxiety, depression, OCD, aggression
- Naltrexone: hyperactivity, inattention
- Stimulants: hyperactivity, impulsiveness, inattention
- Mood Stabilizers: mood lability
- Treats issues not autism itself!

What does science tell us?

What we know about Early Intervention

- Early intensive behavioral intervention (EIBI) is very effective for some children
- A substantial proportion of children are able to attend regular education classes
- Children with higher IQs are likely to respond better
- Gains are stable into adolescence
- Early intervention is cost-effective

Elements of successful EIBI program:

- EIBI:
  - Comprehensive curriculum addressing attention, imitation, language, play, and social interaction
  - Sensitivity to normal developmental sequence
  - Highly supportive teaching strategies (based on ABA)
  - Behavioral strategies for disruptive behaviors
  - Parent involvement
  - Gradual, careful transition from highly supportive to naturalistic environment
  - Intensive intervention of about 25 hours per week for 2 years
  - Onset of intervention around 2-4 years

Review Study

- Systematic review of studies conducted between 2000 to 2010.
- Included studies published in English, with >30 participants, and relevant to ASD.
- 159 unique studies: 13 good quality, 56 fair, 90 poor.
  - Risperidone & aripiprazole improves challenging behaviors, but have side effects. Do not address social-communication impairments.
  - Early Intensive Behavioral Intervention (Lovaas model or Early Start Denver Model) improves cognition, language skills, & adaptive behavior.
  - Cognitive Behavior Therapy shows promise for social communication & challenging behaviors (e.g., anxiety).
  - Parent training augments treatment outcomes.
  - TEACCH model shows improvements in cognition.
  - Little empirical support for any other treatments (including any CAM treatments).

Warren et al, 2011

Current Status of Intervention Research in ASD

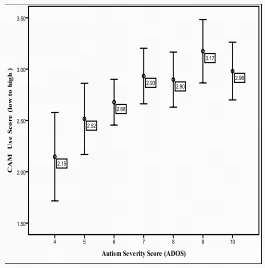
- Most intervention research has been conducted with children over the age of 3 years.
- Few randomized, controlled trials have been conducted.
- Even fewer interventions have been studied in community settings.
- There is support for both behavioral and developmental approaches.
- Most interventions are associated with improvements for SOME children. No interventions show improvement for ALL children.

Understanding individual differences in treatment response

- Key Research Questions:
  - Which interventions are more effective for which children (and which skills)?
  - What are the characteristics of treatment responders (e.g. joint attention, imitation, object play)?
  - How can we match children to the most appropriate interventions for them?

CAM Use for SSC participants

- 74% of SSC participants report using a CAM approach to address behavior.
- CAM use is related to child characteristics.
- Families twice as likely to use CAM if child also had intellectual disability

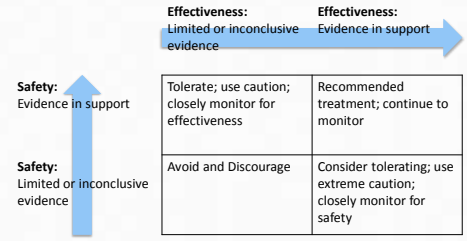


What to expect in the near future

- Parent focused intervention
- Examination/evaluation of CAM
- Use of technology in intervention
- Interventions tied to specific gene functions and disrupted proteins

Considering Intervention Options

Thinking about interventions



Cohen & Eisenberg, 2002

Questions to Ask regarding a Specific Treatment

- Will the treatment result in harm?
- Is the treatment developmentally appropriate?
- How will failure of the treatment affect the family?
- Has the treatment been validated scientifically?
- How will the treatment be integrated in to the child's current program?

Freeman, Journal of Autism & Dev. Disorders, 27(6), 1997

Guidelines for Evaluating Treatments

- Approach any treatment with hopeful skepticism
- BEWARE any treatment which states: "appropriate for every person with autism"
- BEWARE any treatment which thwarts individualization
- Be AWARE that any one treatment represents one of several options

Freeman, Journal of Autism & Dev. Disorders, 27(6), 1997

Guidelines for Evaluating Treatments

- Be AWARE any treatment should depend on independent assessment of the child which points the intervention as appropriate for the child
- Be AWARE that no treatment should be started until the proponents identify what assessments are needed to determine that it is the appropriate intervention for that child
- Be AWARE that debates over treatment can sometimes develop into superficial arguments
- Be AWARE that often new treatments have not been validated scientifically

Freeman, Journal of Autism & Dev. Disorders, 27(6), 1997

State of the Science on Intervention Approaches

Questions?