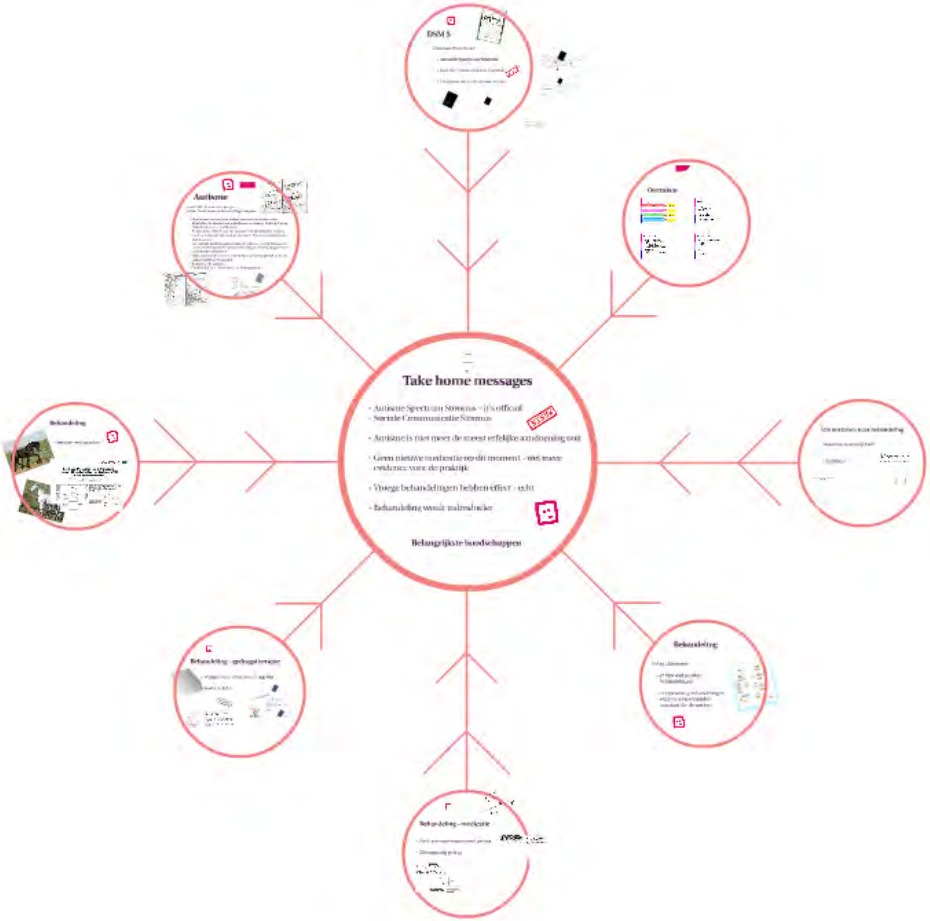
Nederlandse Vereniging voor Autisme



Autisme Netwerk Groningen

Erik Mulder

accare kinder- en jeugdpsychiatrie

REGIO Groningen-Drenthe

NVA Nederlandse Vereniging voor Autisme

7 oktober 2015, 19.30 - 21.00 uur
UMCG – Blauwe zaal – Groningen

Autisme – een update

Dr. Erik Mulder

Dr. Erik Mulder is kinder- en jeugdpsychiater en directeur behandelzaken bij Accare. Hij heeft als specialisme Autisme Spectrum Stoornissen. Thema: welke invloed hebben recente wetenschappelijke ontwikkelingen op hoe wij aankijken tegen autisme? Denk hierbij bijvoorbeeld aan erfelijkheid en invloed van de omgeving, hoe kijken we nu aan tegen de diagnoses autisme, Asperger en PDD-NOS, etc.

Toegang gratis - vooraf aanmelden verplicht – en vol is vol!

Bij aanmelding duidelijk vermelden:

- Lid NVA / Lid Balans / geen lid
- Naam; adresgegevens
- Aantal personen (maximaal 4)
- Indien van toepassing: ook naam organisatie

aanmelden: nva.lezing@gmail.com
vanaf: 5 september
sluitingsdatum: 6 oktober

Take home messages

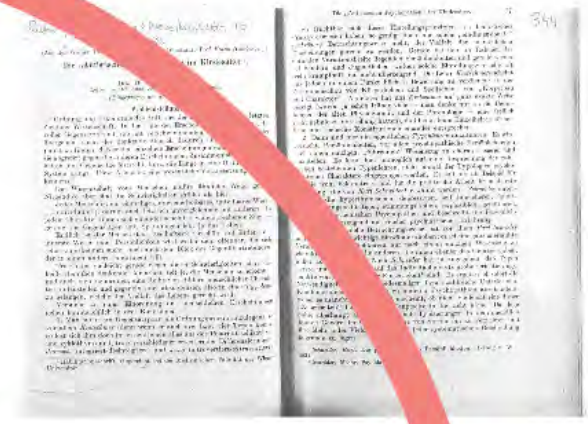
- Autisme Spectrum Stoornis - it's official
- Sociale Communicatie Stoornis

- Autisme is niet meer de meest erfelijke aandoening ooit
- Geen nieuwe medicatie op dit moment - wel meer evidence voor de praktijk
- Vroege behandelingen hebben effect - echt
- Behandeling wordt individueler

Belangrijkste boodschappen

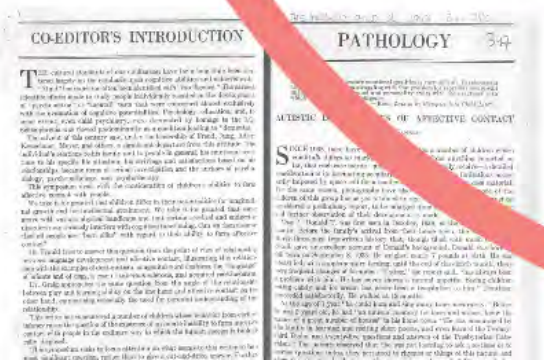
accare
Kinder- en jeugdpsychiatrie

Autisme



jaren 1940 -Kanner en Asperger
stabiel vanaf de eerste beschrijving in de jaren

- Moeite met contact met andere mensen, het contact was bijzonder, de manier van aankijken was anders, mimiek viel op (Sociale interactie problemen)
- Praten soms laat of niet, het spreken zelf opvallend qua toon, maar ook inhoud, bijzondere woorden (Taal en communicatie problemen)
- Opvallende hobbies soms, soms erg gehecht aan regelmaat en ritme, soms bijzondere manier om dingen te doen (Rigiditeit en opvallende interessen)
- Hoge pijndrempel, over- of ondergevoelig voor geluid, licht, en andere prikkels (Sensoriek)
- Komt voor in families
- Onduidelijk wat de invloed van de omgeving is.



DSM IV

299.00 Autistische Stoornis
299.80 Stoornis van Asperger
299.80 Stoornis van Rett
299.80 Desintegratiestoornis
299.80 Pervasieve ontwikkelingsstoornis, NAO (PDD-NOS)

- Kwalitatieve beperkingen in de sociale interactie
- Kwalitatieve beperkingen in de communicatie
- Beperkte, zich herhalende stereotiep patronen van gedrag, belangstelling en activiteiten

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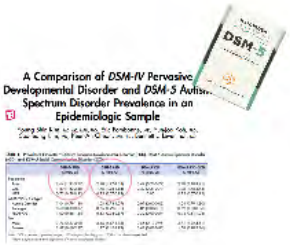
DSM 5

Uit onderzoek blijkt:

- **Autisme Spectrum Stoornis**
- Sociale Communicatie Stoornis
- Diagnose nu en diagnose straks

NIEUW

+ ADHD & ASS mag samen geclassificeerd



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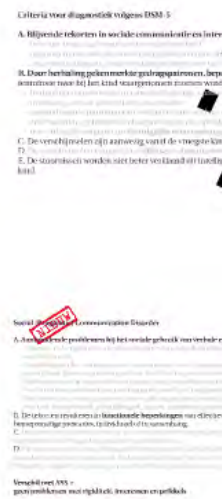

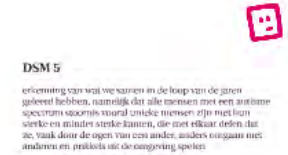

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1. A new symptom model for autism cross-validated in an independent sample.
   Boomsma A, Van Lang ND, De Jonge MV, De Bildt AA, Van Engeland H, Minderaa RB.
   J Child Psychol Psychiatry. 2008 Aug;49(8):809-16. doi: 10.1111/j.1469-7610.2008.01897.x. Epub 2008 May 19. Erratum in: J Child Psychol Psychiatry. 2009 Oct;50(10):1326.
   PMID: 18492042 [PubMed - indexed for MEDLINE]
   Related citations Remove from clipboard

2. Structural equation analysis of a hypothesised symptom model in the autism spectrum.
   van Lang ND, Boomsma A, Sytema S, de Bildt AA, Kraijer DW, Ketelaars C, Minderaa RB.
   J Child Psychol Psychiatry. 2006 Jan;47(1):37-44.
   PMID: 16405639 [PubMed - indexed for MEDLINE]
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3. How to use the ADI-R for classifying autism spectrum disorders? Psychometric properties of criteria from the literature in 1,204 Dutch children.
   de Bildt A, Oosterling IJ, van Lang ND, Kuijper S, Dekker V, Sytema S, Oerlemans AM, van Steijn DJ, Visser JC, Rommelse NN, Minderaa RB, van Engeland H, van der Gaag RJ, Buitelaar JK, de Jonge MV.
   J Autism Dev Disord. 2013 Oct;43(10):2280-94. doi: 10.1007/s10803-013-1783-1.
   PMID: 23397166 [PubMed - indexed for MEDLINE]
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4. Diagnosing autism spectrum disorders in adults: the use of Autism Diagnostic Observation Schedule (ADOS) module 4.
   Bastiaansen JA, Meffert H, Hein S, Huizinga P, Ketelaars C, Pijnenborg M, Bartels A, Minderaa R, Keysers C, de Bildt A.
   J Autism Dev Disord. 2011 Sep;41(9):1256-66. doi: 10.1007/s10803-010-1157-x.
   PMID: 21153873 [PubMed - indexed for MEDLINE] **Free PMC Article**
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5. Standardized ADOS scores: measuring severity of autism spectrum disorders in a Dutch sample.
   de Bildt A, Oosterling IJ, van Lang ND, Sytema S, Minderaa RB, van Engeland H, Roos S, Buitelaar JK, van der Gaag RJ, de Jonge MV.
   J Autism Dev Disord. 2011 Mar;41(3):311-9. doi: 10.1007/s10803-010-1057-0.
   PMID: 20617374 [PubMed - indexed for MEDLINE] **Free PMC Article**
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6. Evaluation of the ADOS revised algorithm: the applicability in 558 Dutch children and adolescents.
   de Bildt A, Sytema S, van Lang ND, Minderaa RB, van Engeland H, de Jonge MV.
   J Autism Dev Disord. 2009 Sep;39(9):1350-8. doi: 10.1007/s10803-009-0749-9. Epub 2009 May 19.
   PMID: 19452268 [PubMed - indexed for MEDLINE] **Free PMC Article**
   Related citations Remove from clipboard

Criteria voor diagnostiek volgens DSM-5

A. Blijvende tekorten in sociale communicatie en interactie, die niet verklaard

- tekorten in sociaal-emotionele wederkerigheid
- tekorten in het voor sociale omgang gebruikelijke non-verbale communicat
- tekorten in aangaan, onderhouden en begrijpen van relaties

B. Door herhaling gekenmerkte gedragspatronen, beperkte interesses en bep

tenminste twee bij het kind waargenomen moeten worden om de diagnose te k

- herhaling en stereotypie in motorisch gedrag, spreken, gebruik van voorwer
- overmatig en star vasthouden aan routines
- geritualiseerde patronen van verbaal of non-verbaal gedrag
- zeer beperkte, gefixeerde belangstellingen die opvallen door hun intensiteit
- over- of onderreageren op **zintuiglijke waarnemingen** of ongewone aandac

C. De verschijnselen zijn aanwezig vanaf de vroegste kindertijd (**maar worden s**

D. De verschijnselen veroorzaken **klinisch relevante verstoring** van het huidige

E. De stoornissen worden niet beter verklaard uit intelligentiestoornis of global

kind.

om de diagnose te kunnen stellen:

gebruik van voorwerpen

drag

door hun intensiteit en onderwerp

of ongewone aandacht voor zintuiglijk waarneemba

rtijd **(maar worden soms pas later onderkend)**

ring van het huidige functioneren in sociale of and

tiestoornis of globale ontwikkelingsachterstand en

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NIEUW

Social (Pragmatic) Communication Disorder

A. Aanhoudende problemen bij het sociale gebruik van verbale en non-verbale communi

- Tekorten in het gebruik van communicatie voor sociale doeleinden, zoals het begroeten sociale context.
- Beperking van het vermogen om communicatie aan te passen aan de context of de node speelplaats, anders spreken tegen een kind dan tegen een volwassene, en het vermijden
- Problemen om de (ongeschreven) regels voor gesprekken en het vertellen van verhalen t herformuleren wanneer iets niet begrepen wordt, en begrijpen wanneer je verbale en no
- Problemen om te begrijpen wat niet expliciet genoemd wordt (bijvoorbeeld het maken v van taal (bijvoorbeeld uitdrukkingen, humor, metaforen, meerdere betekenissen die afh

B. De tekorten resulteren in **functionele beperkingen** van effectieve communicatie, maatsc beroepsmatige prestaties, individueel of in samenhang.

C. Het begin van de symptomen is in de vroege ontwikkelingsperiode (maar beperkingen ku communicatie de beperkte capaciteit overschrijden).

D. De symptomen kunnen niet toegeschreven worden aan een andere medische of neurolo woordstructuur en grammatical, en kunnen niet beter verklaard worden door een autisme s Disorder), algehele vertraging in de ontwikkeling, of een andere psychische stoornis.

Verschil met ASS =
geen problemen met rigiditeit, interessen en prikkels

NIEUW

A Comparison of *DSM-IV* Pervasive Developmental Disorder and *DSM-5* Autism Spectrum Disorder Prevalence in an Epidemiologic Sample

Young Shin Kim, MD, MS, MPH, PhD, Eric Fombonne, MD, Yun-Joo Koh, PhD, Soo-Jeong Kim, MD, Keun-Ah Cheon, MD, PhD, Bennett L. Leventhal, MD

TABLE 1 Prevalence Estimates:[a] *DSM-IV* Pervasive Developmental Disorder (PDD), *DSM-5* Autism Spectrum Disorder (ASD), and *DSM-5* Social Communication Disorder (SCD)

	DSM-IV PDD % (95% CI)	*DSM-5* ASD % (95% CI)	*DSM-5* SCD % (95% CI)	*DSM-5* ASD+SCD % (95% CI)
Population				
Total	2.64 (1.91-3.37)	2.20 (1.77-2.64)	0.49 (0.21-0.77)	2.70 (2.18-3.21)
GPS	1.89 (1.43-2.36)	1.46 (1.06-1.85)	0.49 (0.21-0.77)	1.95 (1.46-2.43)
HPG	0.75 (0.58-0.93)	0.75 (0.57-0.92)	0.00	0.75 (0.57-0.92)
DSM-IV PDD Subtypes				
Autistic Disorder	1.04 (0.79-1.30)	1.03 (0.78-1.29)	0.01 (0.00-0.03)	1.04 (0.79-1.30)
Asperger	0.60 (0.33-0.87)	0.55 (0.29-0.80)	0.05 (0.00-0.13)	0.59 (0.33-0.86)
PDD-NOS	1.00 (0.66-1.34)	0.63 (0.38-0.87)	0.32 (0.09-0.54)	0.94 (0.61-1.28)
Sex				
Male	3.74 (2.57-4.90)	3.16 (2.47-3.85)	0.56 (0.17-0.95)	3.71 (2.92-4.51)
Female	1.47 (0.60-2.37)	1.17 (0.62-1.72)	0.42 (0.02-0.81)	1.58 (0.90-2.26)

Note: GPS = general population sample; HPG = high-probability group; NOS = not otherwise specified.
[a]*From a representative total population of Korean school-aged children.*

- ADHD & ASS mag samen geclassificeerd

DSM 5

erkenning van wat we samen in de loop van de jaren geleerd hebben, namelijk dat alle mensen met een autisme spectrum stoornis vooral unieke mensen zijn met hun sterke en minder sterke kanten, die met elkaar delen dat ze, vaak door de ogen van een ander, anders omgaan met anderen en prikkels uit de omgeving spelen

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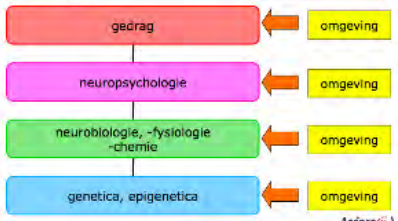
Oorzaken

Etiologie



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Historie

- Kanner
  - Aangeboren
  - Slechte opvoeding
- Bettelheim
  - Koelkastmoeder
- Rutter / Rimland
  - Familiair / genetisch
- Heden
  - Gen-omgevingsinteractie

Accare

Is autisme erfelijk?
Familie studies

- Prevalentie in de bevolking
  - Jaren 70-80 – 0.0006% (Autisme)
  - Heden:
    - 0.1-0.3% (Autisme)
    - 0.3-0.6% (Autisme Spectrum)

    Fombonne (2003) Journal of Autism and Dev Dis
- Recurrence risk in sibs
  - 2-8% (Autisme)

    Simonoff (1998) Journal of Autism and Dev Dis

Accare

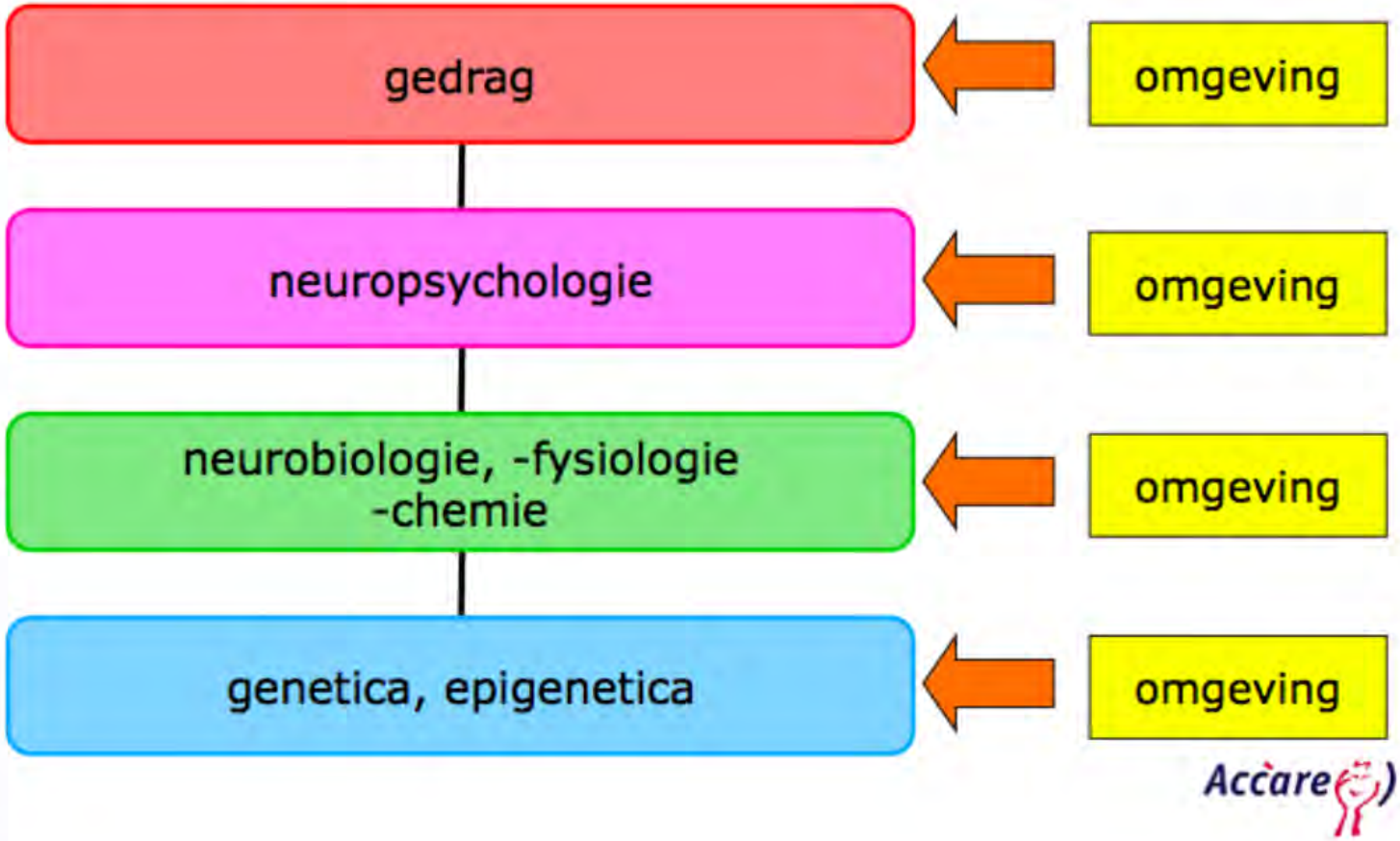
Is autisme erfelijk?
Tweeling studies / Adoptie studies

- Tweeling *Bailey et al. (1995) Psychological Medicine*
  - Monozygote tweelingen 60-92%
  - Dizygote tweelingen 0-10%
- Adoptie
  - Geen…
- Heritability – 90%

  Bailey et al. (1995)

Accare

Etiologie


gedrag
omgeving
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-chemie
omgeving
genetica, epigenetica
omgeving
Accàre

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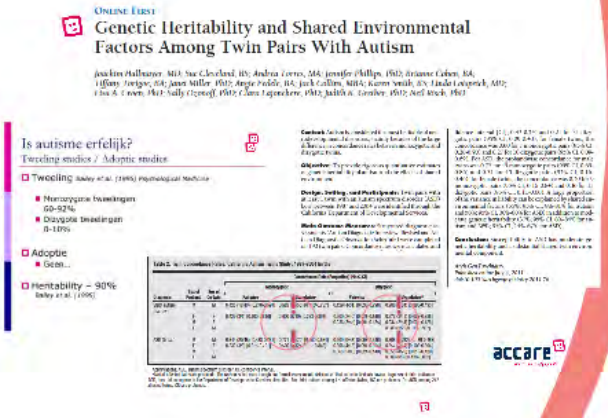
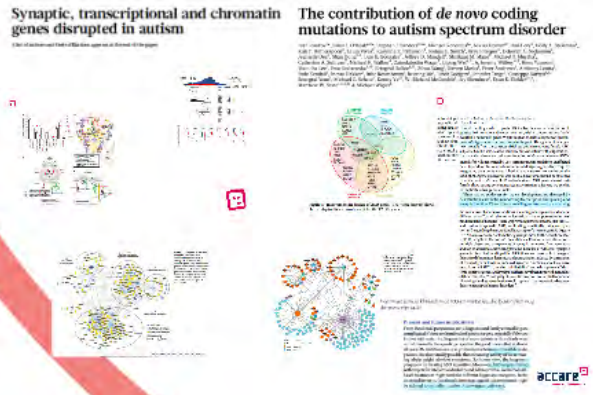
Van oorzaken naar behandeling

- omgeving en erfelijkheid
- erfelijkheid





ONLINE FIRST

Genetic Heritability and Shared Environmental Factors Among Twin Pairs With Autism

Joachim Hallmayer, MD; Sue Cleveland, BS; Andrea Torres, MA; Jennifer Phillips, PhD; Brianne Cohen, BA; Tiffany Torigoe, BA; Janet Miller, PhD; Angie Fedele, BA; Jack Collins, MBA; Karen Smith, BS; Linda Lotspeich, MD; Lisa A. Croen, PhD; Sally Ozonoff, PhD; Clara Lajonchere, PhD; Judith K. Grether, PhD; Neil Risch, PhD

Is autisme erfelijk?

Tweeling studies / Adoptie studies

- Tweeling *Bailey et al. (1995) Psychological Medicine*
  - Monozygote tweelingen 60-92%
  - Dizygote tweelingen 0-10%
- Adoptie
  - Geen...
- Heritability – 90% *Bailey et al. (1995)*

Context: Autism is considered the most heritable of neurodevelopmental disorders, mainly because of the large difference in concordance rates between monozygotic and dizygotic twins.

Objective: To provide rigorous quantitative estimates of genetic heritability of autism and the effects of shared environment.

Design, Setting, and Participants: Twin pairs with at least 1 twin with an autism spectrum disorder (ASD) born between 1987 and 2004 were identified through the California Department of Developmental Services.

Main Outcome Measures: Structured diagnostic assessments (Autism Diagnostic Interview–Revised and Autism Diagnostic Observation Schedule) were completed on 192 twin pairs. Concordance rates were calculated and fidence interval [CI], 0.42-0.74) and 0.21 for 31 dizygotic pairs (95% CI, 0.09-0.43); for female twins, the concordance was 0.60 for 7 monozygotic pairs (95% CI, 0.28-0.90) and 0.27 for 10 dizygotic pairs (95% CI, 0.09-0.69). For ASD, the probandwise concordance for male twins was 0.77 for 45 monozygotic pairs (95% CI, 0.65-0.86) and 0.31 for 45 dizygotic pairs (95% CI, 0.16-0.46); for female twins, the concordance was 0.50 for 9 monozygotic pairs (95% CI, 0.16-0.84) and 0.36 for 13 dizygotic pairs (95% CI, 0.11-0.60). A large proportion of the variance in liability can be explained by shared environmental factors (55%; 95% CI, 9%-81% for autism and 58%; 95% CI, 30%-80% for ASD) in addition to moderate genetic heritability (37%; 95% CI, 8%-84% for autism and 38%; 95% CI, 14%-67% for ASD).

Conclusion: Susceptibility to ASD has moderate genetic heritability and a substantial shared twin environmental component.

Arch Gen Psychiatry.
Published online July 4, 2011.
doi:10.1001/archgenpsychiatry.2011.76

Table 2. Twin Concordance Rates, California Autism Twins Study, 1987-2004 Births

			Concordance Rate (Proportion) [95% CI]			
			Monozygotic		Dizygotic	
Diagnosis	Sex of Proband	Sex of Co-twin	Pairwise	Probandwise[a]	Pairwise	Probandwise[a]
Strict autism (narrow)	M	M	0.425 (17/40) [0.270-0.591]	0.582 (32/55) [0.415-0.737]	0.129 (4/31) [0.036-0.298]	0.206 (7/34) [0.086-0.433]
	F	F	0.429 (3/7) [0.100-0.816]	0.600 (6/10) [0.283-0.895]	0.200 (2/10) [0.025-0.556]	0.273 (3/11) [0.093-0.685]
	M	F	...	...	0.036 (2/55) [0.004-0.125]	0.037 (2/54) [0.005-0.128]
	F	M	...	...	...	0.500 (1/2) [0.013-0.987]
ASD (broad)	M	M	0.644 (29/45) [0.488-0.781]	0.771 (54/70) [0.650-0.864]	0.200 (9/45) [0.096-0.346]	0.308 (16/52) [0.159-0.463]
	F	F	0.333 (3/9) [0.075-0.701]	0.500 (6/12) [0.157-0.843]	0.308 (4/13) [0.091-0.614]	0.357 (5/14) [0.108-0.604]
	M	F	...	...	0.063 (5/80) [0.021-0.140]	0.053 (4/76) [0.015-0.129]
	F	M	...	...	...	0.500 (3/6) [0.118-0.882]

Abbreviations: ASD, autism spectrum disorder; CI, confidence interval.
[a] Not all affected twins are probands. The reason is that even though our formal assessments determined that some individuals have a diagnosis of strict autism or ASD, they did not appear in the Department of Developmental Services client files. For strict autism, among 171 affected twins, 166 are probands. For ASD, among 242 affected twins, 230 are probands.

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Is autisme erfelijk?

Tweeling studies / Adoptie studies

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			Pairwise	
Strict autism (narrow)	M	M	0.425 (17/40) [0.270-0.591]	0.582
	F	F	0.429 (3/7) [0.100-0.816]	0.600
	M	F	…	
	F	M	…	

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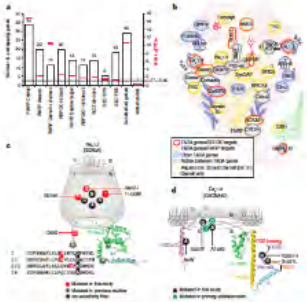
	F	F	0.429 (3/7) [0.100-0.816]	0.600 (6/10) [0.283-0.895]	0.200 (2/10) [0.025-0.556]	0.273 (3/11) [0.093-0.685]
	M	F	...	...	0.036 (2/55) [0.004-0.125]	0.037 (2/54) [0.005-0.128]
	F	M	...	...	...	0.500 (1/2) [0.013-0.987]
ASD (broad)	M	M	0.644 (29/45) [0.488-0.781]	0.771 (54/70) [0.650-0.864]	0.200 (9/45) [0.096-0.346]	0.308 (16/52) [0.159-0.463]
	F	F	0.333 (3/9) [0.075-0.701]	0.500 (6/12) [0.157-0.843]	0.308 (4/13) [0.091-0.614]	0.357 (5/14) [0.108-0.604]
	M	F	...	...	0.063 (5/80) [0.021-0.140]	0.053 (4/76) [0.015-0.129]
	F	M	...	...	...	0.500 (3/6) [0.118-0.882]

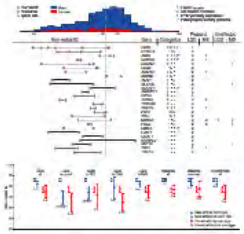
Abbreviations: ASD, autism spectrum disorder; CI, confidence interval.

[a] Not all affected twins are probands. The reason is that even though our formal assessments determined that some individuals have a diagnosis of strict autism or ASD, they did not appear in the Department of Developmental Services client files. For strict autism, among 171 affected twins, 166 are probands. For ASD, among 242 affected twins, 230 are probands.

Synaptic, transcriptional and chromatin genes disrupted in autism

A list of authors and their affiliations appears at the end of the paper





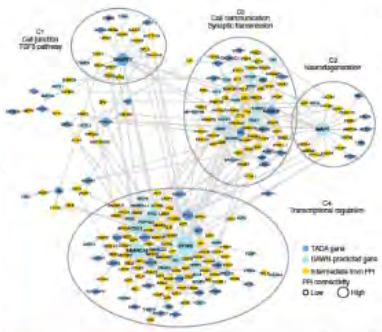


Figure 2 | ASD genes in neuronal subnetwork. Protein-protein interaction network created by seeding TADA and DAWN-predicted genes. Only intermediate genes that share known interact with at least two TADA and/or DAWN genes are included. Four mutual clusters (C1–C4) are demarcated with black ellipses. All nodes are sized on the basis of degree of connectivity.

The contribution of *de novo* coding mutations to autism spectrum disorder

Ivan Iossifov[1]*, Brian J. O'Roak[2,3]*, Stephan J. Sanders[4,5]*, Michael Ronemus[1]*, Niklas Krumm[2], Dan Levy[1], Holly A. Stessman[2], Kali T. Witherspoon[2], Laura Vives[2], Karynne E. Patterson[2], Joshua D. Smith[2], Bryan Paeper[2], Deborah A. Nickerson[2], Jeanselle Dea[4], Shan Dong[5,6], Luis E. Gonzalez[7], Jeffrey D. Mandell[4], Shrikant M. Mane[8], Michael T. Murtha[7], Catherine A. Sullivan[7], Michael F. Walker[4], Zainulabedin Waqar[7], Liping Wei[6,9], A. Jeremy Willsey[4,5], Boris Yamrom[1], Yoon-ha Lee[1], Ewa Grabowska[1,10], Ertugrul Dalkic[1,11], Zihua Wang[1], Steven Marks[1], Peter Andrews[1], Anthony Leotta[1], Jude Kendall[1], Inessa Hakker[1], Julie Rosenbaum[1], Beicong Ma[1], Linda Rodgers[1], Jennifer Troge[1], Giuseppe Narzisi[1,10], Seungtai Yoon[1], Michael C. Schatz[1], Kenny Ye[12], W. Richard McCombie[1], Jay Shendure[2], Evan E. Eichler[2,13], Matthew W. State[4,5,7,14] & Michael Wigler[1]

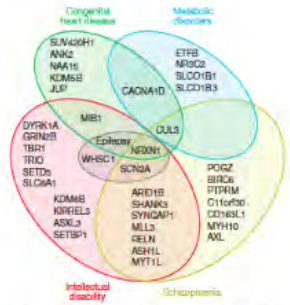


Figure 5 | Involvement in disease of ASD genes. The Venn diagram shows the overlap in disease involvement for the TADA genes.

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Conclusions

Complementing earlier reports, ASD subjects show a clear excess of *de novo* LoF mutations above expectation, with a concentration of such events in a handful of genes. While this handful has a large effect on risk, most ASD genes have a much smaller impact. This gradient emerges most notably from the contrast of risk variation in male and female ASD subjects. Unlike some earlier studies, but consistent with expectation, the data also show clear evidence for effect of *de novo* missense SNVs on risk; for risk generated by LoF variants transmitted from unaffected parents; and for the value of case-control design in gene discovery. By integrating data on *de novo*, inherited and case-control variation, the yield of ASD gene discoveries was doubled over what would be obtained from a count of *de novo* LoF variants alone. ASD genes almost uniformly show strong constraints against variation, a feature we exploit to implicate other genes in risk.

Three critical pathways for typical development are damaged by risk variation: chromatin remodelling, transcription and splicing, and synaptic function. Chromatin remodelling controls events underlying the formation of neural connections, including neurogenesis and neural differentiation[44], and relies on epigenetic marks as post-translational modifications of histones. Here we provide extensive evidence for HMGs and readers in sporadic ASD, implicating specifically lysine methylation and extending the mutational landscape of the emergent ASD gene *CHD8* to missense variants. Splicing is implicated by the enrichment of RBFOX targets in the top ASD candidates. Risk variation also affects multiple classes and components of synaptic networks, from receptors and ion channels to scaffolding proteins. Because a wide set of synaptic genes is disrupted in idiopathic ASD, it seems reasonable to suggest that altered chromatin dynamics and transcription, induced by disruption of relevant genes, leads to impaired synaptic function as well. *De novo* mutations in ASD[11–15], intellectual disability[45] and schizophrenia[46] cluster to synaptic genes, and synaptic defects have been reported in models of these disorders[46]. Integrity of synaptic function is essential for neural physiology, and its perturbation could represent the intersection between diverse neuropsychiatric disorders[47].

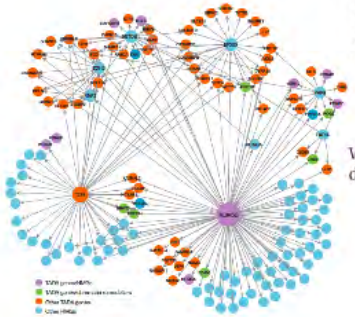

Figure 4 | Transcription regulation network of TADA genes. Edges include transcription regulation (interrupted) and flow gene (arrows, target nodes) based on the Chip-seq data, interaction among only TADAs are ignored.

Wat moet je nou klinisch met 100-en mutaties, die bovendien nog de-novo zijn ook?

Present and future implications

From the clinical perspective, early diagnosis and family counselling are complicated if there are hundreds of genetic targets, especially if few are known with certainty. Sequencing of more cohorts is thus clearly warranted. From the therapeutic perspective, the good news is that in almost all cases DN mutations occur in probands in whom a normal allele is also present. It is theoretically possible that enhancing activity of the remaining alleles might alleviate symptoms. So in our view, the long-term prognosis for treating ASD is positive. Moreover, ASD targets overlap with targets for intellectual disability and schizophrenia, so mechanism-based treatments might work for different diagnostic categories. In the intermediate term, functional clustering suggests that treatments might be tailored to a smaller number of convergent pathways.

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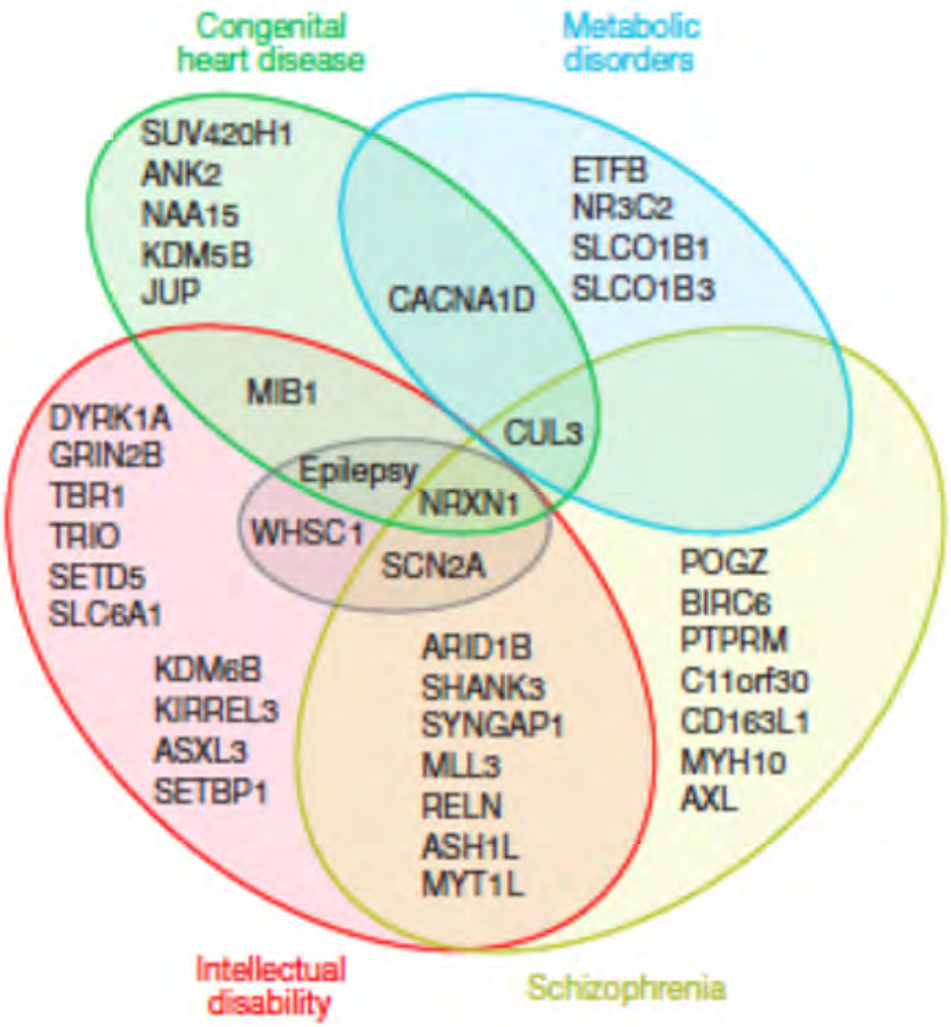

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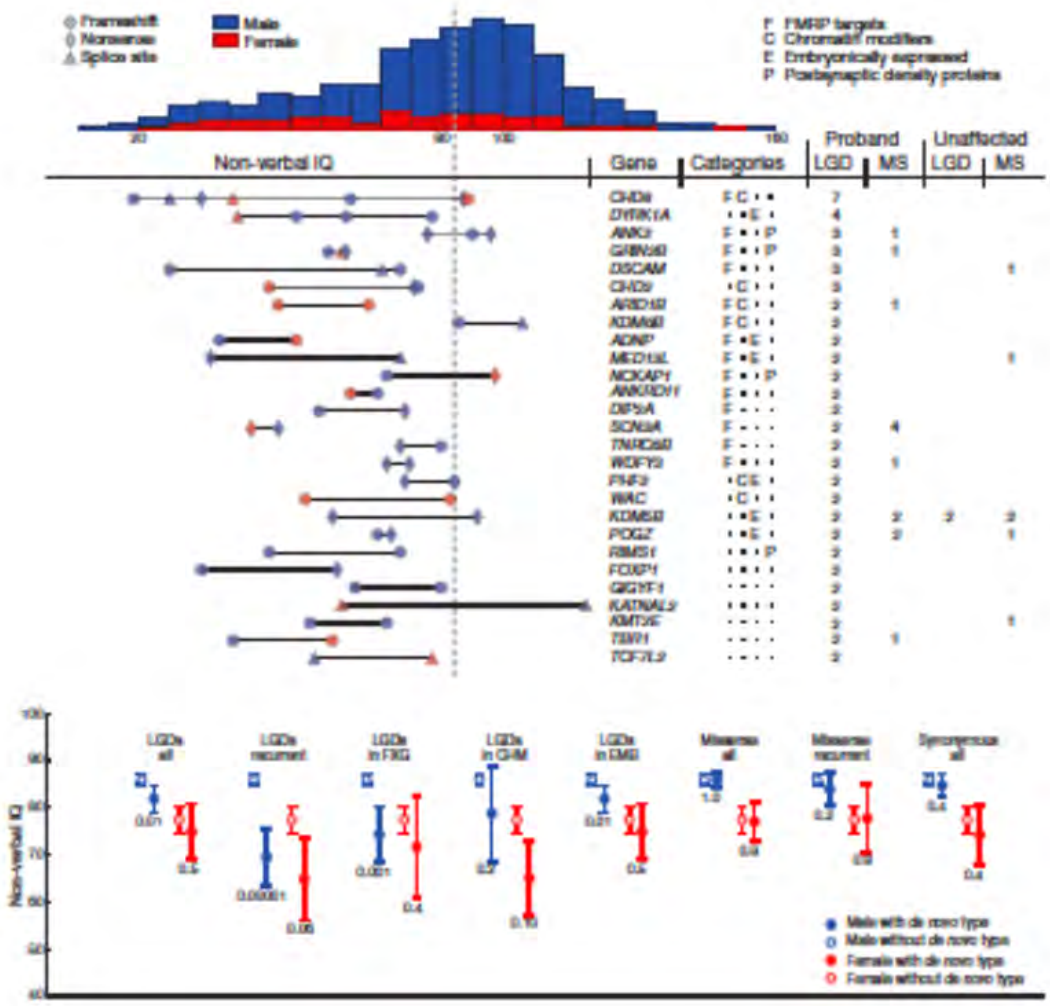
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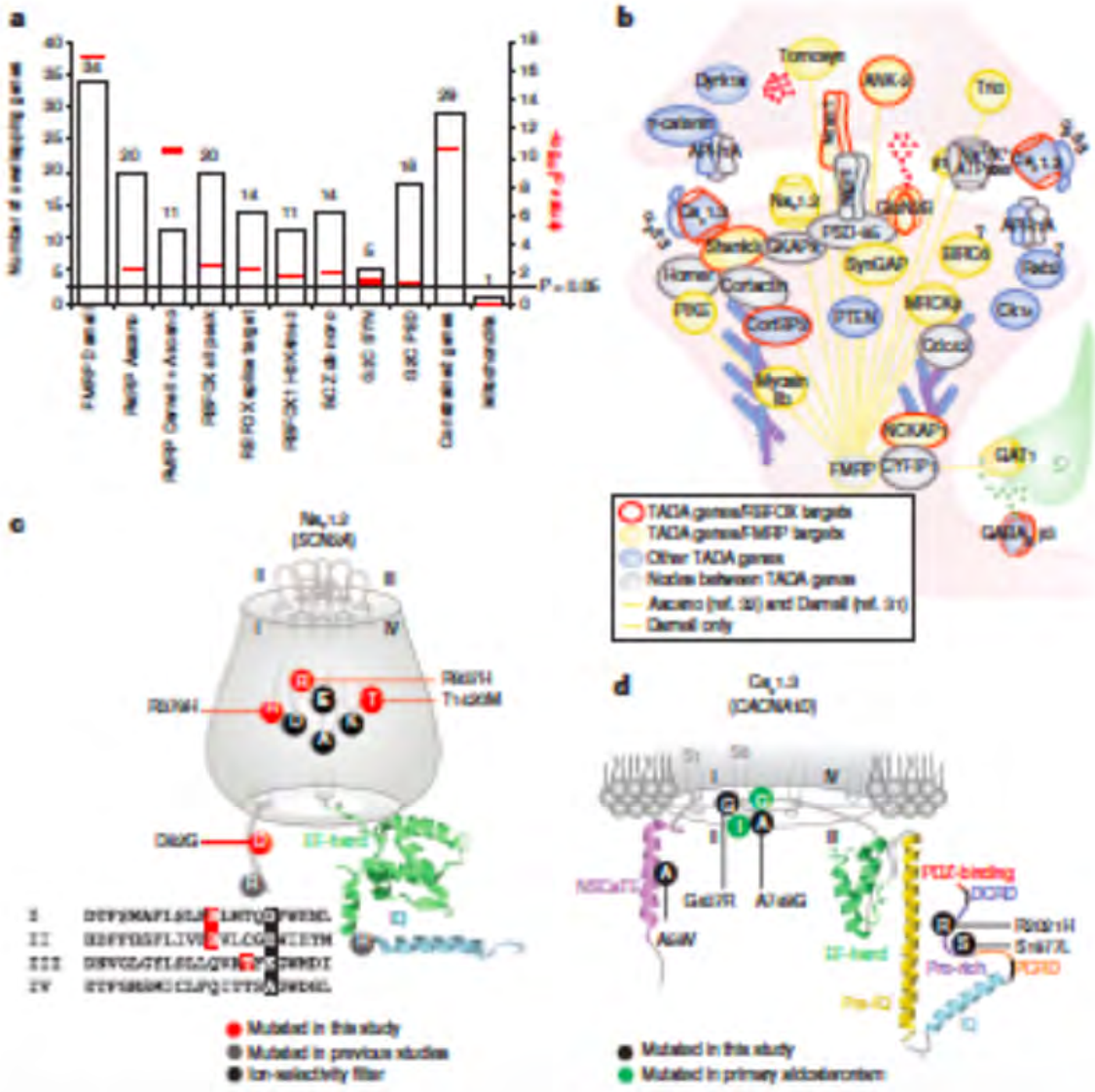
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Mutated in previous studies
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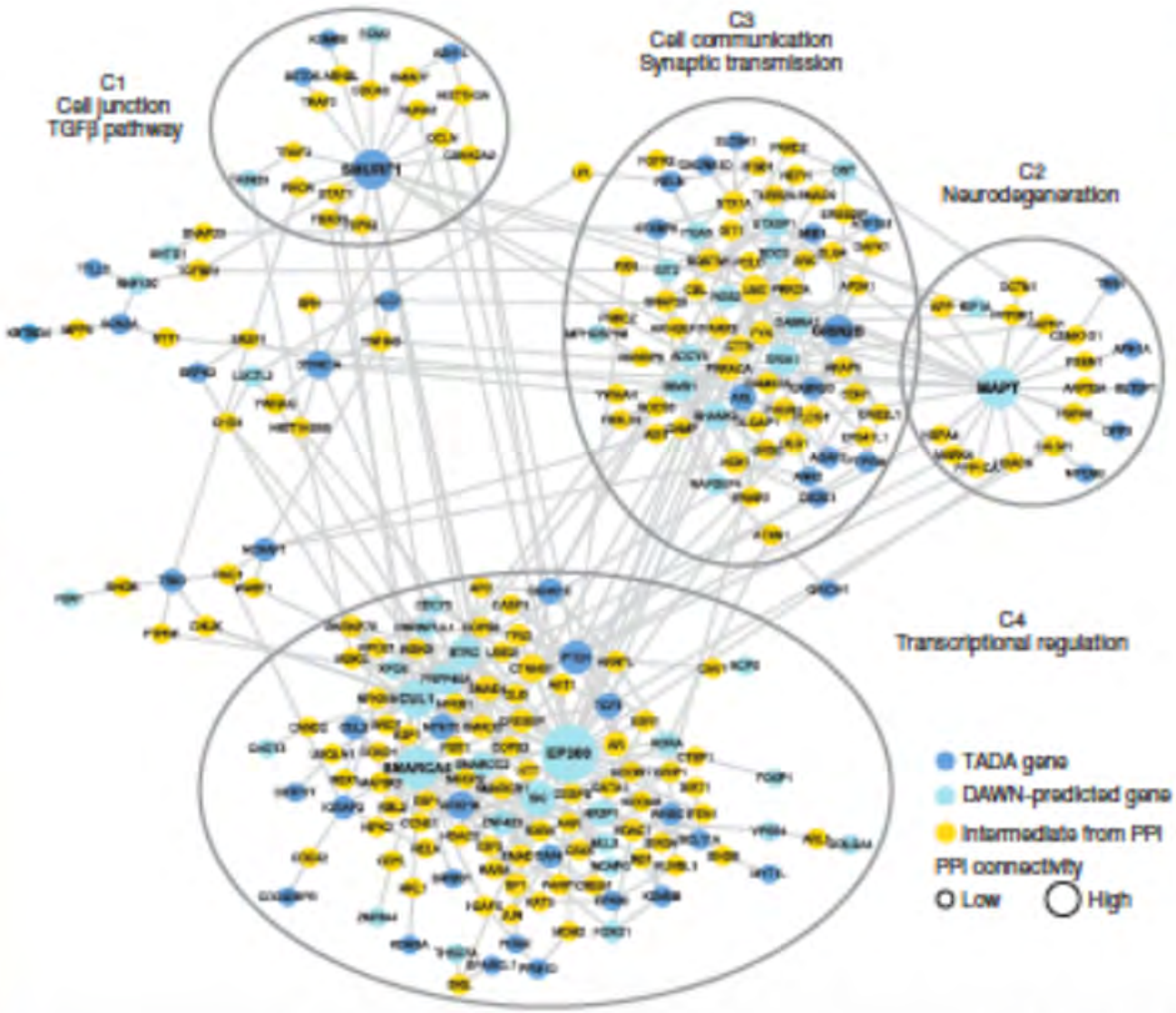
Mutated in primary aldosteronism



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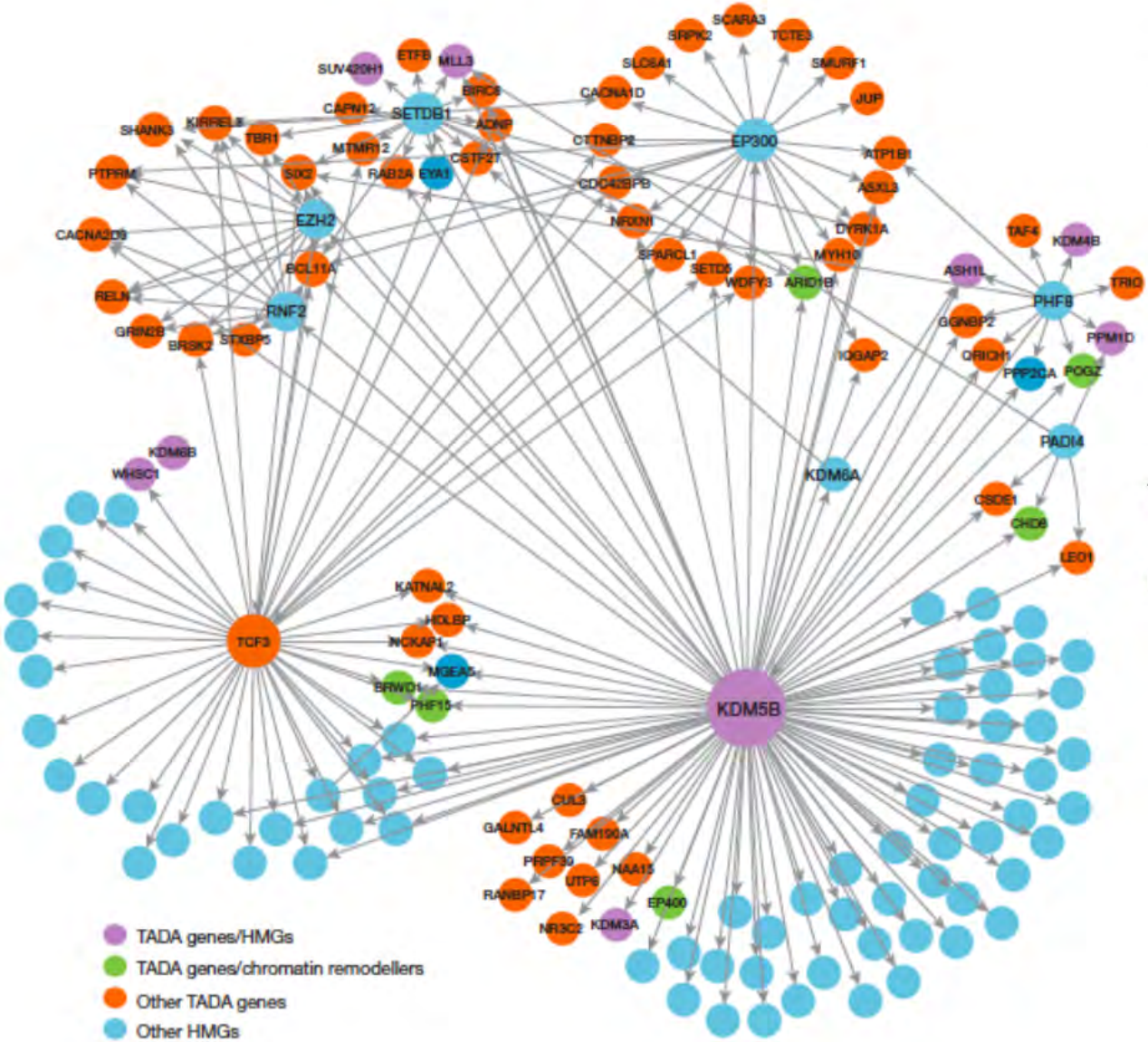

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Wat moet je nou klinis
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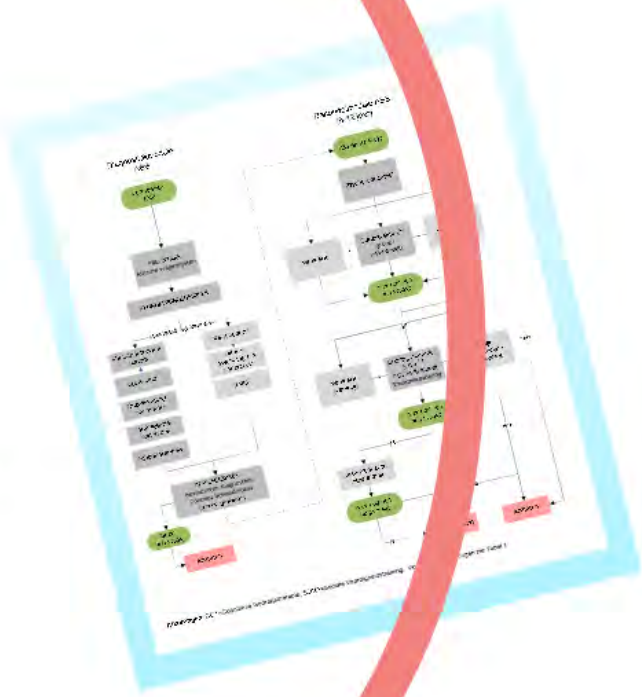
Behandeling

in het algemeen:

- er zijn veel soorten behandelingen
- er zijn weinig behandelingen waarvan onomstotelijk vaststaat dat de werken



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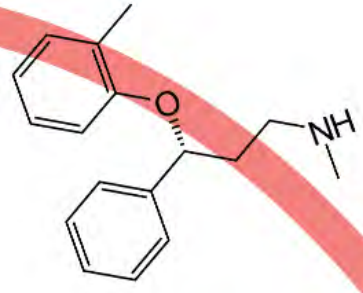
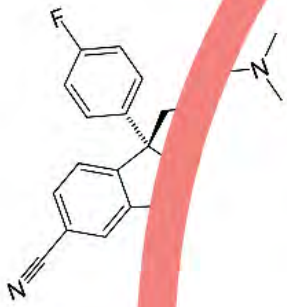
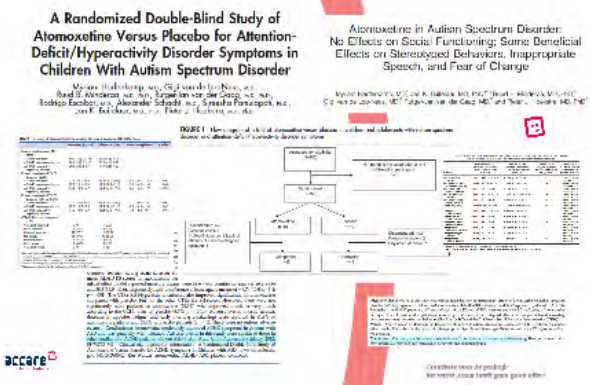
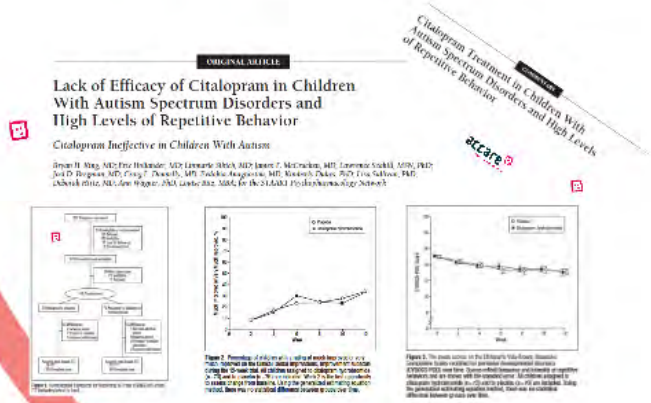




Behandeling - medicatie

- Druk en ongeconcentreerd gedrag
- Dwangmatig gedrag





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A Randomized Double-Blind Study of Atomoxetine Versus Placebo for Attention-Deficit/Hyperactivity Disorder Symptoms in Children With Autism Spectrum Disorder

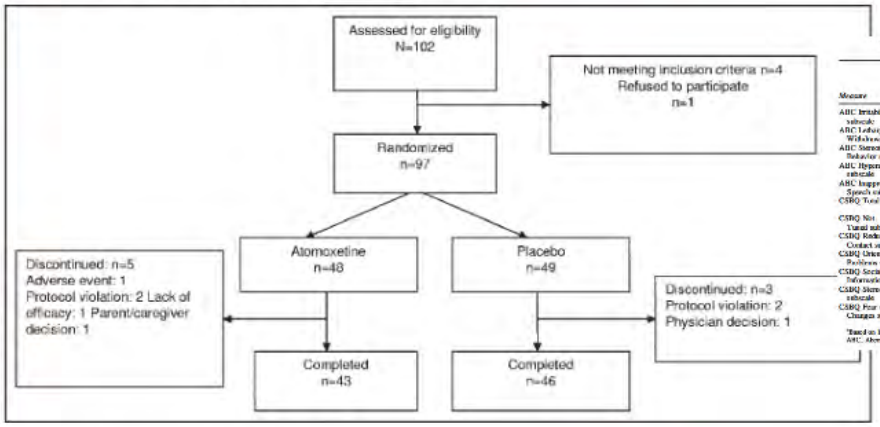
Myriam Harfterkamp, M.D., Gigi van de Loo-Neus, M.D., Ruud B. Minderaa, M.D., Ph.D., Rutger-Jan van der Gaag, M.D., Ph.D., Rodrigo Escobar, M.D., Alexander Schacht, Ph.D., Sireesha Pamulapati, Ph.D., Jan K. Buitelaar, M.D., Ph.D., Pieter J. Hoekstra, M.D., Ph.D.

TABLE 2 Summary of Attention-Deficit/Hyperactivity Disorder Rating Scale (ADHD-RS) Scores

	Atomoxetine (n = 48)	Placebo (n = 49)	Atomoxetine-placebo[a]	p Value[b]
Score at baseline, mean (SD), range				
ADHD-RS total score	40.7 (7.5), 28–54	38.6 (8.4), 20–54		
ADHD-RS inattention subscore	20.7 (3.9), 12–27	20.6 (4.6), 5–27		
ADHD-RS hyperactivity-impulsivity subscore	20.0 (5.3), 8–27	17.9 (6.1), 7–27		
LS mean at endpoint (95% CI) based on MMRM				
ADHD-RS total score	31.6 (29.2–33.9)	38.3 (36.0–40.6)	−6.7 (−10.0–−3.4)	<.001
ADHD-RS inattention subscores	17.2 (15.9–18.4)	19.9 (18.7–21.1)	−2.7 (−4.5–−1.0)	.003
ADHD-RS hyperactivity-impulsivity subscore	14.5 (13.0–15.9)	18.4 (17.0–19.7)	−3.9 (−5.9–−1.9)	<.001
LS mean at endpoint (95% CI) based on LOCF ANCOVA				
ADHD-RS total score	31.2 (28.9–33.6)	38.3 (36.0–40.5)	−7.0 (−10.3–−3.7)	<.001
ADHD-RS inattention subscores	17.0 (15.7–18.3)	19.9 (18.6–21.1)	−2.8 (−4.6–−1.0)	.002
ADHD-RS hyperactivity-impulsivity subscore	14.2 (12.8–15.7)	18.4 (17.0–19.7)	−4.1 (−6.1–−2.1)	.001
CGI-ADHD-I scores at endpoint, n (%)				
Very much improved	0 (0)	1 (2.2)		
Much improved	9 (20.9)	3 (6.5)		
Minimally improved	12 (27.9)	6 (13.0)		
No change	16 (37.2)	30 (65.2)		
Minimally worse	4 (9.3%)	3 (6.5)		
Much worse	2 (4.7%)	3 (6.5)		
Very much worse	0 (0)	0 (0)		

Note: Use mixed models for repeated measurements (MMRM), last observation carried forward (LOCF) analysis of covariance (ANCOVA), and Clinical Global Impressions of ADHD Improvement (CGI-ADHD) scores into trial of atomoxetine versus placebo in children and adolescents with autism spectrum disorder (ASD) and ADHD symptoms. CI = confidence interval.
[a]Difference in least square (LS) means: Atomoxetine and placebo.
[b]Main treatment effect for MMRM and treatment effect for ANCOVA, respectively.

FIGURE 1 Flow diagram of a trial of atomoxetine versus placebo in children and adolescents with autism spectrum disorder and attention deficit/hyperactivity disorder symptoms.



Conners Teacher Rating Scale-Revised: Sh
mean ADHD-RS scores for atomoxetine ver
mixed-effect model repeated-measure means were 31.6 (95% confidence interval 29.2–33.9) and 38.3 (36.0–40.6), respectively, with a difference in least square means of −6.7 (−10.0 to −3.4; $p < .001$). The CTRS-R:S Hyperactivity subscore also improved significantly for atomoxetine compared with placebo, but not the other CTRS-R:S subscores. However, there were not significantly more patients on atomoxetine (20.9%) who improved much, or very much according to the CGI-I, than on placebo (8.7%; $p = 0.14$). Adverse events (mostly nausea, decrease in appetite, fatigue, and early morning awakening) were reported in 81.3% of atomoxetine patients and 65.3% of placebo patients ($p > .1$). There were no serious adverse events. **Conclusions:** Atomoxetine moderately improved ADHD symptoms in patients with ASD and was generally well tolerated. Adverse events in this study were similar to those in other studies with ADHD patients without ASD. J. Am. Acad. Child Adolesc. Psychiatry, 2012; 51(7):733–741. Clinical trial registration information—A Randomized Double-Blind Study of Atomoxetine Versus Placebo for ADHD Symptoms in Children with ASD; www.clinicaltrials.gov; NCT00380692. **Key Words:** atomoxetine, ADHD, ASD, placebo-controlled

Atomoxetine in Autism Spectrum Disorder: No Effects on Social Functioning; Some Beneficial Effects on Stereotyped Behaviors, Inappropriate Speech, and Fear of Change

Myriam Harfterkamp, MD,[1] Jan K. Buitelaar, MD, PhD,[2,3] Ruud B. Minderaa, MD, PhD,[1] Gigi van de Loo-Neus, MD,[2] Rutger-Jan van der Gaag, MD,[2] and Pieter J. Hoekstra, MD, PhD[1]

TABLE 2. CHANGES IN ABC SUBSCALES AND CSBQ TOTAL SCALE AND SUBSCALE SCORES FROM BASELINE TO 8 WEEKS OF DOUBLE-BLIND TREATMENT WITH ATOMOXETINE VERSUS PLACEBO

Measure	Atomoxetine (n=48) Mean (SD) at baseline, LS mean after 8 weeks (95% CI)[a]	Placebo (n=49) Mean (SD) at baseline, LS mean after 8 weeks (95% CI)[a]	Cohen's d effect size for difference between atomoxetine and placebo[a]	Difference in LS means between atomoxetine and placebo (95% CI)[a]
ABC Irritability subscale	17.3 (9.1), 16.6 (12.7–18.4)	16.2 (9.5), 15.8 (13.8–17.3)	0.2	1.0 (−3.5–1.6) $p=0.452$
ABC Lethargy/Social Withdrawal subscale	12.5 (8.4), 11.4 (8.9–13.0)	12.5 (8.0), 11.7 (10.1–13.3)	0.0	0.3 (−2.4–3.0) $p=0.850$
ABC Stereotypic Behavior subscale	6.5 (5.1), 5.0 (2.1–5.9)	4.1 (4.5), 4.6 (3.7–5.4)	0.5	1.6 (−2.8–4.1) $p=0.064$
ABC Hyperactivity subscale	26.4 (9.3), 22.2 (18.8–23.6)	25.4 (11.3), 23.6 (21.3–27.5)	0.6	4.4 (−7.8–1.1) $p=0.010$
ABC Inappropriate Speech subscale	4.3 (3.2), 3.7 (3.3–4.3)	4.6 (3.4), 4.3 (4.0–5.1)	0.4	0.9 (−1.7–0.0) $p=0.045$
CSBQ Total scale	53.6 (16.8), 46.2 (43.7–48.5)	55.1 (15.7), 50.1 (47.5–53.1)	0.4	5.9 (−8.1–0.3) $p=0.069$
CSBQ Not Liked subscale	16.8 (6.8), 13.0 (12.0–14.0)	16.9 (5.3), 14.1 (13.3–15.1)	0.5	1.2 (−2.6–0.3) $p=0.106$
CSBQ Reduced Contact subscale	10.5 (5.5), 8.9 (8.0–9.8)	10.9 (4.4), 9.5 (8.6–10.4)	0.2	0.6 (−1.9–0.8) $p=0.251$
CSBQ Orientation Problems subscale	9.8 (2.7), 8.9 (8.2–9.7)	9.9 (2.3), 9.0 (8.3–9.8)	0.0	0.1 (−1.2–1.0) $p=0.880$
CSBQ Social Information subscale	9.4 (5.3), 8.7 (8.1–9.2)	9.6 (3.4), 9.3 (8.8–9.8)	0.3	0.6 (−1.4–0.1) $p=0.110$
CSBQ Stereotyped subscale	5.8 (4.0), 4.2 (3.4–4.9)	4.8 (3.7), 5.0 (4.3–5.7)	0.2	0.8 (−1.8–0.2) $p=0.121$
CSBQ Fear of Changes subscale	3.3 (1.9), 2.6 (2.3–3.0)	3.0 (1.9), 3.2 (2.8–3.5)	0.4	0.6 (−1.1–0.0) $p=0.033$

[a]Based on last observation carried forward analysis of covariance (ANCOVA).
ABC, Aberrant Behavior Checklist; CSBQ, Children's Social Behavior Questionnaire; LS, Least Squares.

Objective: The objective of this study was to investigate the short-term treatment effects of atomoxetine on autism spectrum disorder (ASD) symptoms in children and adolescents with both ASD and attention-deficit/hyperactivity disorder (ADHD).
Methods: A total of 97 patients 6–17 years of age, with ASD and ADHD, were treated with 1.2 mg/kg/day of atomoxetine during an 8 week double-blind placebo-controlled period. Here, we investigated effects on two parent-based secondary outcome measures, the Aberrant Behavior Checklist (ABC) and the Children's Social Behavior Questionnaire (CSBQ).
Results: After 8 weeks of double-blind treatment, atomoxetine administration was associated with significant treatment effects on the ABC subscales Hyperactivity, Inappropriate Speech, and Stereotypic Behavior, and on the CSBQ subscale Fear for Changes.
Conclusions: Our study results indicate no beneficial effects of atomoxetine on social functioning. However, atomoxetine may ameliorate restricted and stereotyped behaviors and communication.

Conclusie voor de praktijk:
het werkt, maar heeft geen groot effect

accare

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FIGURE 1 Flow diagram of a trial of atomoxetine versus placebo in children and adolescents with autism spectrum disorder and attention deficit/hyperactivity disorder symptoms.

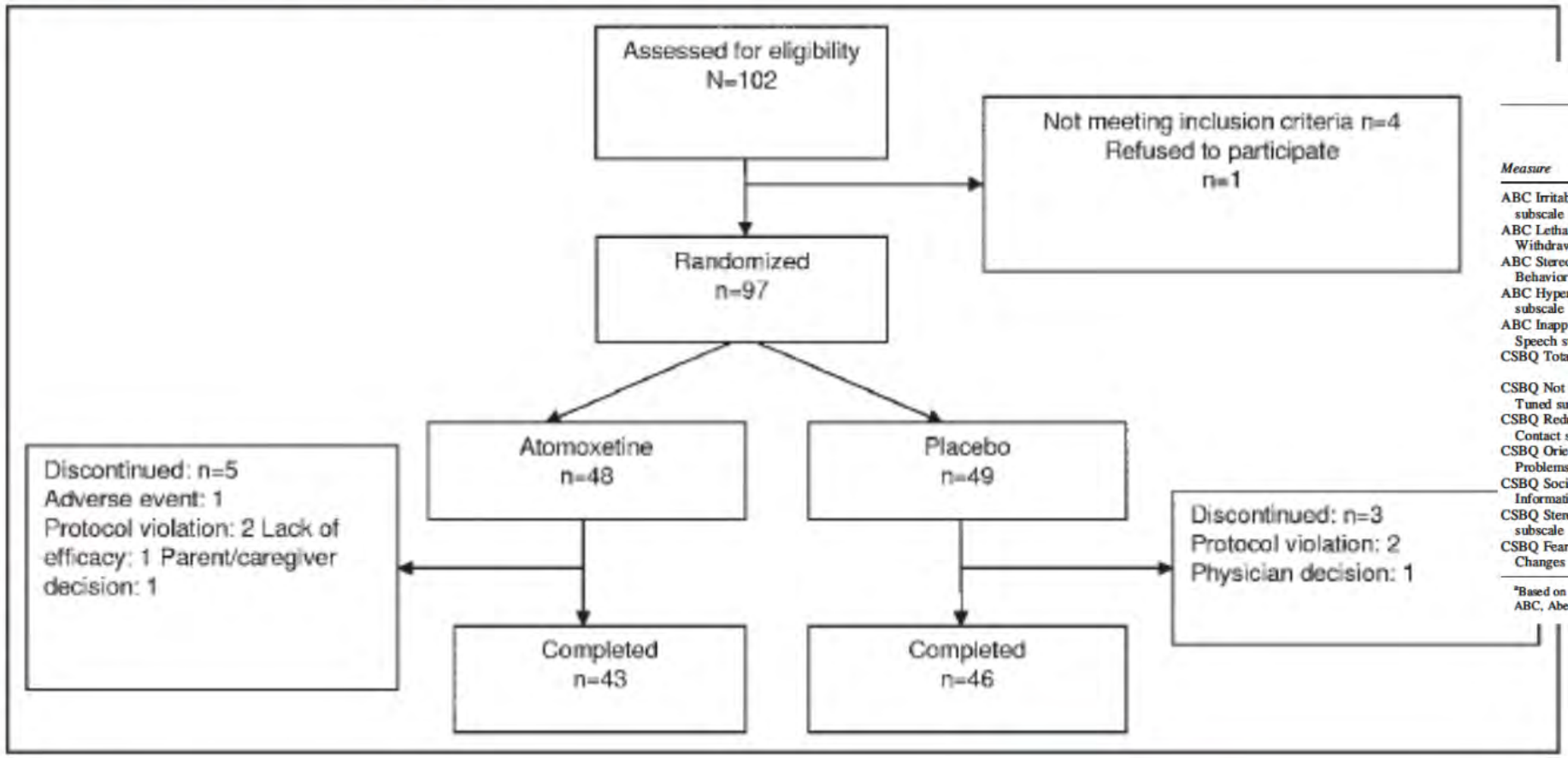

TABLE 2. CHANGES
TO 8 WE

Measure	At Mea LS
ABC Irritability subscale	17.3 (9
ABC Lethargy/Social Withdrawal subscale	12.5 (8
ABC Stereotypic Behavior subscale	6.5 (5
ABC Hyperactivity subscale	28.4 (9
ABC Inappropriate Speech subscale	4.7 (3
CSBQ Total scale	53.6 (1
CSBQ Not Tuned subscale	14.8 (4
CSBQ Reduced Contact subscale	10.5 (5
CSBQ Orientation Problems subscale	9.8 (2
CSBQ Social Information subscale	9.4 (3
CSBQ Stereotyped subscale	5.8 (4
CSBQ Fear of Changes subscale	3.3 (1

[a]Based on last observation carried
ABC, Aberrant Behavior Checklist;

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re means were 31.6 (95% confidence interval 29.2–33.9)
h a difference in least square means of −6.7 (–10.0 to –3.4;
ity subscore also improved significantly for atomoxetine
e other CTRS-R:S subscores. However, there were not
noxetine (20.9%) who improved much, or very much
cebo (8.7%; p = 0.14). Adverse events (mostly nausea,
early morning awakening) were reported in 81.3% of

Objective: The objective of this study was to investigate the short-ter
disorder (ASD) symptoms in children and adolescents with both AS
Methods: A total of 97 patients 6–17 years of age, with ASD and A
during an 8 week double-blind placebo-controlled period. Here, w
outcome measures, the Aberrant Behavior Checklist (ABC) and the

TABLE 2 Summary of Attention-Deficit/Hyperactivity Disorder Rating Scale (ADHD-RS) Scores

	Atomoxetine (n = 48)	Placebo (n = 49)	Atomoxetine-placebo[a]	p Value[b]
Score at baseline, mean (SD), range				
ADHD-RS total score	40.7 (7.5), 28–54	38.6 (8.4), 20–54		
ADHD-RS inattention subscore	20.7 (3.9), 12–27	20.6 (4.6), 5–27		
ADHD-RS hyperactivity-impulsivity subscore	20.0 (5.3), 8–27	17.9 (6.1), 7–27		
LS mean at endpoint (95% CI) based on MMRM				
ADHD-RS total score	31.6 (29.2–33.9)	38.3 (36.0–40.6)	−6.7 (−10.0–−3.4)	<.001
ADHD-RS inattention subscore	17.2 (15.9–18.4)	19.9 (18.7–21.1)	−2.7 (−4.5–−1.0)	.003
ADHD-RS hyperactivity-impulsivity subscore	14.5 (13.0–15.9)	18.4 (17.0–19.7)	−3.9 (−5.9–−1.9)	<.001
LS mean at endpoint (95% CI) based on LOCF ANCOVA				
ADHD-RS total score	31.2 (28.9–33.6)	38.3 (36.0–40.5)	−7.0 (−10.3–−3.7)	<.001
ADHD-RS inattention subscore	17.0 (15.7–18.3)	19.9 (18.6–21.1)	−2.8 (−4.6–−1.0)	.002
ADHD-RS hyperactivity-impulsivity subscore	14.2 (12.8–15.7)	18.4 (17.0–19.7)	−4.1 (−6.1–−2.1)	.001
CGI-ADHD-I scores at endpoint, n (%)				
Very much improved	0 (0)	1 (2.2)		
Much improved	9 (20.9)	3 (6.5)		
Minimally improved	12 (27.9)	6 (13.0)		

ANCOVA, respectively.

Conners Teacher Rating Scale-Revised: Sh
mean ADHD-RS scores for atomoxetine ver
mixed-effect model repeated-measure means were 31.6 (95% confidence interval 29.2–33.9) and 38.3 (36.0–40.6), respectively, with a difference in least square means of −6.7 (–10.0 to –3.4; $p < .001$). The CTRS-R:S Hyperactivity subscore also improved significantly for atomoxetine compared with placebo, but not the other CTRS-R:S subscores. However, there were not significantly more patients on atomoxetine (20.9%) who improved much, or very much according to the CGI-I, than on placebo (8.7%; $p = 0.14$). Adverse events (mostly nausea, decrease in appetite, fatigue, and early morning awakening) were reported in 81.3% of atomoxetine patients and 65.3% of placebo patients ($p > .1$). There were no serious adverse events. **Conclusions:** Atomoxetine moderately improved ADHD symptoms in patients with ASD and was generally well tolerated. Adverse events in this study were similar to those in other studies with ADHD patients without ASD. J. Am. Acad. Child Adolesc. Psychiatry, 2012; 51(7):733–741. Clinical trial registration information—A Randomized Double-Blind Study of Atomoxetine Versus Placebo for ADHD Symptoms in Children with ASD; www.clinicaltrials.gov; NCT00380692. **Key Words:** atomoxetine, ADHD, ASD, placebo-controlled

TABLE 2. CHANGES IN ABC SUBSCALES AND CSBQ TOTAL SCALE AND SUBSCALE SCORES FROM BASELINE TO 8 WEEKS OF DOUBLE-BLIND TREATMENT WITH ATOMOXETINE VERSUS PLACEBO

Measure	*Atomoxetine (n=48) Mean (SD) at baseline, LS mean after 8 weeks (95% CI)*[a]	*Placebo (n=49) Mean (SD) at baseline, LS mean after 8 weeks (95% CI)*[a]	*Cohen's d effect size for difference between atomoxetine and placebo*[a]	*Difference in LS means between atomoxetine and placebo (95% CI)*[a]
ABC Irritability subscale	17.3 (9.1), 14.6 (12.7–16.4)	16.2 (9.5), 15.6 (13.8–17.3)	0.2	1.0 (−3.5–1.6) $p=0.452$
ABC Lethargy/Social Withdrawal subscale	12.5 (8.4), 11.4 (9.9–13.0)	12.5 (8.0), 11.7 (10.1–13.2)	0.0	0.2 (−2.4–2.0) $p=0.850$
ABC Stereotypic Behavior subscale	6.5 (5.1), 3.0 (2.1–3.9)	4.1 (4.5), 4.6 (3.7–5.4)	0.5	1.6 (−2.8–0.3) $p=0.014$
ABC Hyperactivity subscale	28.4 (9.3), 21.2 (18.8–23.6)	25.4 (11.5), 25.6 (23.3–27.9)	0.6	4.4 (−7.8–1.1) $p=0.010$
ABC Inappropriate Speech subscale	4.7 (3.2), 3.7 (3.3–4.3)	4.6 (3.4), 4.5 (4.0–5.1)	0.4	0.9 (−1.7–0.0) $p=0.045$
CSBQ Total scale	53.6 (14.8), 46.2 (43.2–49.2)	53.1 (15.7), 50.1 (47.2–53.1)	0.4	3.9 (−8.1–0.3) $p=0.069$
CSBQ Not Tuned subscale	14.8 (4.8), 13.0 (12.0–14.0)	14.9 (5.3), 14.1 (13.2–15.1)	0.3	1.2 (−2.6–0.3) $p=0.106$
CSBQ Reduced Contact subscale	10.5 (5.5), 8.9 (8.0–9.8)	10.9 (4.4), 9.5 (8.6–10.4)	0.2	0.6 (−1.9–0.6) $p=0.331$
CSBQ Orientation Problems subscale	9.8 (2.7), 8.9 (8.2–9.7)	9.9 (3.5), 9.0 (8.3–9.8)	0.0	0.1 (−1.2–1.0) $p=0.889$
CSBQ Social Information subscale	9.4 (3.3), 8.7 (8.1–9.2)	9.6 (3.4), 9.3 (8.8–9.8)	0.3	0.6 (−1.4–0.1) $p=0.110$
CSBQ Stereotyped subscale	5.8 (4.0), 4.2 (3.4–4.9)	4.8 (3.7), 5.0 (4.3–5.7)	0.3	0.8 (−1.8–0.2) $p=0.123$
CSBQ Fear of Changes subscale	3.3 (1.9), 2.6 (2.3–3.0)	3.0 (1.9), 3.2 (2.8–3.5)	0.4	0.6 (−1.1–0.0) $p=0.035$

[a]Based on last observation carried forward analysis of covariance (ANCOVA)
ABC, Aberrant Behavior Checklist; CSBQ, Children's Social Behavior Questionnaire; LS, Least Squares.

Objective: The objective of this study was to investigate the short-term treatment effects of atomoxetine on autism spectrum disorder (ASD) symptoms in children and adolescents with both ASD and attention-deficit/hyperactivity disorder (ADHD).
Methods: A total of 97 patients 6–17 years of age, with ASD and ADHD, were treated with 1.2 mg/kg/day of atomoxetine during an 8 week double-blind placebo-controlled period. Here, we investigated effects on two parent-based secondary outcome measures, the Aberrant Behavior Checklist (ABC) and the Children's Social Behavior Questionnaire (CSBQ).
Results: After 8 weeks of double-blind treatment, atomoxetine administration was associated with significant treatment effects on the ABC subscales Hyperactivity, Inappropriate Speech, and Stereotypic Behavior, and on the CSBQ subscale Fear for Changes.
Conclusions: Our study results indicate no beneficial effects of atomoxetine on social functioning. However, atomoxetine may ameliorate restricted and stereotyped behaviors and communication.

Conclusie voor de praktijk:
het werkt, maar heeft geen groot effect

COMMENTARY

Citalopram Treatment in Children With Autism Spectrum Disorders and High Levels of Repetitive Behavior

ORIGINAL ARTICLE

Lack of Efficacy of Citalopram in Children With Autism Spectrum Disorders and High Levels of Repetitive Behavior

Citalopram Ineffective in Children With Autism

Bryan H. King, MD; Eric Hollander, MD; Linmarie Sikich, MD; James T. McCracken, MD; Lawrence Scahill, MSN, PhD; Joel D. Bregman, MD; Craig L. Donnelly, MD; Evdokia Anagnostou, MD; Kimberly Dukes, PhD; Lisa Sullivan, PhD; Deborah Hirtz, MD; Ann Wagner, PhD; Louise Ritz, MBA; for the STAART Psychopharmacology Network

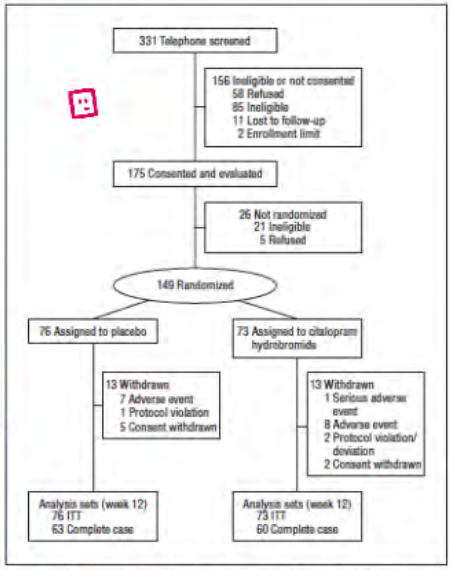


Figure 1. Consolidated Standards for Reporting of Trials (CONSORT) chart. ITT indicates intent to treat.

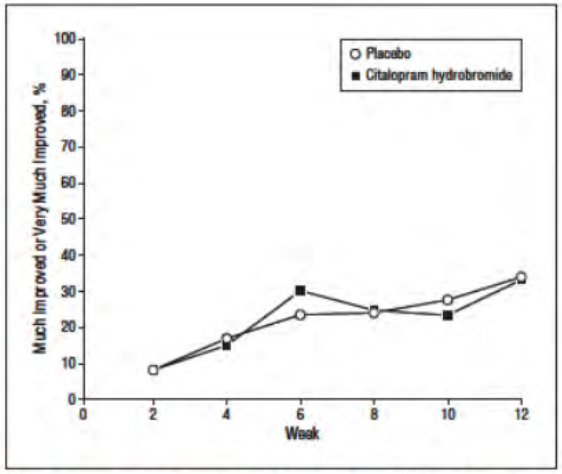


Figure 2. Percentage of children with a rating of much improved or very much improved on the Clinical Global Impressions, Improvement subscale during the 12-week trial. All children assigned to citalopram hydrobromide (n=73) and to placebo (n=76) are included. Week 2 is the first opportunity to assess change from baseline. Using the generalized estimating equation method, there was no statistical difference between groups over time.

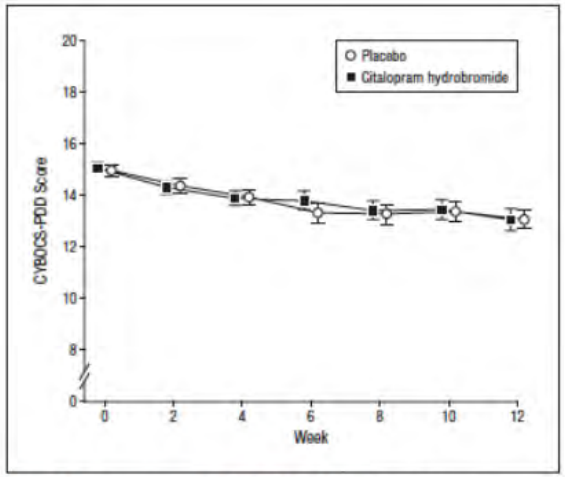
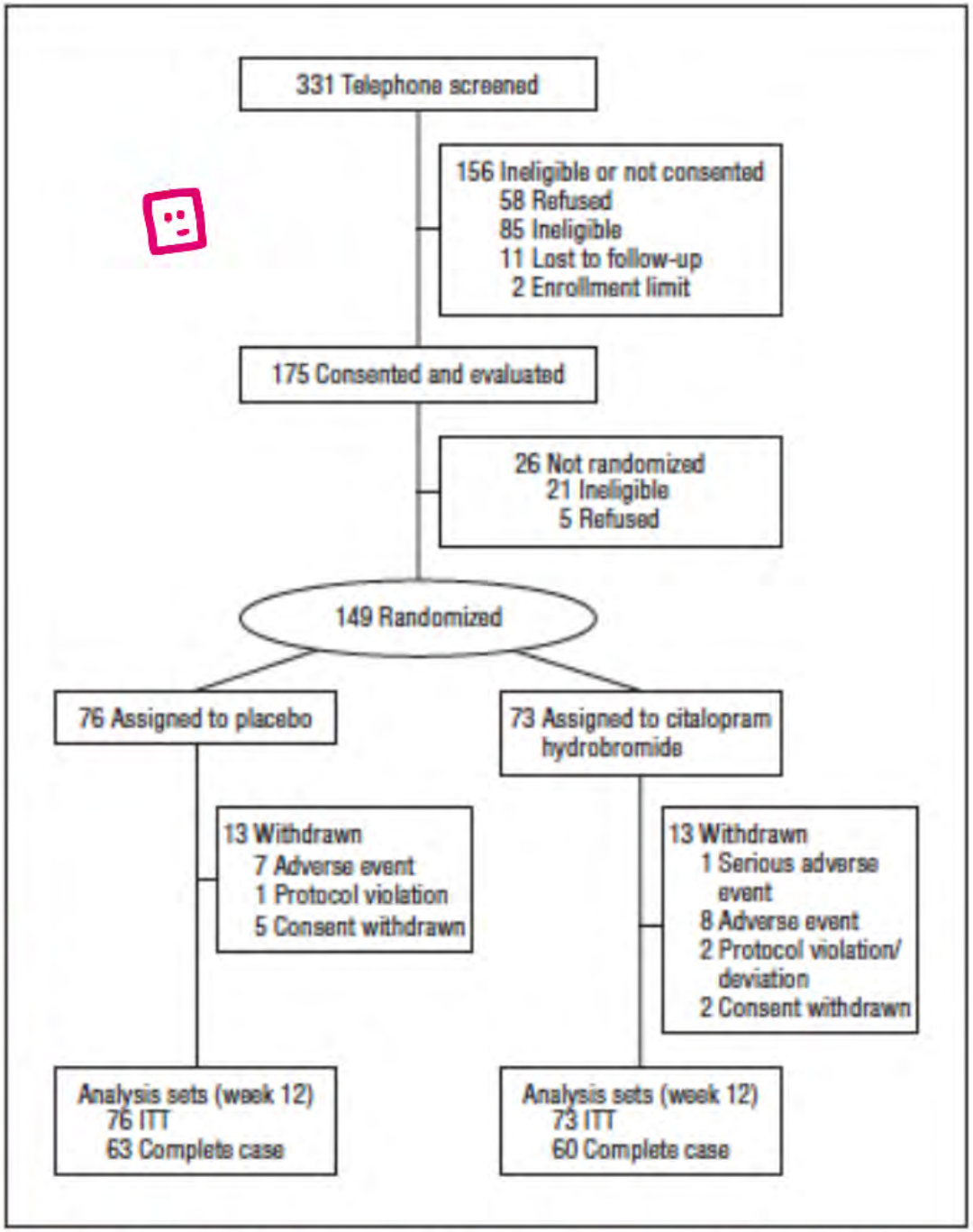


Figure 3. The mean scores on the Children's Yale-Brown Obsessive Compulsive Scales modified for pervasive developmental disorders (CYBOCS-PDD) over time. Scores reflect frequency and intensity of repetitive behaviors and are shown with the standard error. All children assigned to citalopram hydrobromide (n=73) and to placebo (n=76) are included. Using the generalized estimating equation method, there was no statistical difference between groups over time.



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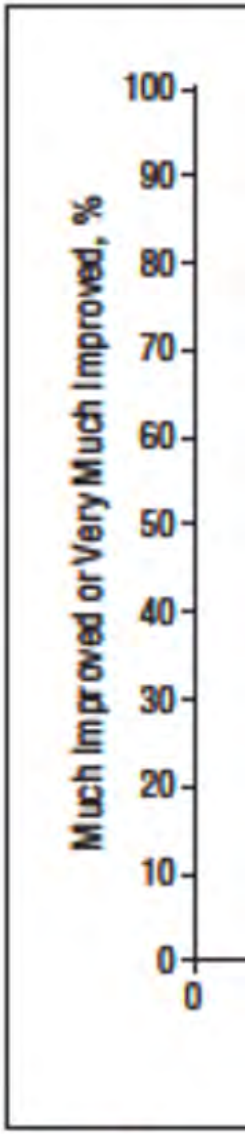

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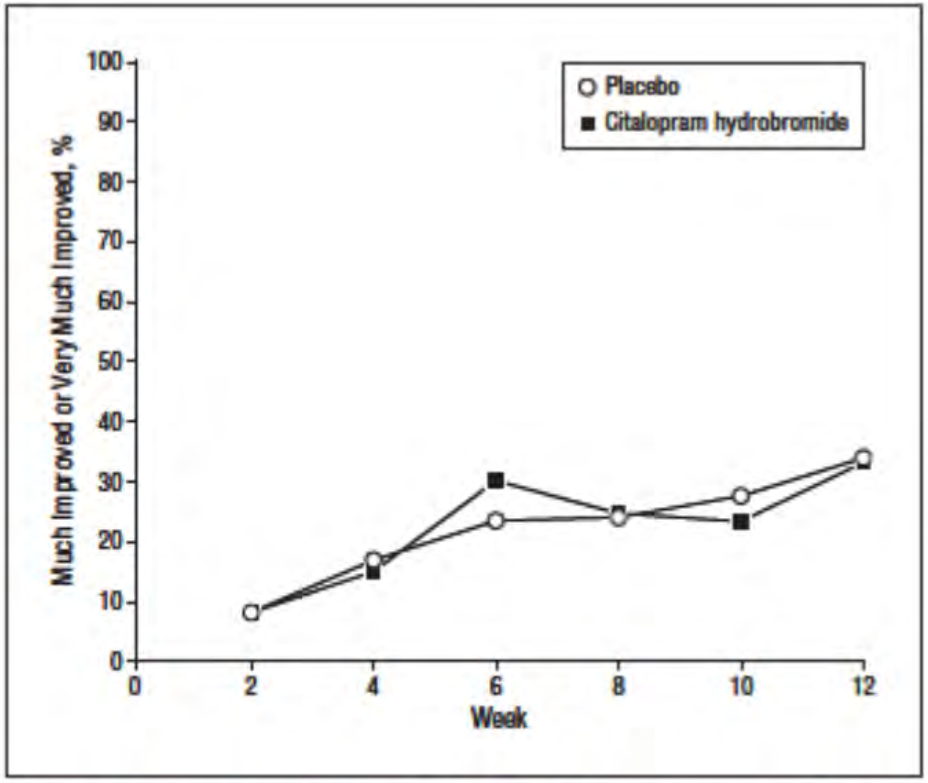

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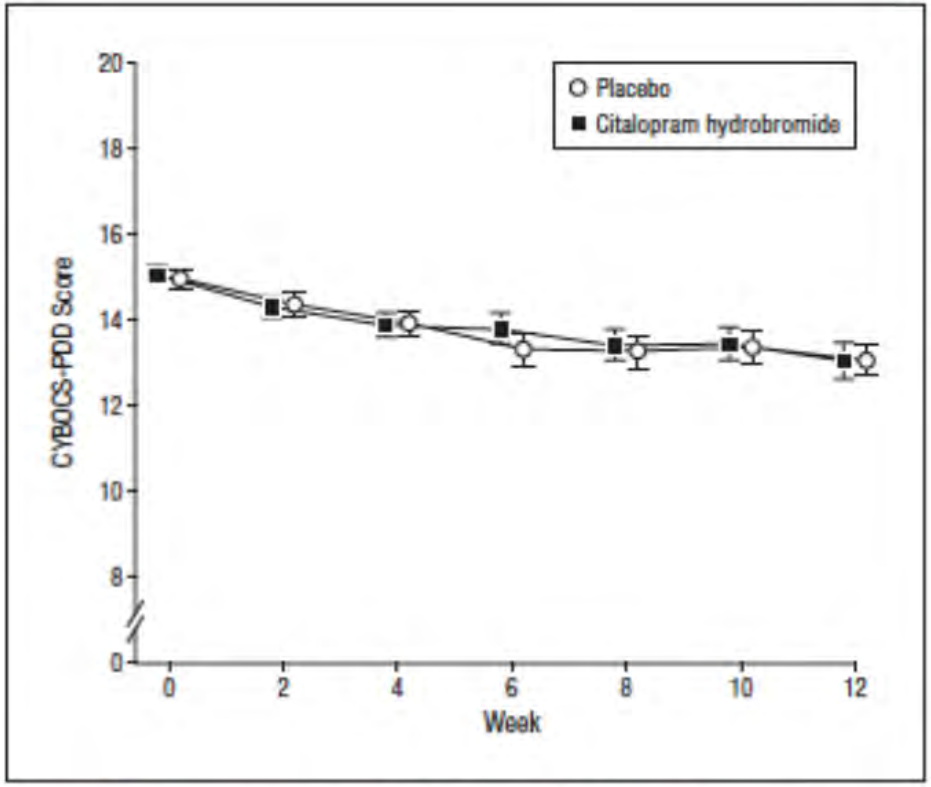

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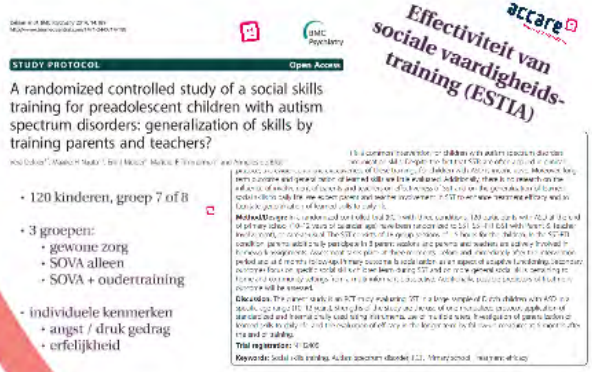
Behandeling - gedragstherapie

- vroege interventie wordt regulier
- werkt SOVA?



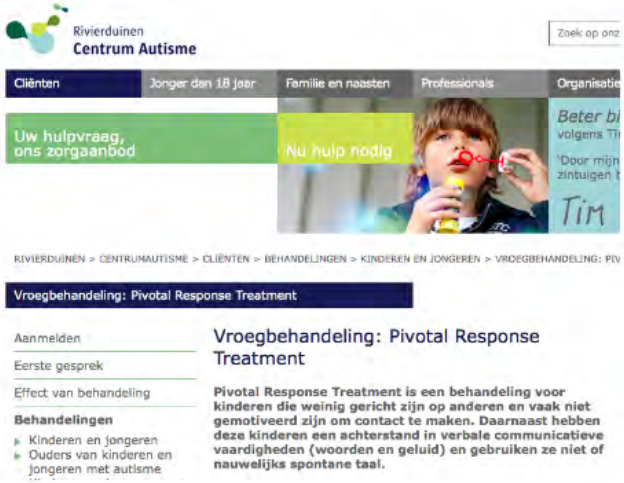
Effectiviteit van sociale vaardigheids-training (ESTIA)

accare
kinder- en jeugdpsychiatrie

Development and Psychopathology 25 (2013), 1455–1472
© Cambridge University Press 2013
doi:10.1017/S0954579413000710

A quarter century of progress on the early detection and trea
of autism spectrum disorder

GERALDINE DAWSON[a] AND RAPHAEL BERNIER[b]
[a]*Duke University; and* [b]*University of Washington, Seattle*

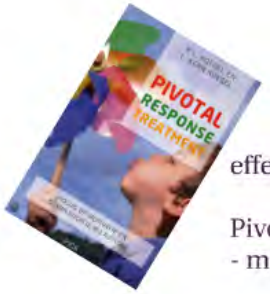


Lovaas/ABA/Discrete trail

Pivotal Response Treatment

- behandeling waar het kind is
- ouders betrekken
- keuze taken en materiaal met de kinderen zelf
- variatie
- minder uren

accare



effect op communicatie

Pivotal response treatment
- meest bewezen behandeling bij jonge kinderen met ASS

Effectiviteit van sociale vaardigheids-training (ESTIA)

accare
kinder- en jeugdpsychiatrie

Dekker *et al. BMC Psychiatry* 2014, **14**:189
http://www.biomedcentral.com/1471-244X/14/189

BMC Psychiatry

STUDY PROTOCOL Open Access

A randomized controlled study of a social skills training for preadolescent children with autism spectrum disorders: generalization of skills by training parents and teachers?

Vera Dekker[1*], Maaike H Nauta[1,2], Erik J Mulder[1], Marieke E Timmerman[2] and Annelies de Bildt[1]

- 120 kinderen, groep 7 of 8
- 3 groepen:
  - gewone zorg
  - SOVA alleen
  - SOVA + oudertraining
- individuele kenmerken
  - angst / druk gedrag
  - erfelijkheid

) is a common intervention for children with autism spectrum disorders nmunication skills. Despite the fact that SSTs are often applied in clinical practice, the evidence for the effectiveness of these trainings for children with ASD is inconclusive. Moreover, long term outcome and generalization of learned skills are little evaluated. Additionally, there is no research on the influence of involvement of parents and teachers on effectiveness of SST and on the generalization of learned social skills to daily life. We expect parent and teacher involvement in SST to enhance treatment efficacy and to facilitate generalization of learned skills to daily life.

Method/Design: In a randomized controlled trial (RCT) with three conditions, 120 participants with ASD at the end of primary school (10–12 years of calendar age) have been randomized to SST, SST-PTI (SST with Parent & Teacher Involvement), or care-as-usual. The SST consists of 18 group sessions of 1.5 hours for the children. In the SST-PTI condition, parents additionally participate in 8 parent sessions and parents and teachers are actively involved in homework assignments. Assessment takes place at three moments: before and immediately after the intervention period and at 6 months follow-up. Primary outcome is socialization, as an aspect of adaptive functioning. Secondary outcomes focus on specific social skills children learn during SST and on more general social skills pertaining to home and community settings from a multi-informant perspective. Additionally, possible predictors of treatment outcome will be assessed.

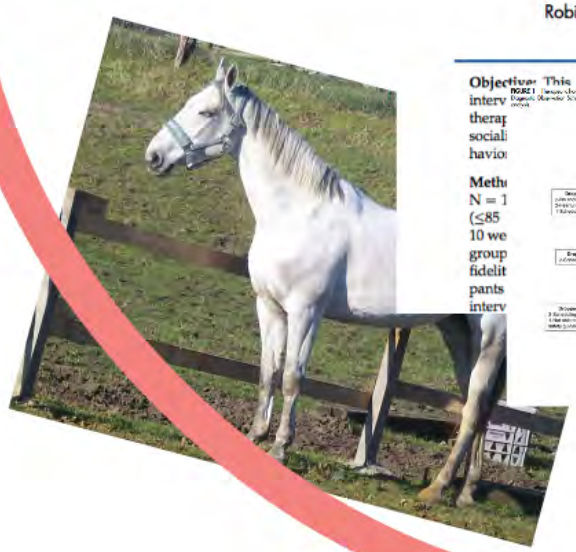
Discussion: The current study is an RCT study evaluating SST in a large sample of Dutch children with ASD in a specific age range (10–12 years). Strengths of the study are the use of one manualized protocol, application of standardized and internationally used rating instruments, use of multiple raters, investigation of generalization of learned skills to daily life, and the evaluation of efficacy in the longer term by follow-up measures at 6 months after the end of training.

Trial registration: NTR2405

Keywords: Social skills training, Autism spectrum disorder, RCT, Primary school, Treatment efficacy

Behandeling

- therapie met paarden



NEW RESEARCH

Randomized Controlled Trial of Therapeutic Horseback Riding in Children and Adolescents With Autism Spectrum Disorder

Robin L. Gabriels, PsyD, Zhaoxing Pan, PhD, Briar Dechant, BS, John A. Agnew, PhD, Natalie Brim, BA, Gary Mesibov, PhD

Objective: This study expands previous equine-assisted
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Meth ars;
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intervention. Significant improvements in the THR group were also o
cognition (p = .05; ES
(p = .003; ES = 0.62
words (p = .01; ES
ES = 0.54) spoken
sample. Sensitivity a
per protocol analyse

Conclusion: This is
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equine-assisted interv

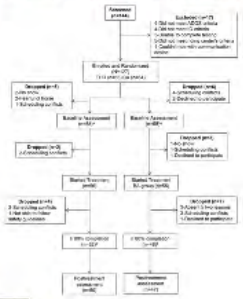
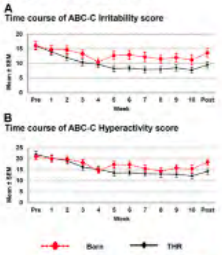


FIGURE 2 Time course of Aberrant Behavior Checklist–Community (ABC-C) subscale scores during the course of the 10-week therapeutic horseback riding (THR) intervention. Note: SEM = standard error of the mean.



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Randomized Controlled Trial of Therapeutic Horseback Riding in Children and Adolescents With Autism Spectrum Disorder

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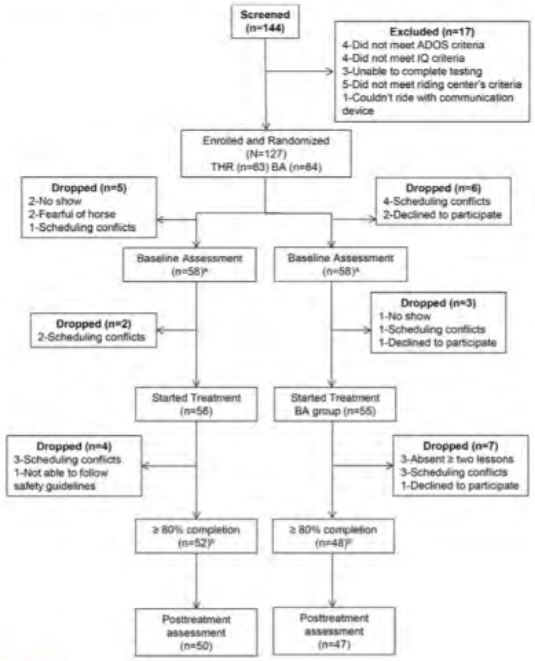
Objective: This study expands previous equine-assisted interv s of therap tion, social be- havio SD).

Meth N = 1 ears; (≤85 ores 10 we for group ntrol fidelit The pants ticipants interv post- ions

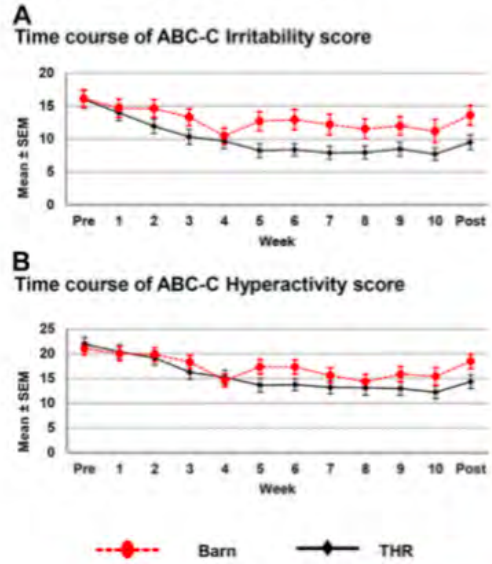
FIGURE 1 Therapeutic horseback riding (THR) and autism spectrum disorder (ASD): participant flowchart. Note: ADOS = Autism Diagnostic Observation Schedule; BA = barn activity; ITT = intent to treat. [a] Eligible for ITT analysis; [b] Eligible for per protocol analysis.



intervention. Significant improvements in the THR group were also o cognition (p = .05; ES (p = .003; ES = 0.63 words (p = .01; ES ES = 0.54) spoken sample. Sensitivity a per protocol analyses

Conclusion: This is controlled trial demor population, and find equine-assisted interv

FIGURE 2 Time course of Aberrant Behavior Checklist–Community (ABC-C) subscale scores during the course of the 10-week therapeutic horseback riding (THR) intervention. Note: SEM = standard error of the mean.



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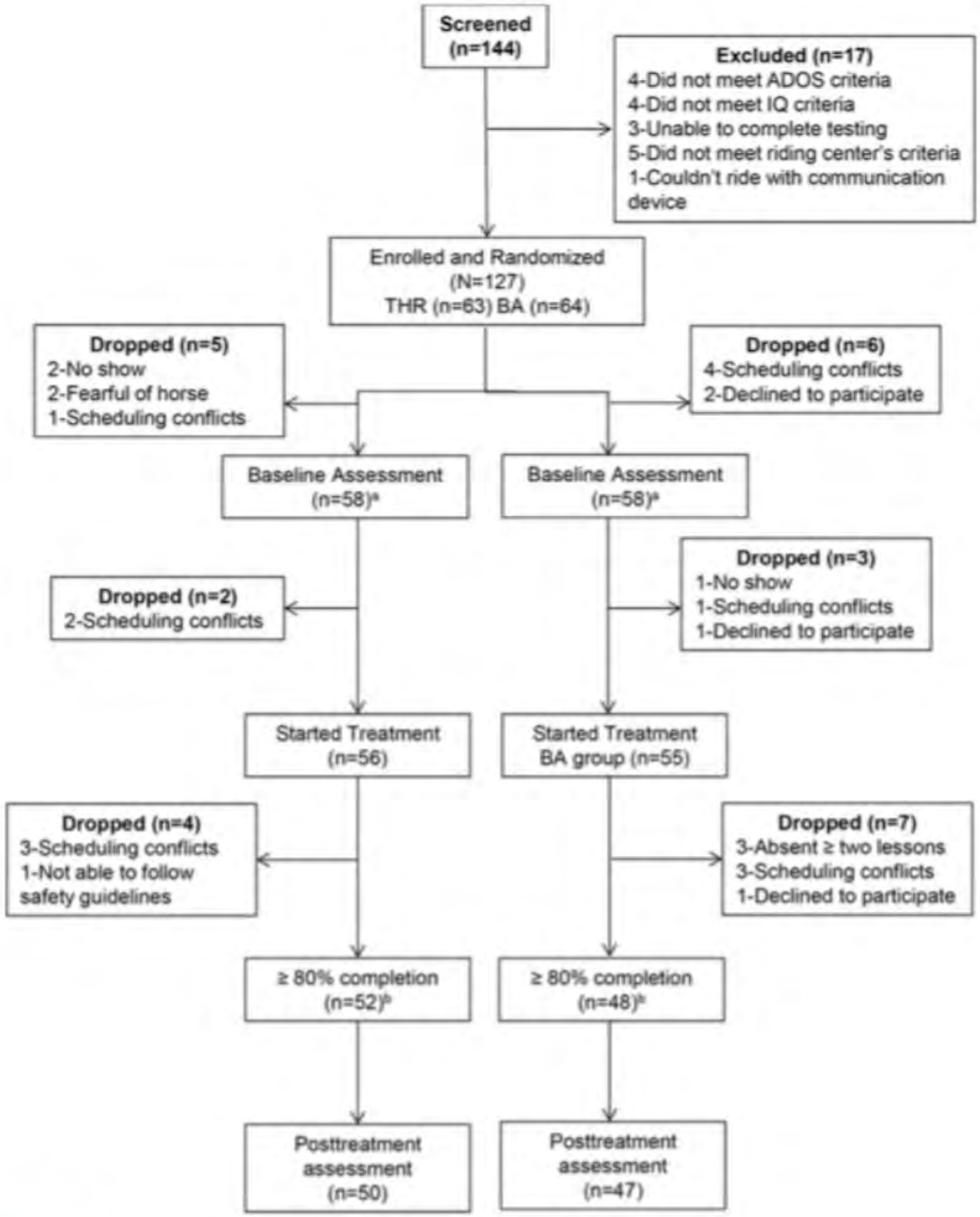

; ES = 0.63
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10 week therapeutic horseback riding (THR) intervention.
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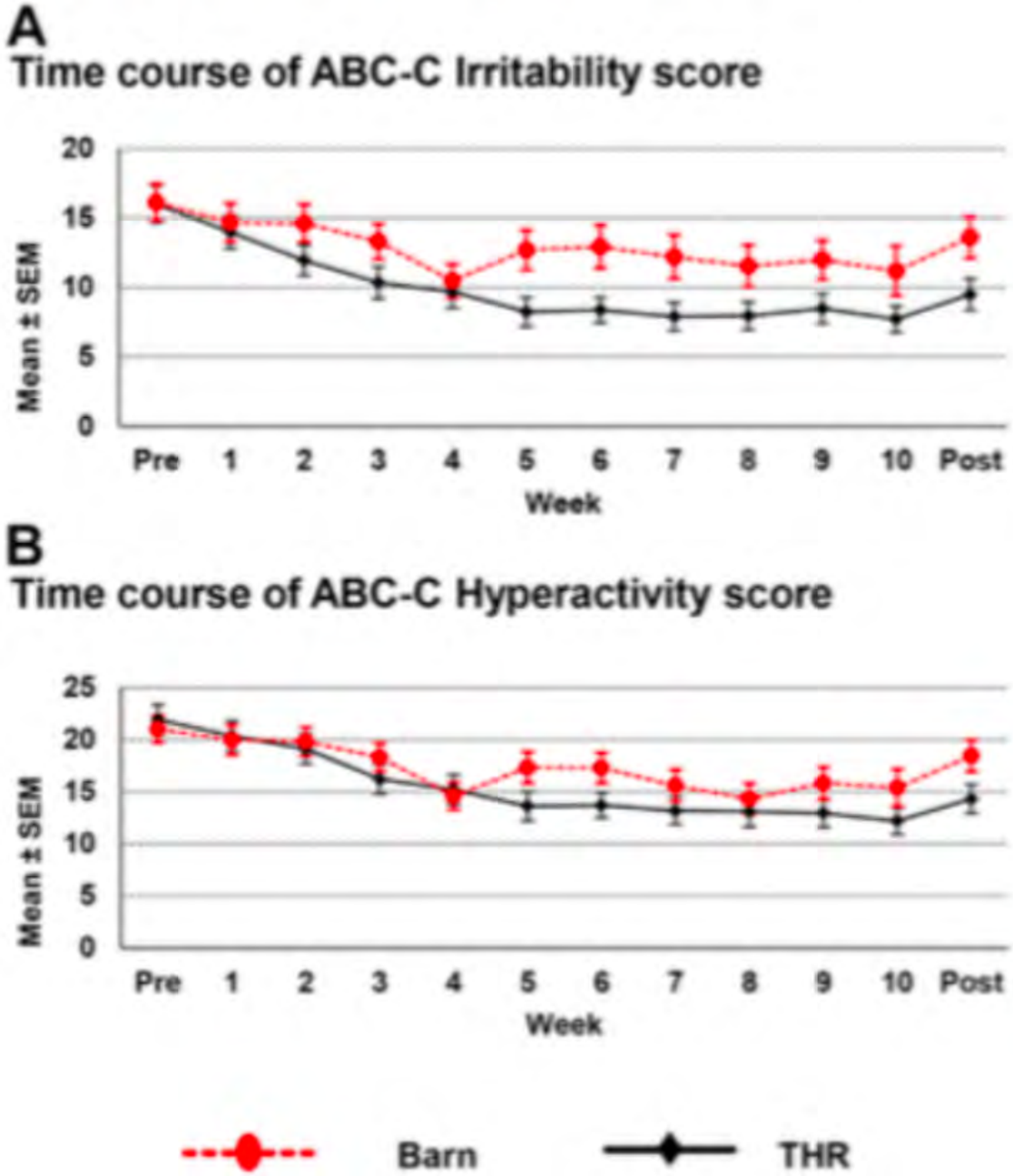

Take home messages

- Autisme Spectrum Stoornis - it's official
- Sociale Communicatie Stoornis

- Autisme is niet meer de meest erfelijke aandoening ooit
- Geen nieuwe medicatie op dit moment - wel meer evidence voor de praktijk
- Vroege behandelingen hebben effect - echt
- Behandeling wordt individueler

Belangrijkste boodschappen

accare
Kinder- en jeugdpsychiatrie