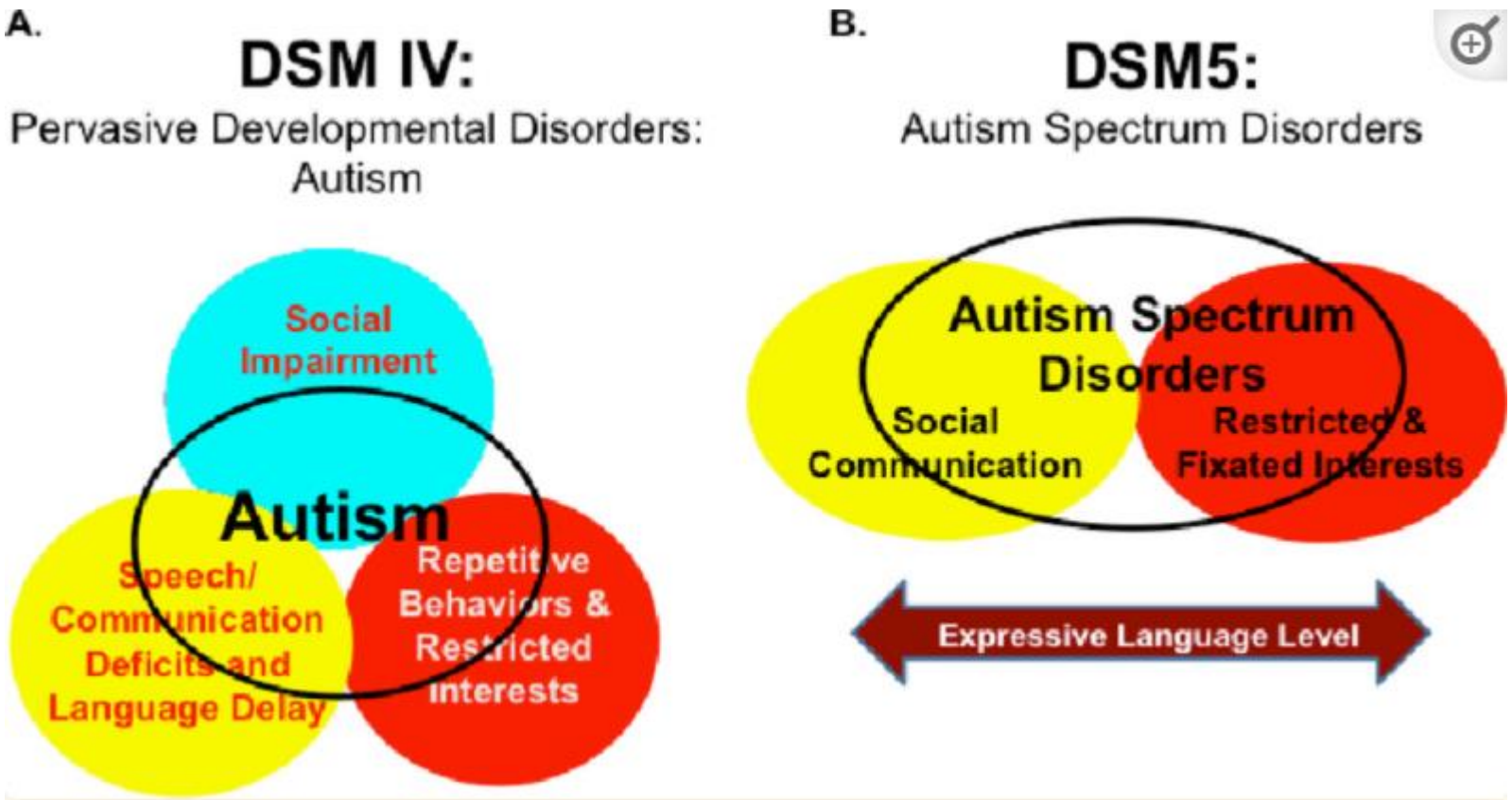
OKB ve Otistik Özelliklerin Nörobiyolojisi

Ne kadar ortak, ne kadar farklı?

Emre Bora

- Giriş
- OSB-OKB: Genetik, Nöropsikolojik ve Beyin Görüntüleme Bulguları
- OSB-OKB Akrabalarında Silik Klinik Bulgular, Nöropsikolojik ve Beyin Görüntüleme Bulguları
- OSB ve OKB ilişkisi



Lord ve Jones, 2013

DSM-5 OSB semptom kümeleri

A- Sosyal-iletişimsel alanda yetersizlikler

- Toplumsal-duygusal karşılıkta yetersizlik
- Sözel olmayan iletişimde yetersizlik
- İlişki kurma ve sürdürmede yetersizlik

B- Tekrarlayıcı ilgiler ve davranışlar

- Basmakalıp ve tekrarlayıcı motor hareketler
- Aynılıkta ısrar, rutine sıkı bağlılık
- Sınırlı ve yoğun ilgi alanı
- Duyusal az ya da çok uyarılma

Sosyal(Toplumsal) İletişim Bozukluğu

A. Aşağıdakilerden tümü ile kendini gösteren, sözel ve sözel olmayan iletişimin toplumsal kullanımında süregiden güçlükler:

1. Selamlaşma gibi toplumsal amaçlı iletişimde eksiklikler

2. İçinde bulunulan durumla ya da dinleyenin gereksinimleriyle eşleşecek biçimde iletişim biçimini değiştirme yeterliliğinde bozukluk

3. Konuşmanın ve anlatımın kurallarına uymakta güçlük (sırayla konuşma, anlaşılmadığında tekrarlama, simgelerin kullanımı gibi)

4. Çıkarımda bulunmada güçlükler

B. İletişimde işlevsel bozulmanın olması

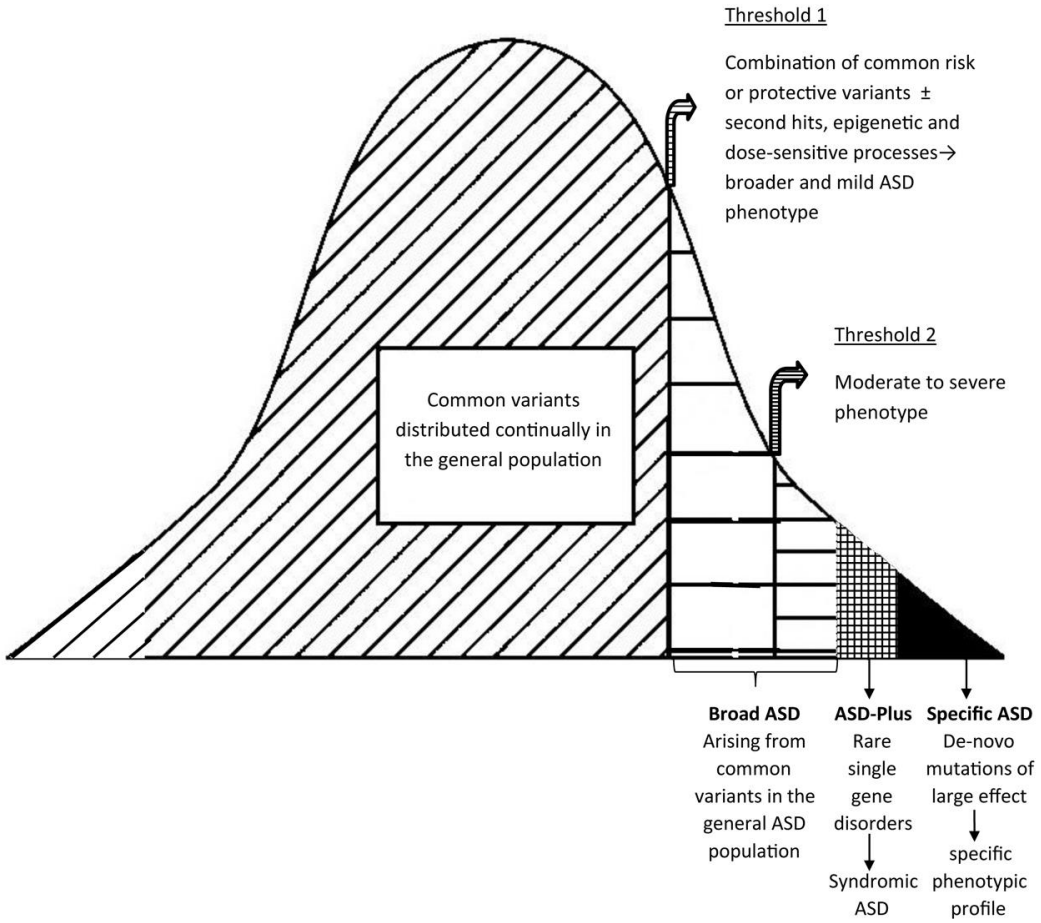
C. Erken gelişim evresinde başlamıştır.

D. Başka bir sağlık durumu veya nörolojik bozukluklarla açıklanamaz.

Adaptif olan ve olmayan özellikleriyle farklılık olarak otizm spektrumu

- Sosyal ve sosyal olmayan beyin etkinlik balansı açısından dengeli (nörotipik) ve sağa/veya sola kaymış (neurodivergent) bireyler var
- OSB trait düzeyinde avantaj ve dezavantajlarla geliyor. Çağa göre değişmekte
- Belirgin olduğunda patolojik olarak sınıflandırmaktayız
- Nörogelişimsel sapmalar görece özgül olmak yanında (saf OSB), yaygın olduğunda OSB yanında entelektüel ve fiziksel sorunlara yol açabilir.

Geniş otizm fenotipi



Eapen ve ark 2013

DSM-5'e göre OKB Tanı Kriterleri[11]:

A. Obsesyonlar, kompulsiyonlar ya da her ikisinin birlikte varlığı:

Obsesyonlar aşağıdakilerden (1) ve (2) ile tanımlanır:

1) Bu bozukluk sırasında kimi zaman zorla ve istenmeden gelen ve çoğu kişide anksiyete ya da sıkıntıya neden olan, yineleyici ve sürekli düşünceler, dürtüler ya da düşlemler.
2) Kişi bu düşünceleri, dürtüleri ya da düşlemlerine önem vermemeye ya da bunları baskılamaya çalışır ya da başka bir düşünce ya da eylemle etkisizleştirmeye çalışır.

Kompulsiyonlar aşağıdakilerden (1) ve (2) ile tanımlanır:

1) Kişinin, obsesyona bir tepki olarak ya da katı bir biçimde uygulaması gereken kurallara göre yapmaktan kendini alıkoyamadığı yineleyici davranışlar (örn. el yıkama, düzene koyma, kontrol etme) ya da zihinsel eylemler (örn. dua etme, sayı sayma, bir takım sözcükleri sessiz bir biçimde söyleyip durma)
2) Davranışlar ya da zihinsel eylemler, sıkıntıdan kurtulmaya ya da var olan sıkıntıyı azaltmaya ya da korku yaratan olay ya da durumdan korunmaya yöneliktir; ancak bu davranışlar ya da zihinsel eylemler ya etkisizleştirilmesi ya da korunulması tasarlanan şeylerle gerçekçi bir biçimde ilişkili değildir ya da açıkça çok aşırı bir düzeydedir.

B. Obsesyon ya da kompulsiyonlar belirgin bir sıkıntıya neden olur, zamanın boşa harcanmasına yol açar (günde 1 saatten daha fazla zaman alırlar) ya da kişinin olağan günlük işlerini, mesleki (ya da eğitimle ilgili) işlevselliğini ya da olağan toplumsal etkinliklerini ya da ilişkilerini önemli ölçüde bozar.

C. Bu bozukluk bir maddenin (örn. kötüye kullanılabilen bir ilaç ya da tedavide kullanılan bir ilaç) ya da genel tıbbi durumun doğrudan fizyolojik etkilerine bağlı değildir.

D. Başka bir eksen 1 bozukluğu varsa, obsesyon ya da kompulsiyonların içeriği bununla sınırlı değildir (örn. bir yeme bozukluğunun olması durumunda yemek konusu üzerinde düşünüp durma; trikotillomaninin olması durumunda saç çekme üzerinde durma; vücut dismorfik bozukluğunun olması durumunda dış görünümle aşırı ilgilenme; bir madde kullanım bozukluğunun olması durumunda ilaçlar üzerinde düşünüp durma; hipokondriyazisin olması durumunda ciddi bir hastalığı olduğu biçiminde düşünüp durma; bir parafilinin olması durumunda cinsel dürtüler ya da fanteziler üzerinde düşünüp durma ya da majör depresif bozukluk olması durumunda suçluluk üzerine geviş getirircesine düşünme).

Varsa belirtiniz:

İç görüsü iyi: Kişi inanışlarının gerçek olmadığının farkındadır.

İç görüsü kötü: Kişi inanışlarının olasılıkla gerçek olduğunu düşünür.

İç görüsü yok/sanrısal inanışlar: Kişi inanışlarının gerçek olduğuna kesin olarak inanmaktadır.

Varsa belirtiniz:

Tikle ilişkili: Kişinin o sırada ya da geçmişte bir tik bozukluğu öyküsü vardır.

OSB-OKB: Genetik, Nöropsikolojik ve Beyin Görüntüleme Bulguları

JAMA. 2017 Sep 26;318(12):1182-1184. doi: 10.1001/jama.2017.12141.

The Heritability of Autism Spectrum Disorder

Sven Sandin [1], Paul Lichtenstein [2], Ralf Kuja-Halkola [2], Christina Hultman [2], Henrik Larsson [3], Abraham Reichenberg [1]

Kalıtılabilirlik oranı % 83

nature genetics
ARTICLES
https://doi.org/10.1038/s41588-019-0344-8

Identification of common genetic risk variants for autism spectrum disorder

Jakob Grove[1,2,3,4], Stephan Ripke[5,6,7], Thomas D. Als[1,2,3], Manuel Mattheisen[1,2,3,8,9],

18,381 OSB ve 27,969 SK, varyasyonu açıklama oranı henüz düşük (% 2-4)

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ScienceDirect
Current Opinion in Neurobiology

Neurodevelopmental impact of CNV models in ASD: Recent advances and future directions

Kota Tamada and Toru Takumi

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by social communication impairments and restricted, repetitive behaviors. ASD exhibits a strong

For complete overview of the section, please refer the article collection - Mechanisms underlying neurodevelopmental disorders 2024
Available online 15 March 2025

Table 1

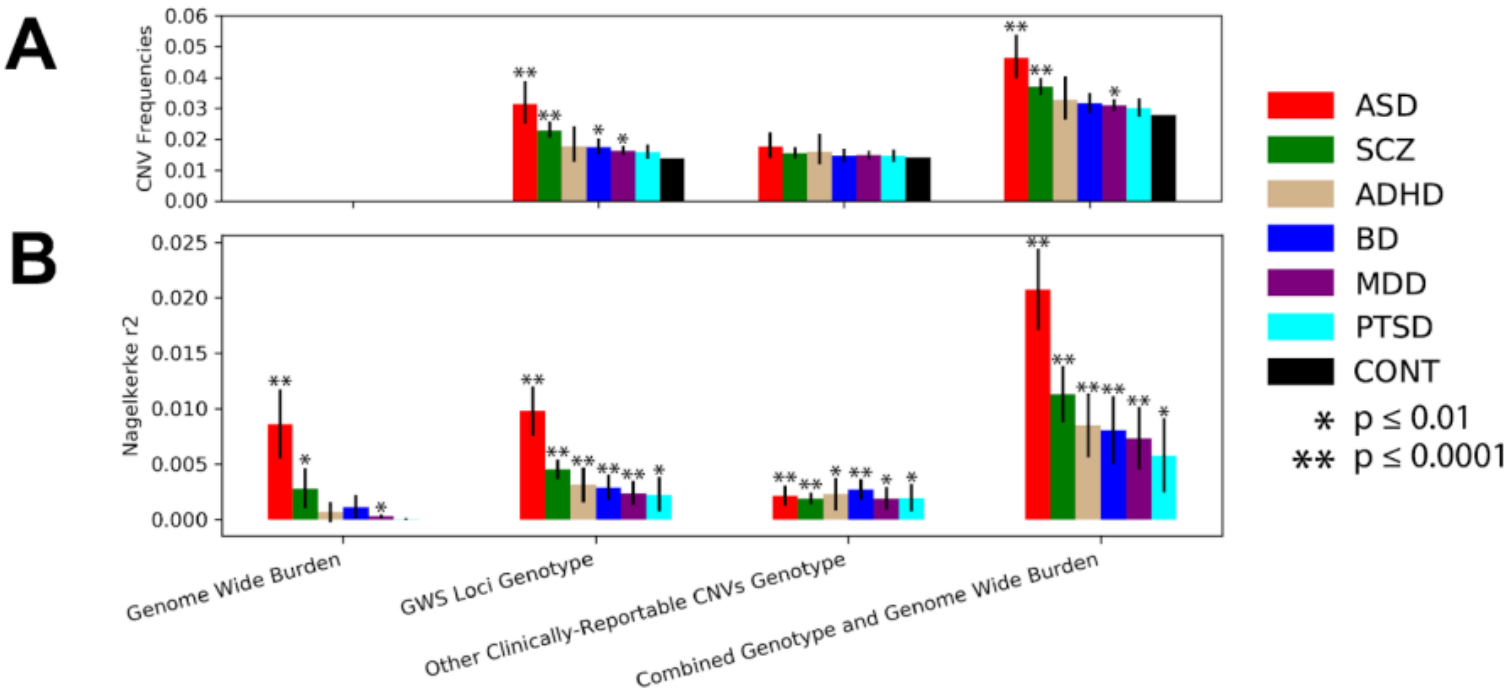
The prevalence of recurrent CNVs in individuals with ASD.

CNV types	Number of cases	Prevalence (%)
1q21.1 deletion	4	0.05
1q21.1 duplication	25[a]	0.33
7q11.23 deletion	1	0.01
7q11.23 duplication	8	0.11
15q11-q13 duplication	19	0.25
15q13.3 deletion	12	0.16
16p11.2 deletion	25[b]	0.33
16p11.2 duplication	14	0.19
22q11.2 deletion	2	0.03
22q11.2 duplication	14	0.19

These data are based on "Table S5H" in Trost et al., 2022 [13].
ASD-associated rare variants in 5100 individuals with ASD from MSSNG and 2419 from SSC.

A cross-disorder analysis of CNVs finds novel loci and dose-dependent relationships of genes to psychiatric traits

Omar Shanta[1,2], Marieke Klein[2,3,4], Molly Sacks[1,2], Jeffrey R. MacDonald[5,6], Adam Maihofer[2,7,8], Mohammad Ahangari[2], Worrawat Engchuan[5,6], Bhooma Thiruvahindrapuram[5,6], James Guevara[2],



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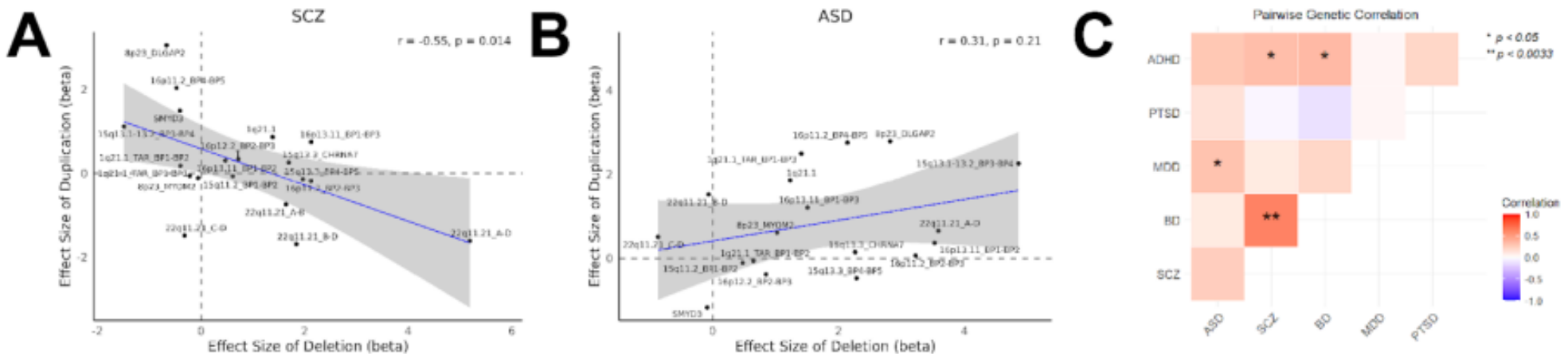
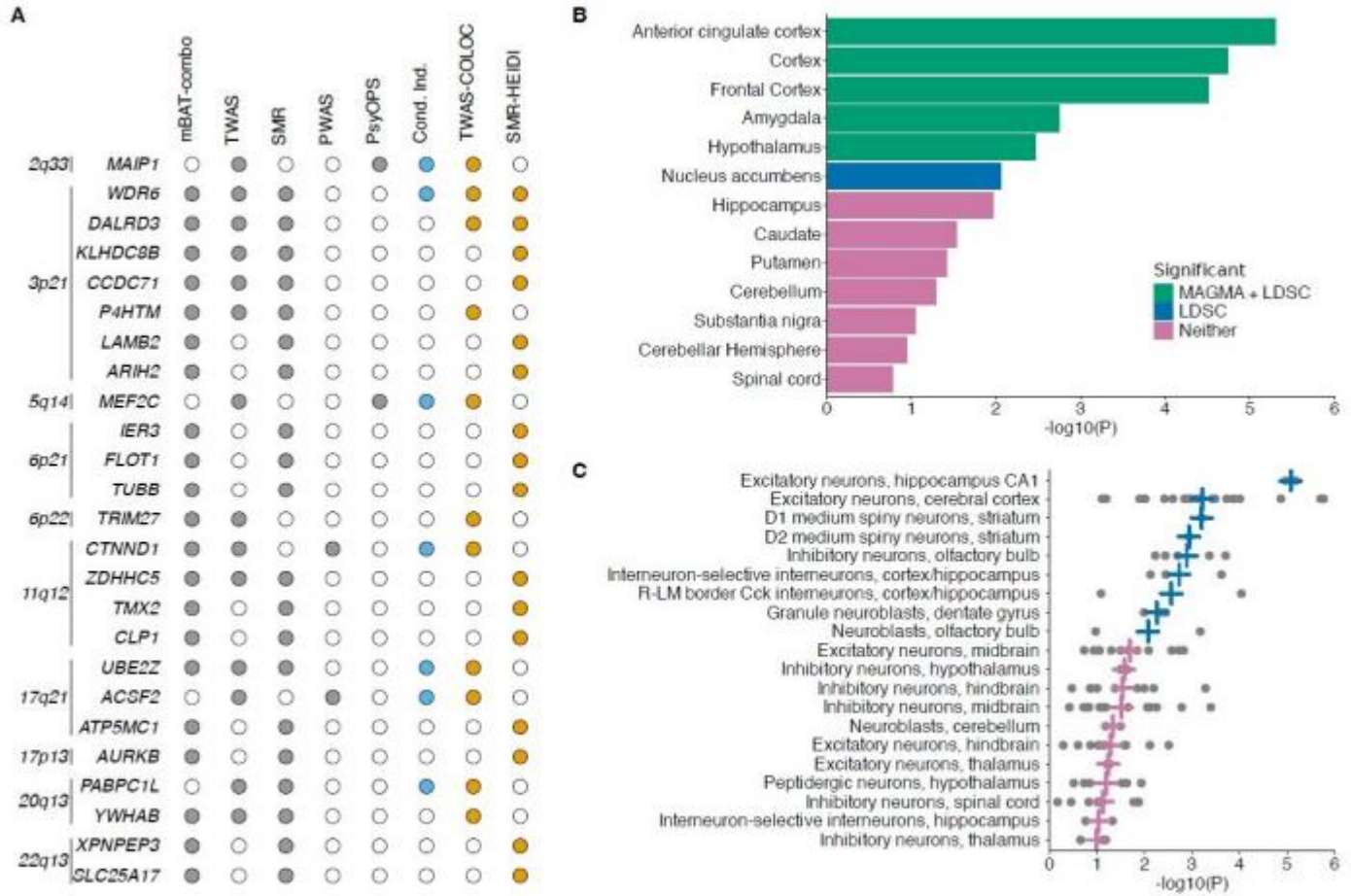


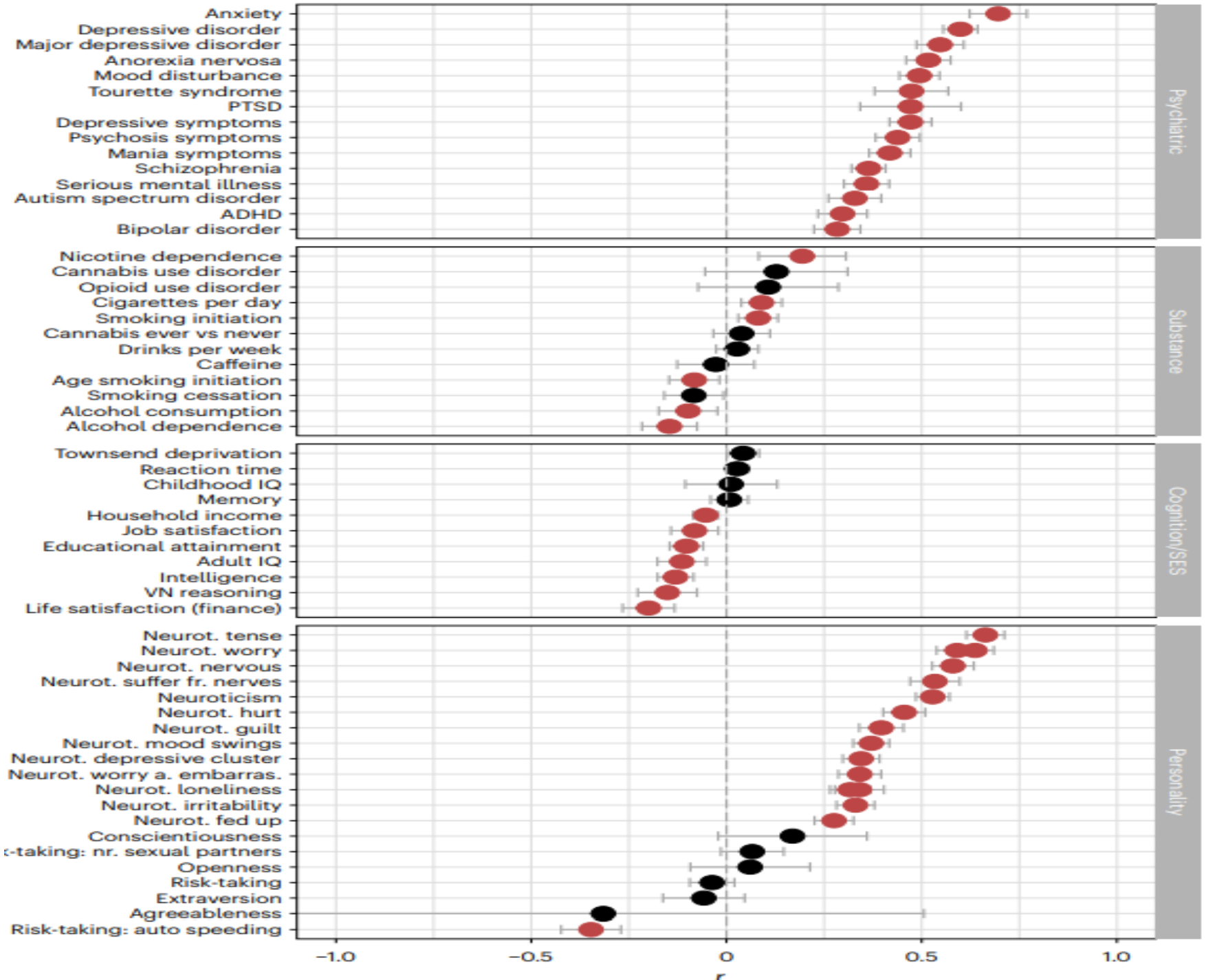
OSB için bütün delesyon ve duplikasyonlar riski artırırken
Şizofreni (ve diğer) zıt etkili örnekler, biri artırırken biri koruyucu

Genome-wide analyses identify 30 loci associated with obsessive-compulsive disorder

Nora I. Strom[1,2,3,4,#], Zachary F. Gerring[5,6,#], Marco Galimberti[7,8,#], Dongmei Yu[9,10,#], Matthew W. Halvorsen[11],

Obsessive-compulsive disorder (OCD) affects ~1% of children and adults and is partly caused by genetic factors. We conducted a genome-wide association study (GWAS) meta-analysis combining 53,660 OCD cases and 2,044,417 controls and identified 30 independent genome-wide significant loci. Gene-based approaches identified 249 potential effector genes for OCD, with 25 of these classified as the most likely causal candidates, including *WDR6*, *DALRD3*, *CTNND1* and multiple genes in the MHC region. We estimated that ~11,500 genetic variants explained 90% of OCD genetic heritability. OCD genetic risk was associated with excitatory neurons in the hippocampus and cortex, along with D1- and D2-type dopamine receptor-containing medium spiny neurons. OCD genetic risk was shared with 65 of 112 additional phenotypes, including all of the psychiatric disorders we examined. In particular, OCD shared genetic risk with anxiety, depression, anorexia nervosa, and Tourette syndrome, and was negatively associated with inflammatory bowel diseases, educational attainment, and body mass index.





OSB ile korelasyon küçük ama anlamlı r=0.28 civarı

www.nature.com/mp

Molecular Psychiatry

ARTICLE OPEN

Check for updates

A burden of rare copy number variants in obsessive-compulsive disorder

Matthew W. Halvorsen[1,2✉], Elles de Schipper[2], Julia Bäckman[2], Nora I. Strom[2,3,4,5], Kristen Hagen[6,7,8], Nordic OCD and Related Disorders Consortium (NORDiC)*, Kerstin Lindblad-Toh[9,10], Elinor K. Karlsson[10,11,12], Nancy L. Pedersen[13], John Wallert[2], Cynthia M. Bulik[13,14,15], Bengt Fundín[13], Mikael Landén[13,16], Gerd Kvale[8,17], Bjarne Hansen[8,18], Jan Haavik[8,19], Manuel Mattheisen[4,20], Christian Rück[2], David Mataix-Cols[2,21] and James J. Crowley[1,2]

Current genetic research on obsessive-compulsive disorder (OCD) supports contributions to risk specifically from common single nucleotide variants (SNVs), along with rare coding SNVs and small insertion-deletions (indels). The contribution to OCD risk from rare copy number variants (CNVs), however, has not been formally assessed at a similar scale. Here we describe an analysis of rare CNVs called from genotype array data in 2248 deeply phenotyped OCD cases and 3608 unaffected controls from Sweden and Norway. Cases carry an elevated burden of CNVs ≥30 kb in size (OR = 1.12, $P = 1.77 \times 10^{-3}$). The excess rate of these CNVs in cases versus controls was around 0.07 (95% CI 0.02–0.11, $P = 2.58 \times 10^{-3}$). This signal was largely driven by CNVs overlapping protein-coding regions (OR = 1.19, $P = 3.08 \times 10^{-4}$), particularly deletions impacting loss-of-function intolerant genes (pLI >0.995, OR = 4.12, $P = 2.54 \times 10^{-5}$). We did not identify any specific locus where CNV burden was associated with OCD case status at genome-wide significance, but we noted non-random recurrence of CNV deletions in cases (permutation $P = 2.60 \times 10^{-3}$). In cases where sufficient clinical data were available ($n = 1612$) we found that carriers of neurodevelopmental duplications were more likely to have comorbid autism ($P < 0.001$), and that carriers of deletions overlapping neurodevelopmental genes had lower treatment response ($P = 0.02$). The results demonstrate a contribution of rare CNVs to OCD risk, and suggest that studies of rare coding variation in OCD would have increased power to identify risk genes if this class of variation were incorporated into formal tests.

Molecular Psychiatry (2025) 30:1510–1517; https://doi.org/10.1038/s41380-024-02763-7

CNV ılımlı artış
Duplikasyonlar komorbid OSB ile açıklanabilir
Delesyonlar bağımsız

Pragmatik dil bozukluğu, OSB ve nöropsikolojik teoriler

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Brain and Language 85 (2003) 451–466

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Weak coherence, no theory of mind, or executive dysfunction? Solving the puzzle of pragmatic language disorders

Ingerith Martin and Skye McDonald*

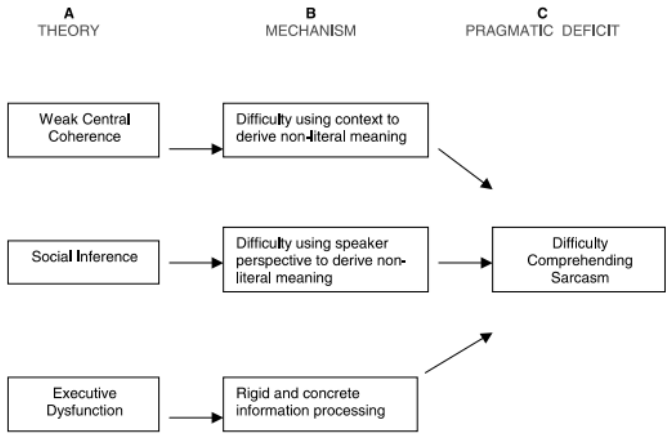

Fig. 3. Mechanisms and predictions of WCC, social inference and executive function accounts of pragmatic impairment.

Theory	Level of Processing	Level of Specificity
Weak Central Coherence	Low level, automatic processing	General and non-domain specific
Social Inference	High level, effortful processing	Specific to the social and emotional domain
Executive Dysfunction	High level, effortful processing	General and non-domain specific

OSB ve Sosyal Biliş
Zihin kuramı ve emosyon tanıma

AMERICAN PSYCHOLOGICAL ASSOCIATION

Psychological Bulletin

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2021, Vol. 147, No. 3, 268–292
http://dx.doi.org/10.1037/bul0000310

A Quantitative Meta-Analysis of Face Recognition Deficits in Autism: 40 Years of Research

Jason W. Griffin, Russell Bauer, and K. Suzanne Scherf
Pennsylvania State University

The ability to recognize an individual face is essential to human social interaction. Even subtle errors in this process can have huge implications for the way we relate to social partners. Because autism spectrum disorder (ASD) is characterized by deficits in social interaction, researchers have theorized about the potential role of atypical face identity processing to the symptom profile of ASD for more than 40 years. We conducted an empirical meta-analysis of this large literature to determine whether and to what extent face identity processing is atypical in ASD compared to typically developing (TD) individuals. We also tested the hypotheses that the deficit is selective to face identity recognition, not perception, and that methodological variation across studies moderates the magnitude of the estimated deficit. We identified 112 studies (5,390 participants) that generated 172 effect sizes from both recognition ($k = 119$) and discrimination ($k = 53$) paradigms. We used state-of-the-art approaches for assessing the validity and robustness of the analyses. We found comparable and large deficits in ASD for both face identity recognition (Hedge's $g = -0.86$) and discrimination (Hedge's $g = -0.82$). This means that the score of an average ASD individual is nearly 1 *SD* below the average TD individual on tasks assessing both aspects of face identity processing. These deficits generalize across age groups, sex, IQ scores, and task paradigms. These findings suggest that deficits in face identity processing may represent a core deficit in ASD.

Journal of Autism and Developmental Disorders
https://doi.org/10.1007/s10803-025-06833-9

ORIGINAL ARTICLE

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Emotional Prosody Recognition in Autism Spectrum Disorder Without Intellectual Disability: A Systematic Review and Meta-Analysis

Qiyun Luo[1] · Xiaoyan Zeng[1] · Fei Chen[1] · Chen Kuang[1]

Psychological Medicine (2016), **46**, 699–716. © Cambridge University Press 2015
doi:10.1017/S0033291715002573

REVIEW ARTICLE

Meta-analysis of social cognition in attention-deficit/hyperactivity disorder (ADHD): comparison with healthy controls and autistic spectrum disorder

E. Bora* and C. Pantelis

Department of Psychiatry, Melbourne Neuropsychiatry Centre, University of Melbourne and Melbourne Health, Carlton South, Victoria 3053, Australia

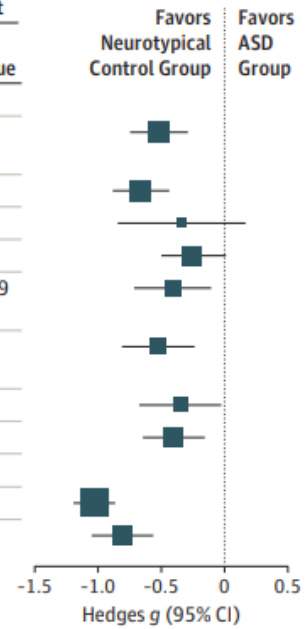
JAMA Psychiatry | Original Investigation

Patterns of Nonsocial and Social Cognitive Functioning in Adults With Autism Spectrum Disorder

A Systematic Review and Meta-analysis

Tjasa Velikonja, PhD; Anne-Kathrin Fett, PhD; Eva Velthorst, PhD

Domain	No. of Studies	No. in ASD Group	No. in Neurotypical Control Group	Hedges *g* Mean Effect (95% CI)	Hedges *g* *P* Value	Test for Heterogeneity Cochran Q Statistic	Test for Heterogeneity *P* Value	Test for Heterogeneity I^2 Statistic, %	Egger Test *P* Value
Nonsocial cognition									
Reasoning and problem solving	22	802	631	-0.51 (-0.74 to -0.28)	<.001	8.83	.99	0	.18
Processing speed	21	715	655	-0.61 (-0.83 to -0.38)	<.001	13.42	.86	0	.92
Attention and vigilance	5	126	105	-0.30 (-0.81 to 0.21)	.09	0.31	.99	0	.32
Working memory	19	589	530	-0.23 (-0.47 to 0.01)	.06	19.97	.39	4	.98
Visual learning and memory	12	429	380	-0.38 (-0.68 to -0.10)	.006	9.34	.59	0	.009
Verbal learning and memory	12	414	362	-0.55 (-0.86 to -0.25)	<.001	34.76	<.001	68	.08
Verbal comprehension	10	325	307	-0.33 (-0.66 to -0.01)	.009	4.09	.91	0	.43
Verbal fluency	15	748	604	-0.38 (-0.63 to -0.13)	<.001	1.98	>.99	0	.41
Social cognition									
Theory of mind	39	2213	4355	-1.09 (-1.25 to -0.92)	<.001	59.79	.01	36	.02
Emotion perception and processing	18	819	620	-0.80 (-1.04 to -0.55)	<.001	31.69	.02	46	.04



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Archives of Clinical Neuropsychology 39 (2024) 498–515

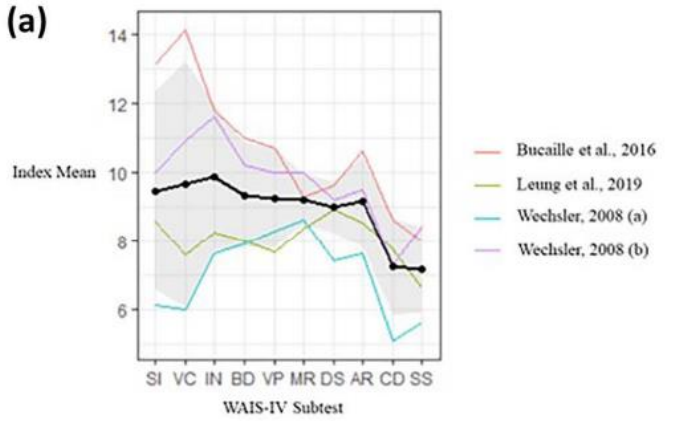
Archives of CLINICAL NEUROPSYCHOLOGY

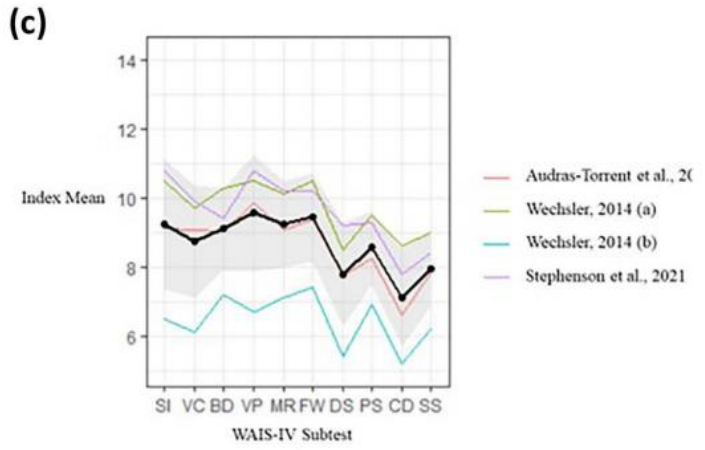
Cognitive Profile in Autism and ADHD: A Meta-Analysis of Performance on the WAIS-IV and WISC-V

Alexander C. Wilson*

OSB Sayı sembol özellikle kötü küplerle desen görece iyi

Sözel IQ, düşük işlevsellikli bireylerde daha kötü olsa yüksek işlevsellikli bireylerde daha iyi





frontiers in Psychiatry

SYSTEMATIC REVIEW
published: 26 March 2020
doi: 10.3389/fpsyt.2020.00187

J Autism Dev Disord
DOI 10.1007/s10803-014-2188-5

ORIGINAL PAPER

Visuo-Spatial Performance in Autism: A Meta-analysis

Anne Muth · Johannes Hönekopp · Christine M. Falter

D [95% CI]

-0.43, 0.81]
1.03, 1.95]
-0.04, 0.85]
0.29, 1.95]
0.03, 1.24]
-0.34, 0.50]

0.18, 1.09]

Abstract Visuo-spatial skills are believed to be enhanced in autism spectrum disorders (ASDs). This meta-analysis tests the current state of evidence for Figure Disembedding, Block Design, Mental Rotation and Navon tasks in ASD and neurotypicals. Block Design ($d = 0.32$) and Figure Disembedding ($d = 0.26$) showed superior performance for ASD with large heterogeneity that is unaccounted for. No clear differences were found for Mental Rotation. ASD samples showed a stronger local processing preference for Navon tasks ($d = 0.35$); less clear evidence for performance differences of a similar magnitude emerged. We discuss the meta-analysis results together with other findings relating to visuo-spatial processing and three cognitive theories of ASD: Weak Central Coherence, Enhanced Perceptual Functioning and Extreme Male Brain theory.

Keywords Autism · Visuo-spatial · Figure Disembedding · Mental Rotation · Block Design · Navon

Introduction

The term autism spectrum disorder (ASD) is an umbrella term that encompasses Autistic disorder, Asperger's syndrome (AS), High-Functioning Autism (HFA) and pervasive developmental disorder not otherwise specified (PDD-NOS). As ASDs are often accompanied by learning and language impairments, it is most common for people with HFA to participate in research studies because their high-functioning level means that they are intellectually capable of meeting the needs of cognitive studies. Disorders of the autism spectrum are characterized by impairments in social interaction and communication, and restricted, repetitive and stereotyped patterns of behavior, interests and activities (Levy et al. 2009; APA 2000).

The question whether individuals with ASD have superior visuo-spatial skills compared to neurotypicals has inspired research for several decades and goes back to

Neuroscience and Biobehavioral Reviews 155 (2023) 105441

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

Comparison of social cognition and neurocognition in schizophrenia and autism spectrum disorder: A systematic review and meta-analysis

Simge Uzman Ozbek [a], Ekin Sut [a], Emre Bora [a,b,c,*]

Belirgin yaş etkisi

Mean weigthed effect sizes for differences of social cognition between participants with schizoprenia and ASD.

Test	Study N	SSD N	ASD N	g	%95 CI	Z	P	Q	Q (P)	Bias (P)	I^2
Emotion recognition	17	642	529	-0,17	[− 0,39–0,05]	-1,56	0,12	45,45	0.0001	0.4720	67.48%
Facial emotion recognition	15	557	451	-0,2	[− 0,45–0,04]	-1,64	0,1	40,52	0.0002	0.5629	68.53%
Emotion regulation	3	169	176	0,13	[− 0,24–0,50]	0,67	0,1	4,5	0.1054	0.7119	57.61%
Social perception	5	200	180	0,09	[− 0,11–0,3]	0,89	0,37	1,71	0.8876	0.1094	0.00%
RMET	13	438	467	-0,12	[− 0,37–0,13]	-0,94	0,35	32,83	.001	0.44	69.21%
ToM reasoning	19	673	536	0,13	[− 0,08–0,34]	1,19	0,24	48,91	0.0001	0.6331	65.25%
General ToM	27	935	799	-0,01	[− 0,19–0,18]	-0,08	0,93	76,50	< .0001	0.27	69.53%
Global social cognition (4 domain)	37	1284	1119	-0,06	[− 0,21–0,08]	-0,83	0,4	95,69	< .0001	0.3	65.05%

Mean weigthed effect sizes for differences of neurocognition between participants with schizoprenia and ASD.

Test	Study N	SSD N	ASD N	g	%95 CI	Z	P	Q	Q (P)	Bias (P)	I^2
Fluency	6	358	356	0,47	[0,17–0,76]	3,11	0,0019	14,89	0,01	0,8421	68.97%
Processing speed	19	754	737	0,41	[0,20–0,62]	3,75	0,0002	61,3	< .0001	0,0849	70.57%
Attention/ vigilance	5	233	249	0,39	[0,21–0,57]	4,19	< .0001	3,39	0,4944	0,3315	0.00%
Working memory	14	586	620	0,19	[− 0,03–0,40]	1,72	0,0853	35,8	0,0006	0,2716	64.37%
Reasoning and problem solving	16	536	567	0,31	[0,15–0,47]	3,89	< .0001	21,51	0,1212	0,2995	32.37%
Verbal learning and memory	7	353	342	0,38	[0,09–0,66]	2,61	0,0091	14,31	0,0264	0,9116	64.41%
Visual learning and memory	3	169	177	-0,08	[− 0,29–0.13]	-0,72	0,47	0,57	0,7508	0,4011	0.00%
WCST	5	129	134	0,35	[− 0,06–0,76]	1,67	0	9,5	0,0495	0,8692	56.84%
RPM	7	165	200	0,22	[− 0,07–0,50]	1,49	0,1367	11,02	0,0879	0,07	45.25%

Mean weigthed effect sizes for differences of IQ and subtests between participants with schizoprenia and ASD.

Test	Study N	SSD	ASD	g	%95 CI	Z	P	Q	Q (P)	Bias (P)	I^2
IQ	27	710	743	0,36	[0,18–0,54]	3,98	< .0001	65,89	< .0001	0.14	60.61%
Premorbid IQ	12	555	506	0,39	[0,23–0,55]	4,74	< .0001	14,38	0,28	0.23	30.08%
Digit symbol	8	211	290	0,39	[0,03–0,74]	2,13	0,0328	21,44	0,0032	0.5276	66.49%
Digit span	8	251	312	0,21	[− 0,02–0,45]	1,79	0,0733	10,84	0,1458	0,1177	36.77%
Similarities	8	222	296	0,3	[− 0,05–0,65]	1,7	0,0881	23,97	0,0012	0,6905	67.86%
Block design	7	200	275	0,63	[0,23–1,03]	3,12	0.0018	23,45	0,0007	0,7272	71.17%
Vocabulary	5	129	252	0,3	[0,07–0,52]	2,59	0,0097	3,72	0,4456	0934	2.20%
Information	6	187	262	0,29	[0,01–0,57]	2	0,0455	8,43	0,1339	0,6237	43.14%
Comprehension	6	187	262	0,25	[0,10–0,6]	1,42	0,1542	12,12	0,0332	0,6207	61.67%
Arithmetic	6	187	262	0,28	[0,01–0,55]	2,02	0043	8,05	0,1534	0,4253	38.02%
Picture completion	5	170	230	0,5	[0,21–0,8]	3,33	0,0009	8,38	0,0785	0,6203	38.39%
Object assembly	5	170	230	0,56	[0,17–0,95]	2,84	0,0045	11,13	252	0,6177	63.74%
Picture arrangement	4	160	215	0,66	[− 0,05–1,38]	1,82	0,0681	22,73	< .0001	0,4323	88.57%

Psychiatry Research 351 (2025) 116633

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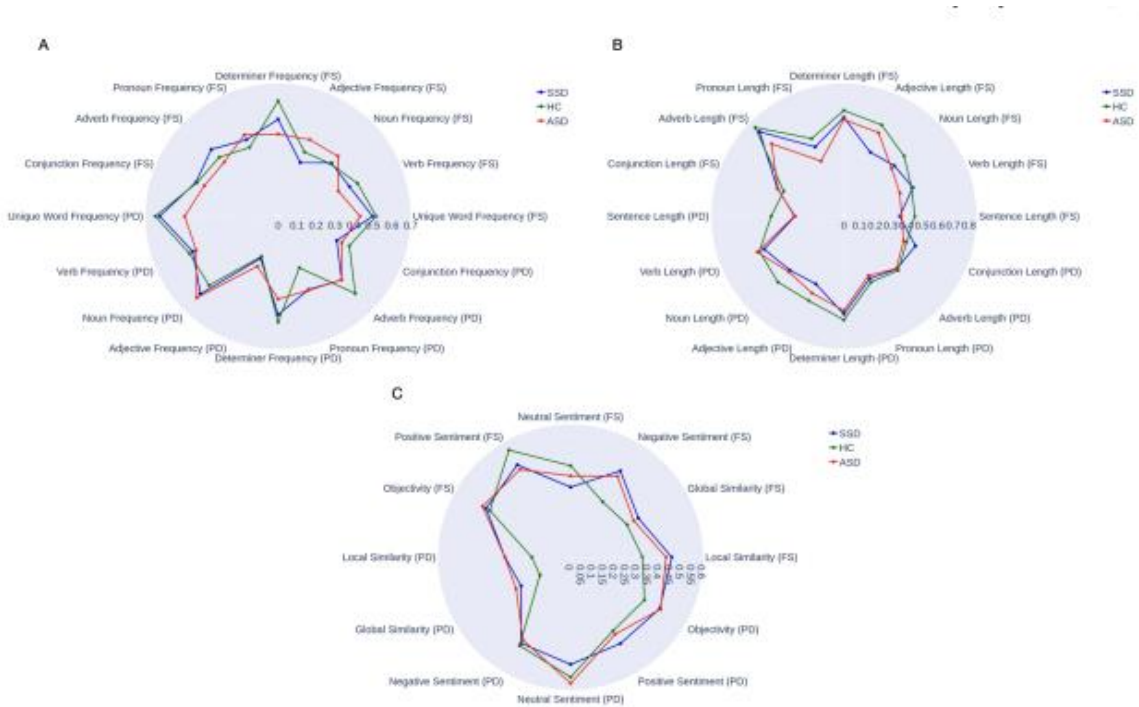
journal homepage: www.elsevier.com/locate/psychres

Computational linguistic investigation in schizophrenia and autism spectrum disorders

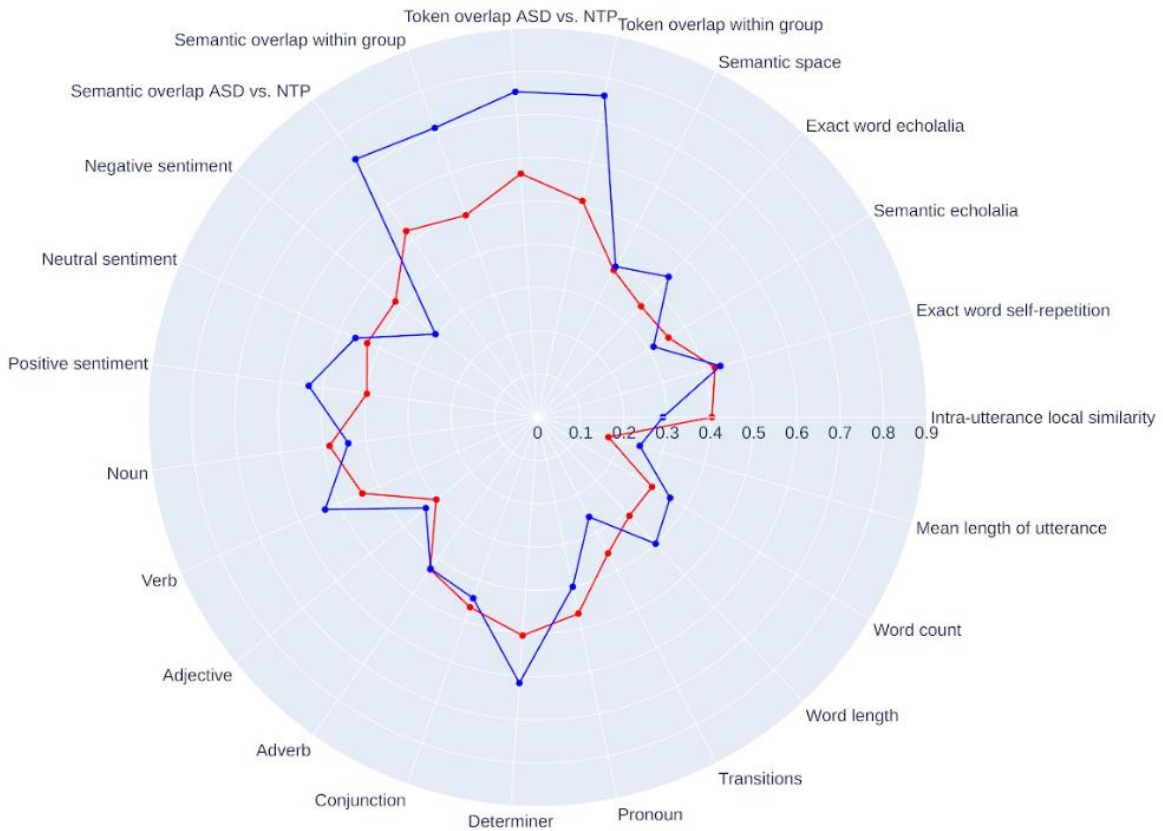
Berat Arslan [a,1,*], Elif Kizilay [a,1], Yaren Ecesu Turan [a], Burcu Verim [a], Cemal Demirlek [b,c], Muhammed Demir [a], Özge İlhan [a], Ezgi Cesim [a], Emre Bora [a,d,e]



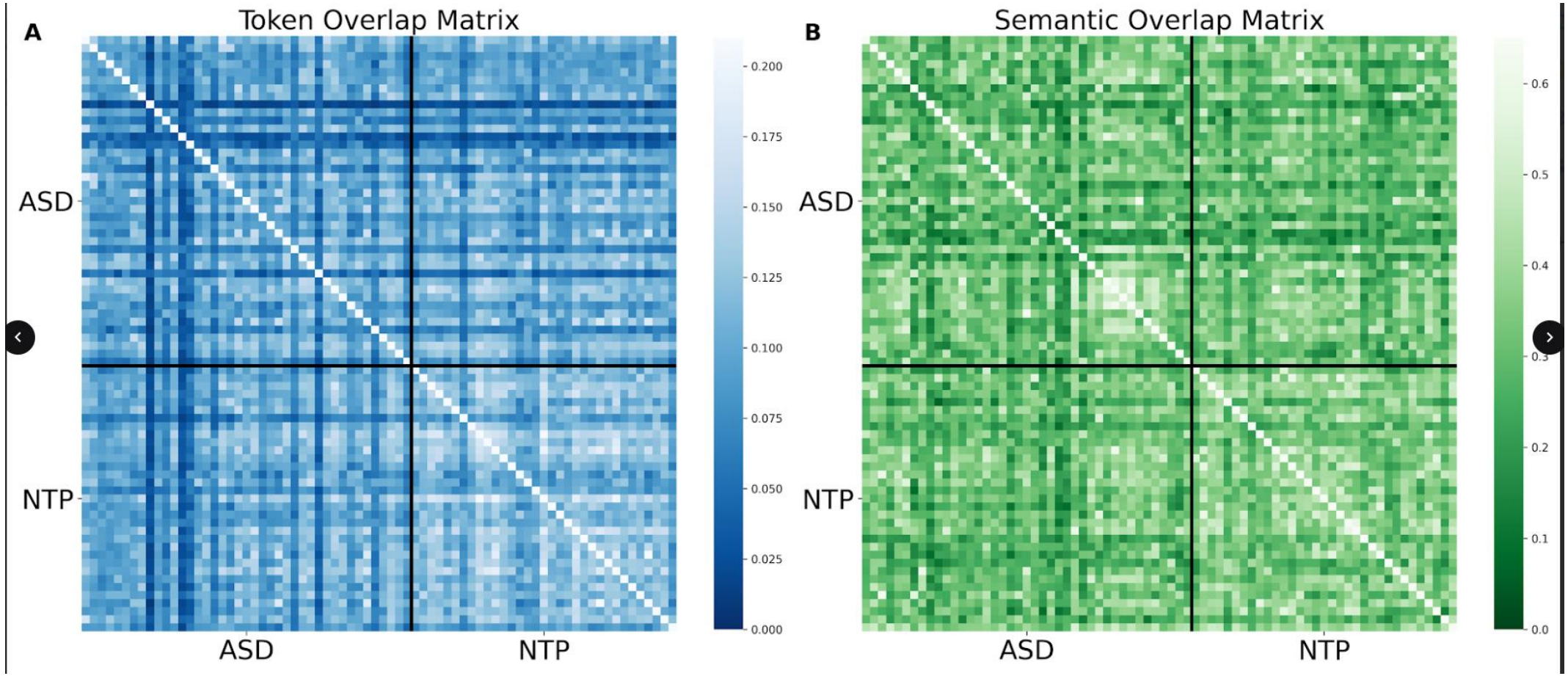
Assessing Communication Impairments in Adults with Autism Using Natural Language Processing

Running Title
Computational Assessment of Autism Speech

Author Names
Elif Kizilay[1,*], Berat Arslan[1], Burcu Verim[1,2], Ezgi Cesim[1], Yaren Ecesu Turan[1], Muhammed Demir[1], Özge İlhan[1], Cemal Demirlek[3], Emre Bora[1,4,5]



conversational speech samples from 41 autistic and 33 neurotypical adults, focusing on repetitive and idiosyncratic language usage, as well as sentiment, syntactic and generic speech features. Autistic individuals exhibited higher intra-utterance local similarity, indicating increased self-repetition; lower token and semantic overlap with other participants, reflecting the use of unusual words and expressions; more negative and less positive sentiment, suggesting a more negative emotional tone; fewer determiners and more turn-taking transitions, indicating reduced reciprocity; and shorter words compared to neurotypical controls. Lower token overlap in ASD was associated with reduced cognitive functioning and decreased mental state decoding and reasoning abilities, while fewer determiners were related to reduced theory of mind ability. These findings highlight that communication impairments can be objectively assessed using computational techniques,


A
Token Overlap Matrix
ASD
NTP
ASD
NTP
0.200
0.175
0.150
0.125
0.100
0.075
0.050
0.025
0.000
B
Semantic Overlap Matrix
ASD
NTP
ASD
NTP
0.6
0.5
0.4
0.3
0.2
0.1
0.0

Intra-utterance local similarity
Exact word self-repetition
Semantic echolalia
Exact word echolalia
Semantic space
Token overlap within group
Token overlap ASD vs. NTP
Semantic overlap within group
Semantic overlap ASD vs. NTP
Negative sentiment
Neutral sentiment
Positive sentiment
Noun
Verb
Adjective
Adverb
Conjunction
Determiner
Pronoun
Transitions
Word length
Word count
Mean length of utterance
SCIP -0.37 0.15 -0.11 0.09 0.11 0.54 0.51 0.12 0.29 -0 -0.14 0.15 -0.14 -0.09 0.15 0.25 0.16 0.25 -0.17 -0.41 0.23 0.22 0.36
RMET -0.15 0.14 -0.06 0.25 0.27 0.53 0.48 0.07 0.19 -0.01 -0.14 0.16 -0.15 -0.07 0.24 0.17 0.11 0.33 -0.15 -0.38 0.22 0.18 0.26
Hinting -0.17 -0.06 -0.09 0.31 0.09 0.42 0.37 0.09 0.14 0.18 -0.14 -0.07 -0.15 0.14 0.09 -0 0.12 0.33 -0.16 -0.18 0.12 0.15 0.08
0.50
0.25
0.00
−0.25
−0.50

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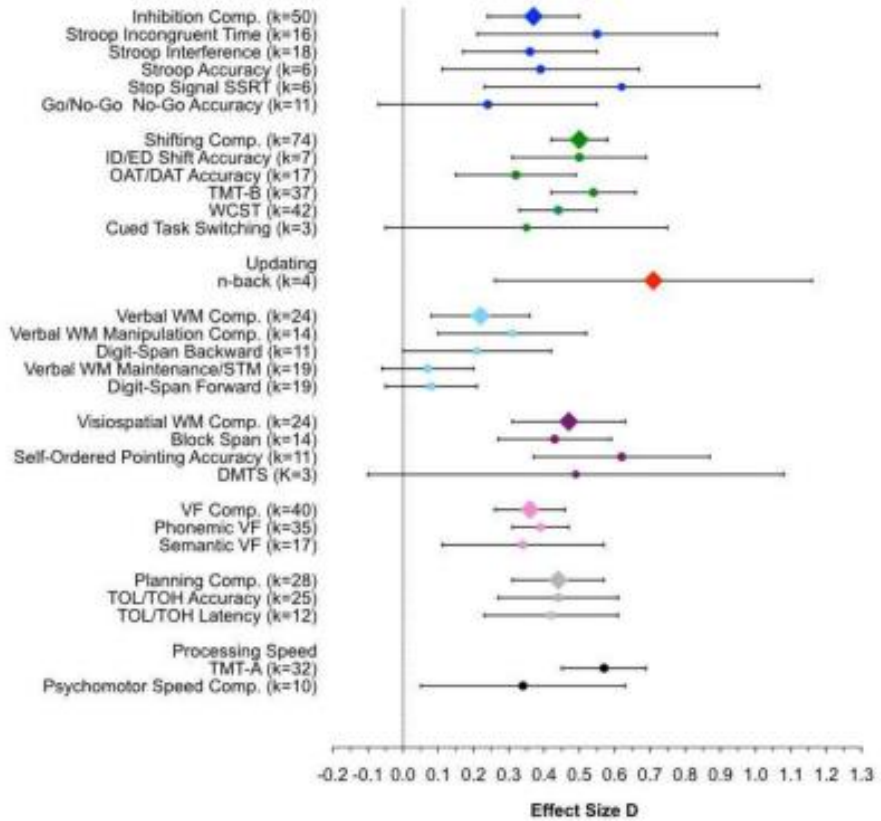
Obsessive-compulsive disorder is associated with broad impairments in executive function: A meta-analysis

Hannah R. Snyder[a,*], **Roselinde H. Kaiser**[b], **Stacie L. Warren**[c], and **Wendy Heller**[d]



Comprehensive Psychiatry 83 (2018) 1–6

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Comprehensive Psychiatry

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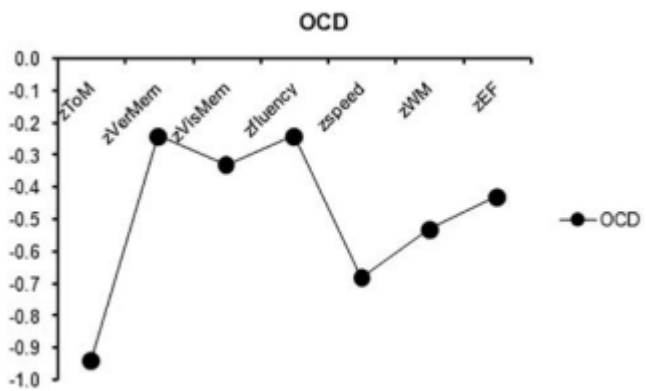
Relationship between social-cognitive and social-perceptual aspects of theory of mind and neurocognitive deficits, insight level and schizotypal traits in obsessive-compulsive disorder

Emre Mısır [a,*], Emre Bora [a,b], Berna Binnur Akdede [a]



Psychiatry Research 316 (2022) 114752

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Review article

Social cognition and empathy in adults with obsessive compulsive disorder: A meta-analysis

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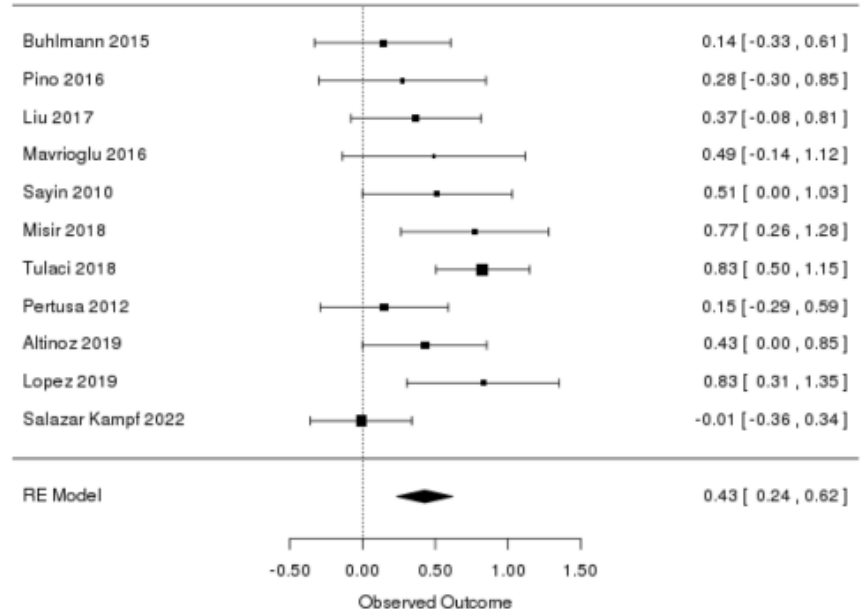
ARTICLE INFO

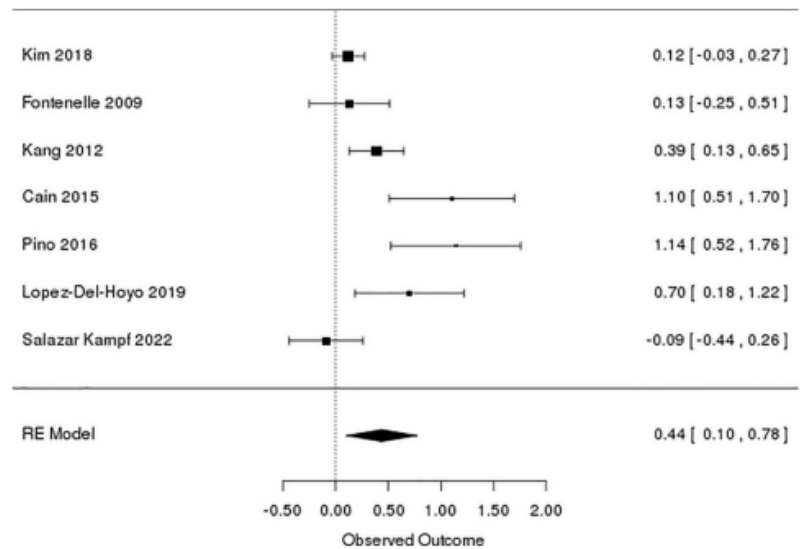
Keywords:
Obsessive-compulsive disorder
Social cognition
Theory of mind
Empathy
Emotion recognition

ABSTRACT

Obsessive-compulsive disorder (OCD) is characterized by intrusive thoughts (obsessions) and compulsions and has been associated with psychosocial impairment. Indeed, a number of studies have highlighted impairments in both social cognitive functions and empathic skills in OCD, despite several inconsistencies. This study aimed to investigate social cognitive dysfunction and empathy deficits in patients with OCD using a meta-analytic approach. A literature search was conducted using the databases Pubmed, PsycINFO, ProQuest and Scopus to identify the relevant studies (January 1980 to March 2020). Following the systematic review of relevant OCD studies, a random-effects meta-analysis was conducted. The current meta-analysis included 25 studies consisting of 1161 patients with OCD and 1329 healthy controls. OCD was associated with decreased performance in theory of mind (ToM). In the facial emotion recognition domain, patients with OCD significantly underperformed healthy controls only in their recognition of disgust. OCD was significantly related to reduced cognitive empathy. OCD was associated with medium-sized impairments in ToM and cognitive empathy, which can likely contribute to psychosocial impairment in this disorder. Further studies are needed to investigate state and trait-related factors using experimental measures of empathy.



2. Forest plot of effect sizes and 95% confidence intervals for ToM diffences between patients with OCD and healthy controls using random effects model.



st plot of effect sizes and 95% confidence intervals for cognitive empathy differences between patients with OCD and healthy controls using random
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BRIEF RESEARCH REPORT
published: 09 May 2022
doi: 10.3389/fpsyt.2022.868921

Cognitive and Experienced Flexibility in Patients With Anorexia Nervosa and Obsessive Compulsive Disorder

Lot Catharina Sternheim[1], Boris van Passel[2,3], Alexandra Dingemans[4], Danielle Cath[5,6] and Unna Nora Danner[1,7]*

[1] Department of Clinical Psychology, Utrecht University, Utrecht, Netherlands, [2] Centre for Anxiety Disorders, Overwaal

Group Embedded Figures Test
CC

ID/EDS[§]					
No. people stage 9 not reached	21	27	23	0.20	0.91
No. trials to reach stage 9 (ED switch-cost)	16.51 (9.30)	15.26 (11.60)	17.39 (10.71)	0.52	0.60
CWIT-rigidity[†,¶]					
Time in sec	52.73 (8.92)	58.98 (16.36)	53.03 (12.17)	1.77	0.17
Error	1.25 (1.47)	1.01 (1.47)	0.98 (1.09)	1.20	0.30
GEFT[††,‡‡]					
No. errors	1.68 (2.04)	3.35 (3.42)	0.46 (1.05)	20.52	<0.001
Time in sec	173.01 (62.33)	190.80 (70.26)	No data	1.74	0.19
DFlex					
Cognitive rigidity	44.48 (11.10)	43.54 (12.76)	27.04 (7.17)	55.35	<0.001
Attention to detail	38.72 (11.73)	40.99 (12.97)	24.85 (8.48)	39.57	<0.001

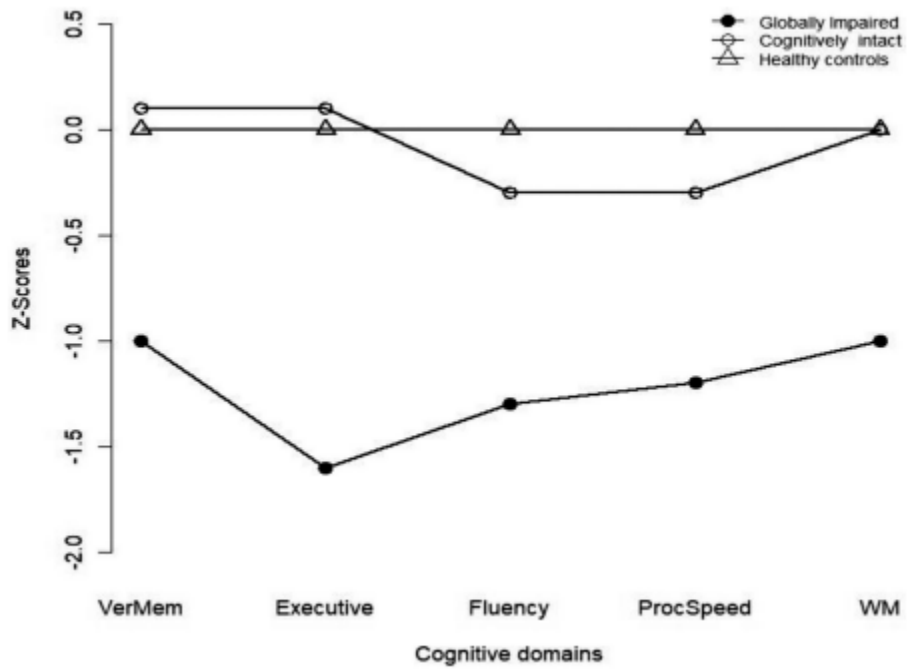
AN, Anorexia Nervosa; OCD, Obsessive-compulsive disorder; HC, Healthy Controls; DART, Dutch adult reading test; BMI, Body mass index; EDEQ, Eating Disorder E

Brain and Behavior
Open Access

ORIGINAL ARTICLE OPEN ACCESS

Clinical Characteristics of Cognitive Subgroups of Obsessive Compulsive Disorder

Emre Mısır[1,2] | Raşit Tükel[3] | Berna Binnur Akdede[4,5] | Emre Bora[4,5]



	Globally impaired ($n = 42$)	Cognitively intact ($n = 93$)	HCs ($n = 106$)	Statistics	Post-hoc
Age	34.79 (11.48)	27.15 (7.44)	29.91 (9.46)	$F(238) = 8.63$ $p < 0.001$	GI > CI = HC
Gender (F/M)	34/8	55/38	56/50	χ^2 (2) = 9.99 $p = 0.007^{**}$	
Education	7.76 (3.27)	12.63 (3.34)	12.51 (4.44)	$F(238) = 35.27$ $p < 0.001$	CI = HC > GI
Married/no married[a]	21/21	29/64	50/56	χ^2 (2) = 6.73 $p = 0.035$	—
Employed/unemployed	16/26	30/63	51/55	χ^2 (2) = 5.28 $p = 0.07$	—
AoO	25.96 (8.82)	20.46 (5.71)		$t(132) = -4.24$ $p < 0.001$	
Disease duration	8.82 (8.46)	6.59 (6.86)		$t(132) = -1.62$ $p = 0.11$	
Medication (Yes/No)	14/28	27/64		χ^2 (1) = 0.09 $p = 0.76$	
Y-BOCS total	25.93 (6.35)	23.24 (7.15)		$t(133) = -2.09$ $p = 0.038$	
Y-BOCS-O	12.95 (3.99)	12.06 (3.59)		$t(133) = -1.35$ $p = 0.18$	
Y-BOCS-C	13.21 (3.92)	11.27 (4.41)		$t(133) = -2.45$ $p = 0.015$	
HDRS-17	6.64 (3.95)	6.22 (4.09)	1.6 (2)	$F(238) = 60.49$ $p<0.001$	CI = GI > HC

Cortical and Subcortical Brain Morphometry Differences Between Patients With Autism Spectrum Disorder and Healthy Individuals Across the Lifespan: Results From the ENIGMA ASD Working Group

Daan van Rooij, Ph.D., Evdokia Anagnostou, M.D., Celso Arango, M.D., Ph.D., Guillaume Auzias, Ph.D., Marlene Behrmann, Ph.D., Geraldo F. Busatto, M.D., Ph.D., Sara Calderoni, M.D., Ph.D., Eileen Daly, Ph.D., Christine Deruelle, Ph.D., Adriana Di Martino, Ph.D., Ilan Dinstein, Ph.D., Fabio Luis Souza Duran, Ph.D., Sarah Durston, Ph.D., Christine Ecker, Ph.D., Damien Fair, Ph.D., Jennifer Fedor, B.S., Jackie Fitzgerald, Ph.D., Christine M. Freitag, Ph.D., Louise Gallagher, M.D., Ph.D., Ilaria Gori, M.Sc., Shlomi Haar, Ph.D., Liesbeth Hoekstra, M.Sc., Neda Jahanshad, Ph.D., Maria Jalbrzikowski, Ph.D., Joost Janssen, Ph.D., Jason Lerch, Ph.D., Beatriz Luna, Ph.D., Mauricio Moller Martinho, M.D., Ph.D., Jane McGrath, Ph.D., Filippo Muratori, Ph.D., Clodagh M. Murphy, M.R.C.Psych., Ph.D., Declan G.M. Murphy, M.D., F.R.C.Psych., Kirsten O'Hearn, Ph.D., Bob Oranje, Ph.D., Mara Parellada, M.D., Ph.D., Alessandra Retico, Ph.D., Pedro Rossa, M.D., Katya Rubia, Ph.D., Devon Shook, Ph.D., Margot Taylor, Ph.D., Paul M. Thompson, Ph.D., Michela Tosetti, Ph.D., Gregory L. Wallace, Ph.D., Fengfeng Zhou, Ph.D., Jan K. Buitelaar, M.D., Ph.D.

Characteristic	Control Group (N=1,651)		ASD Group (N=1,571)	
	Mean	SD	Mean	SD
Age[a] (years)	15.83	8.41	15.41	8.64
IQ[b,c]	111	19.04	103	20.02
	N	%	N	%
Female[b]	393	23.8	224	14.3
Medication use[d]	0	0.0	233	14.8
Comorbidities[e]	0	0.0	148	9.4

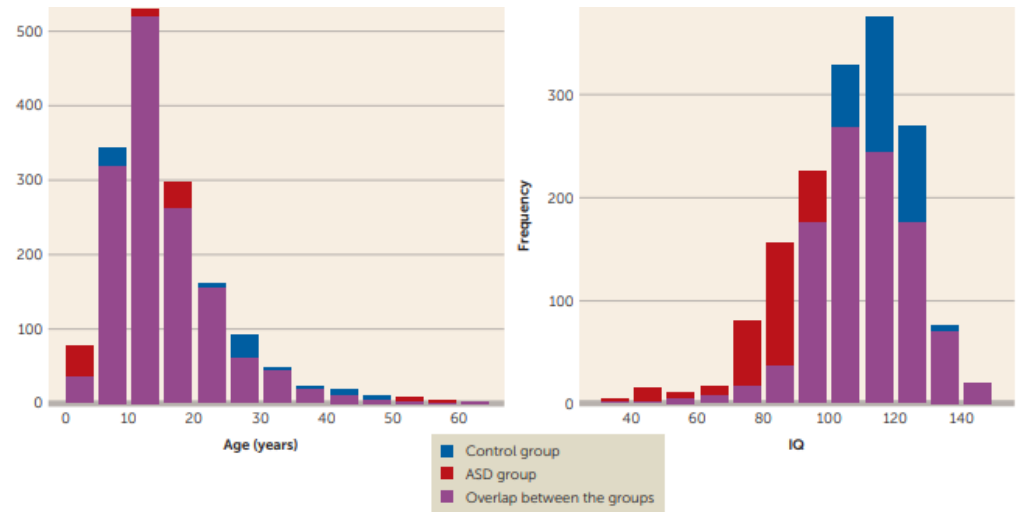
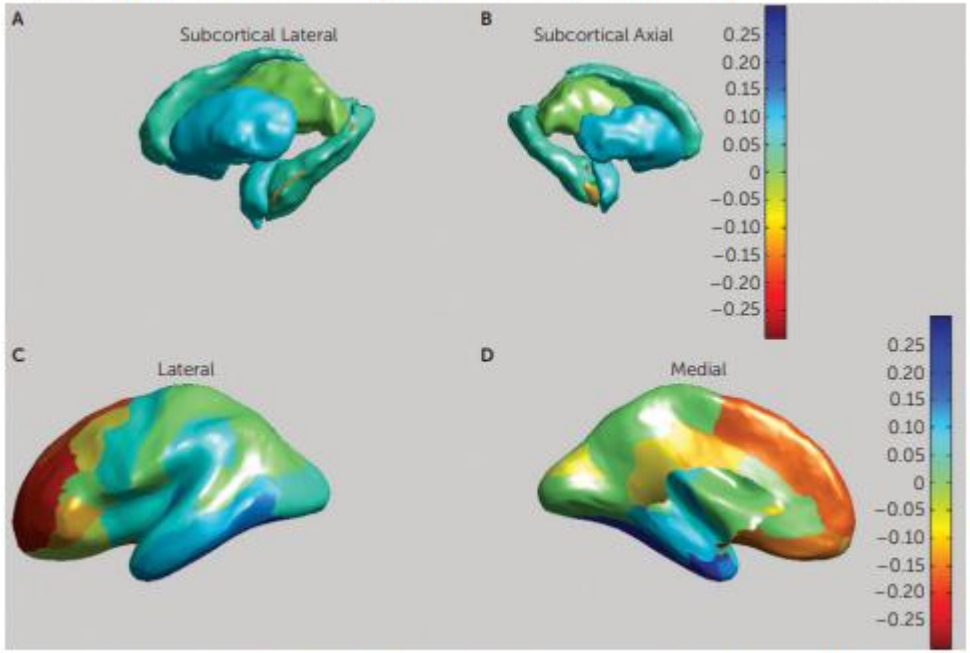


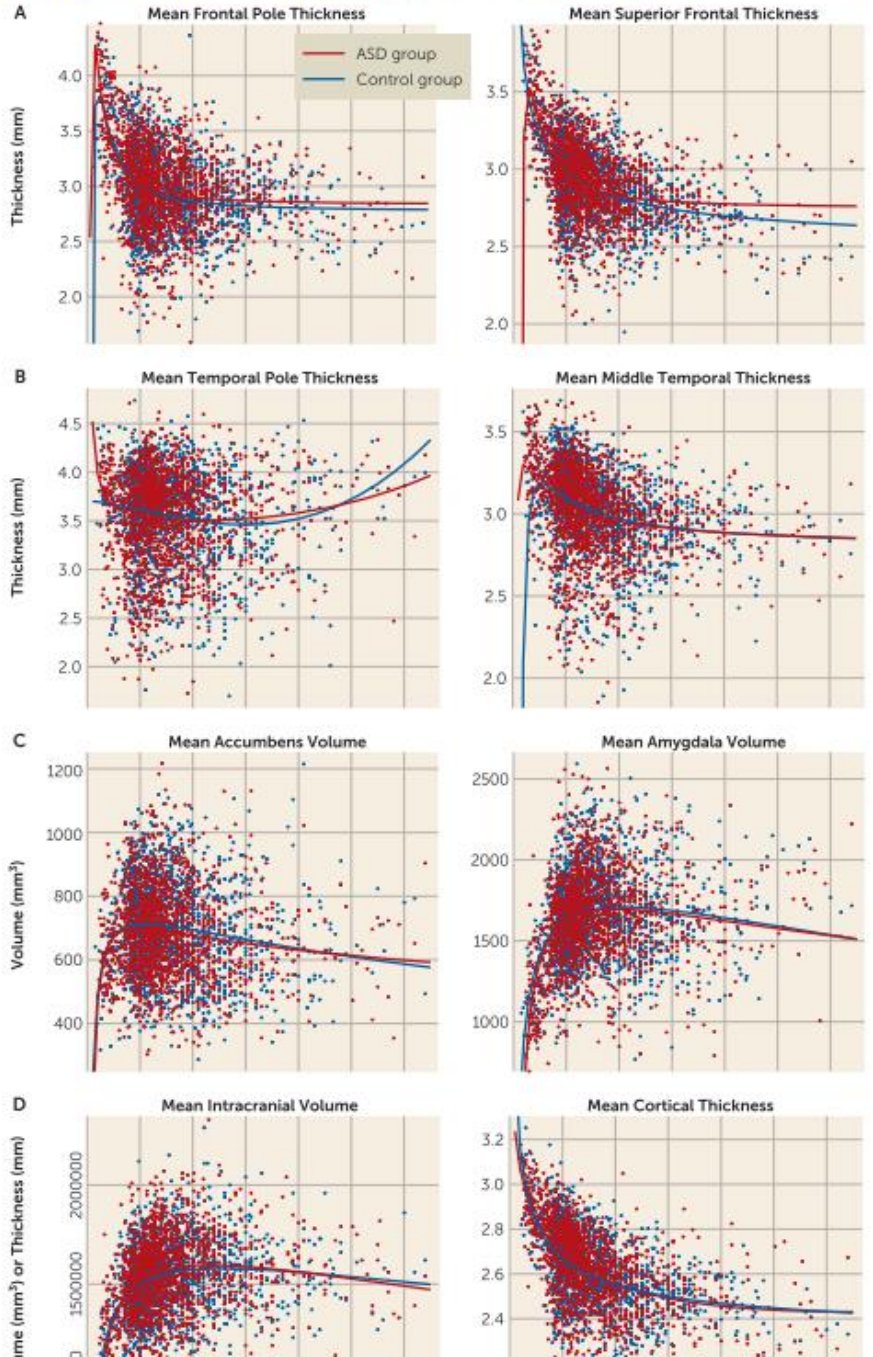
FIGURE 2. Effect Sizes for all Subcortical and Cortical Partitions in a Mega-Analysis of Brain Morphometry in Patients With Autism Spectrum Disorder (ASD) and Healthy Control Subjects[a]



[a] Panels A and B are medial and lateral views of the striatum. Panels C and D are medial and lateral views of cortical thickness. Yellow to red hues indicate higher d values, corresponding to larger volumes in patients with ASD. Blue hues indicate lower volumes in subjects with ASD. Images are in Montreal Neurological Institute space (MNI152).

Frontal korteks kalınlık artışı ve temporal loba azalması
Yaş-tanı etkisi: Bu farklar ergenlerde tepe noktası
Subkortikal hacim azalma (Putamen, pallidum, nucleus accumbens)

FIGURE 3. Fractional Polynomial Best Model Fits for Age in a Mega-Analysis of Brain Morphometry in Patients With Autism Spectrum Disorder (ASD) and Healthy Control Subjects[a]



nature COMMUNICATIONS

ARTICLE

https://doi.org/10.1038/s41467-019-13005-8 OPEN

Altered structural brain asymmetry in autism spectrum disorder in a study of 54 datasets

Merel C. Postema et al.#

Altered structural brain asymmetry in autism spectrum disorder (ASD) has been reported. However, findings have been inconsistent, likely due to limited sample sizes. Here we investigated 1,774 individuals with ASD and 1,809 controls, from 54 independent data sets of the ENIGMA consortium. ASD was significantly associated with alterations of cortical thickness asymmetry in mostly medial frontal, orbitofrontal, cingulate and inferior temporal areas, and also with asymmetry of orbitofrontal surface area. These differences generally involved reduced asymmetry in individuals with ASD compared to controls. Furthermore, putamen volume asymmetry was significantly increased in ASD. The largest case-control effect size was Cohen's $d = -0.13$, for asymmetry of superior frontal cortical thickness. Most effects did not depend on age, sex, IQ, severity or medication use. Altered lateralized neurodevelopment may therefore be a feature of ASD, affecting widespread brain regions with diverse functions. Large-scale analysis was necessary to quantify subtle alterations of brain structural asymmetry in ASD.

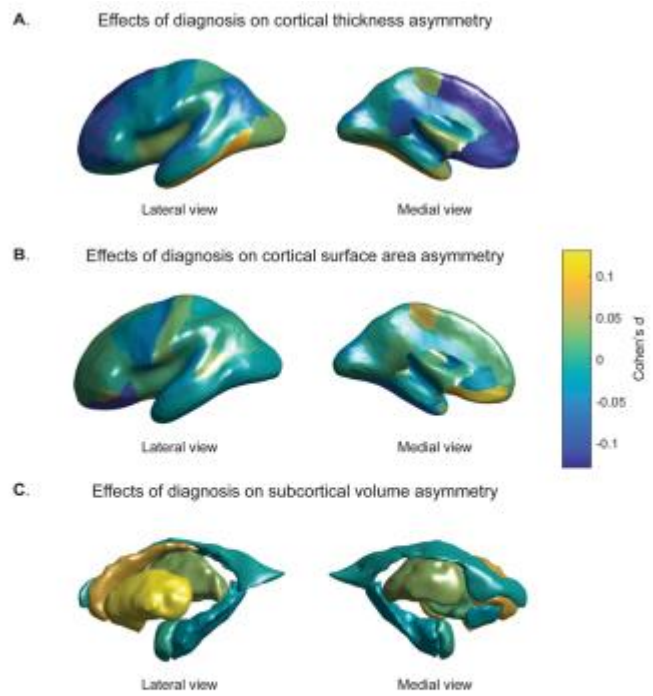


Fig. 1 Cohen's *d* effect sizes of the associations between diagnosis and AIs. **a** regional cortical thickness measures, **b** cortical surface areas, **c** subcortical volumes. Values are overlaid on left hemisphere inflated brains. Positive Cohen's *d* values (yellow) indicate mean shifts towards greater leftward or reduced rightward asymmetry in cases, and negative Cohen's *d* values (blue) indicate mean shifts towards greater rightward asymmetry or reduced leftward asymmetry in individuals with ASD

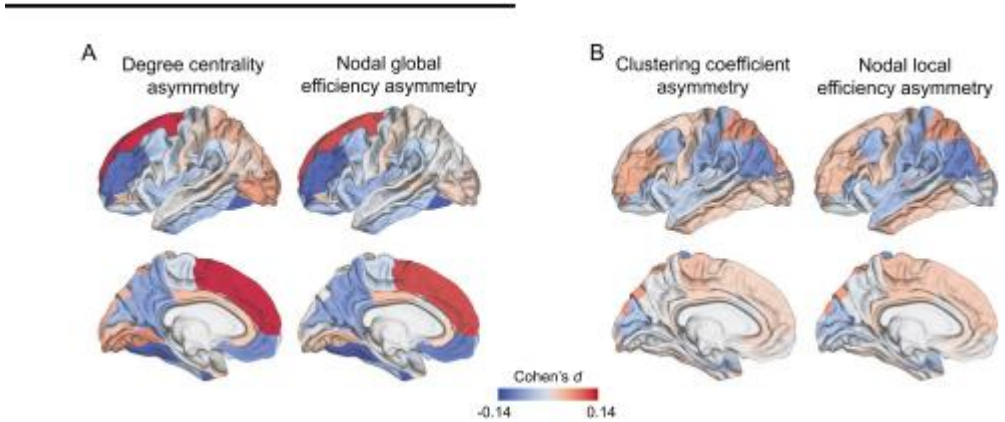
Asimetri de azalma. En belirgin superior frontal korteks

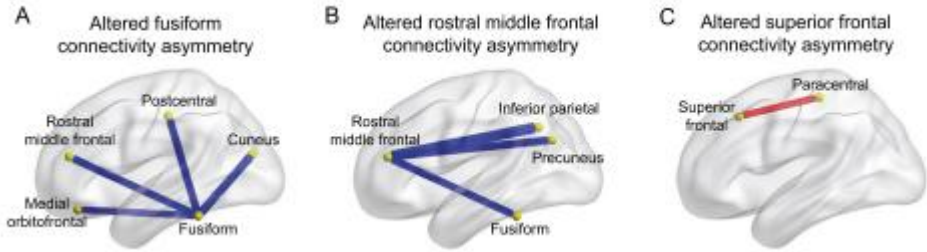
ARTICLE OPEN

Check for updates

Subtly altered topological asymmetry of brain structural covariance networks in autism spectrum disorder across 43 datasets from the ENIGMA consortium

Zhiqiang Sha[1✉], Daan van Rooij[2], Evdokia Anagnostou[3], Celso Arango[4], Guillaume Auzias[5], Marlene Behrmann[6], Boris Bernhardt[7], Sven Bolte[8,9,10], Geraldo F. Busatto[11], Sara Calderoni[12,13], Rosa Calvo[14], Eileen Daly[15], Christine Deruelle[5]



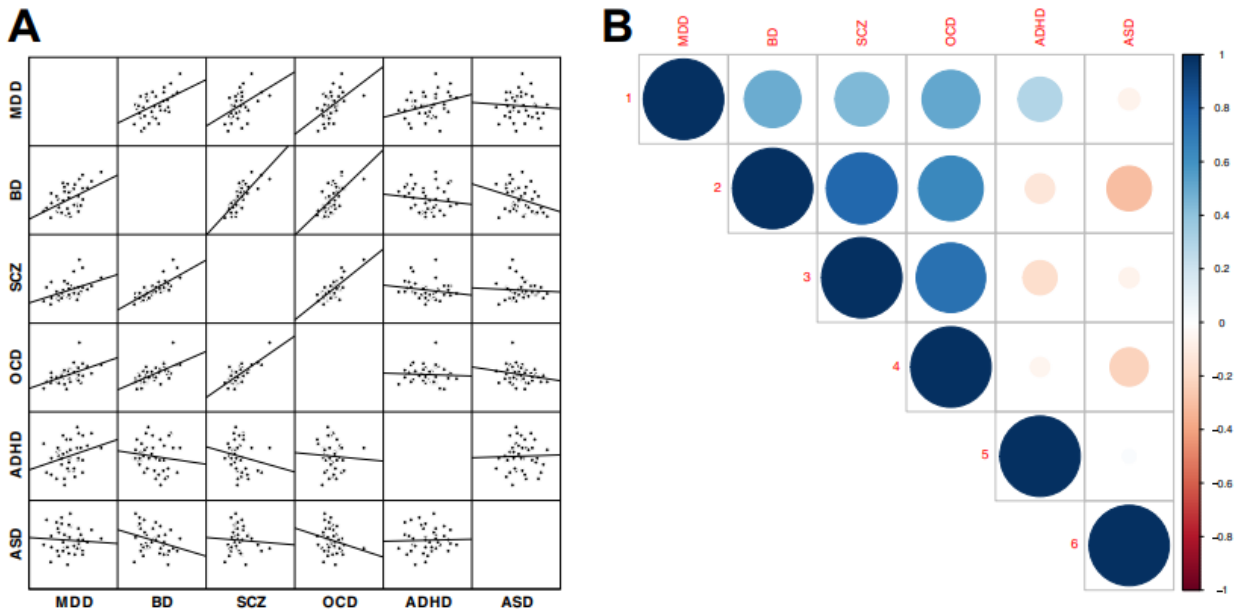


Biological Psychiatry

Archival Report

Cross-Disorder Analysis of Brain Structural Abnormalities in Six Major Psychiatric Disorders: A Secondary Analysis of Mega- and Meta-analytical Findings From the ENIGMA Consortium

Nils Opel, Janik Goltermann, Marco Hermesdorf, Klaus Berger, Bernhard T. Baune, and Udo Dannlowski

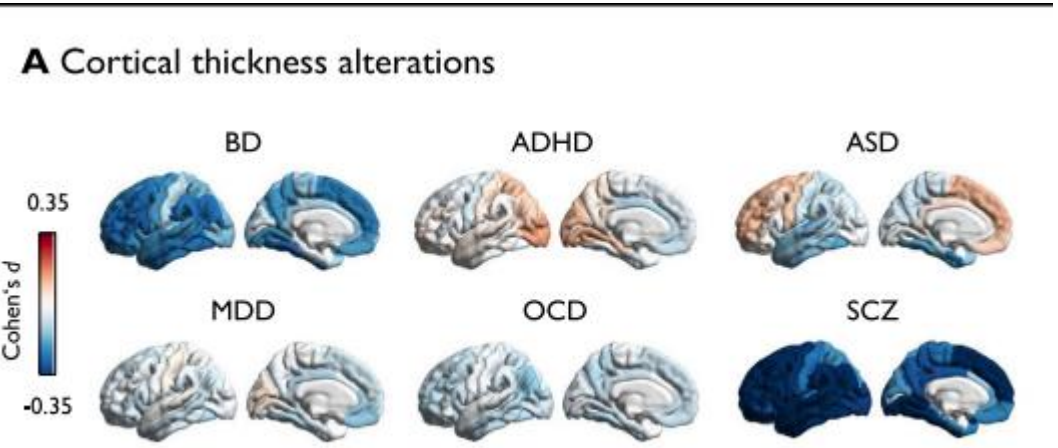


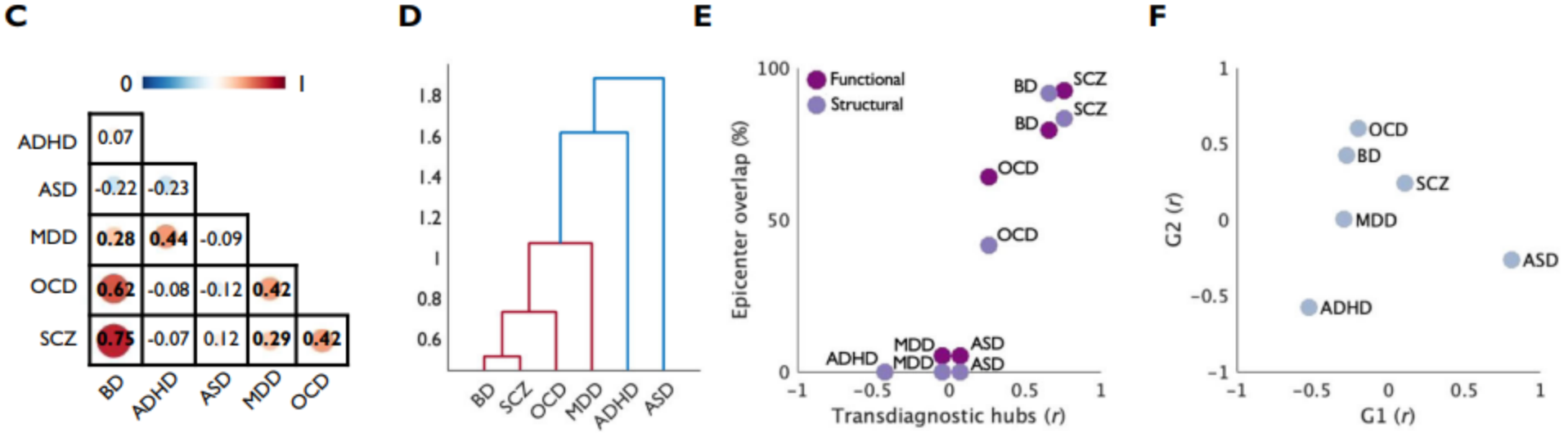
nature communications

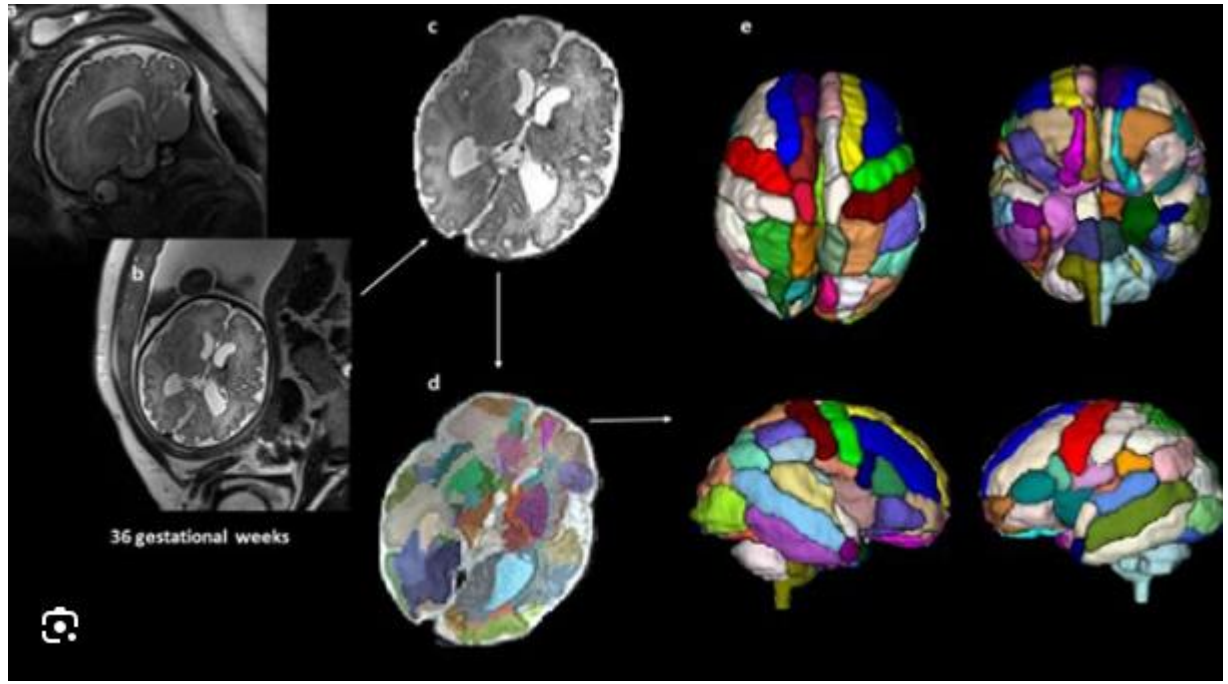
Article

https://doi.org/10.1038/s41467-022-34367-6

Coordinated cortical thickness alterations across six neurodevelopmental and psychiatric disorders





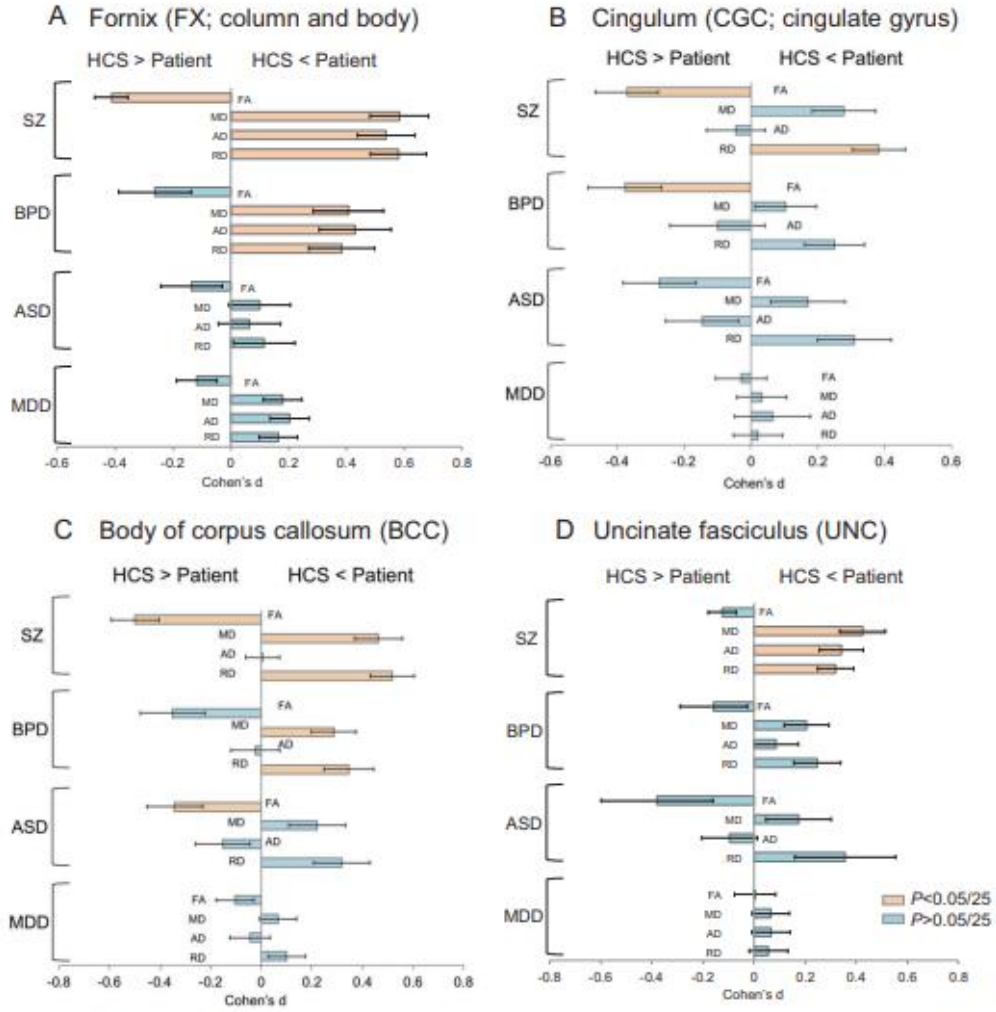

b
c
d
e
36 gestational weeks

Molecular Psychiatry (2020) 25:883–895
https://doi.org/10.1038/s41380-019-0553-7

ARTICLE

White matter microstructural alterations across four major psychiatric disorders: mega-analysis study in 2937 individuals

Daisuke Koshiyama[1] · Masaki Fukunaga[2] · Naohiro Okada[1,3] · Kentaro Morita[1] · Kiyotaka Nemoto[4] · Kaori Usui[1] · Hidenaga Yamamori[5] · Yuka Yasuda[6,7] · Michiko Fujimoto[5] · Noriko Kudo[7] · Hirotsugu Azechi[8] · Yoshiyuki Watanabe[9] · Naoki Hashimoto[10] · Hisashi Narita[10] · Ichiro Kusumi[10] · Kazutaka Ohi[11,12] ·



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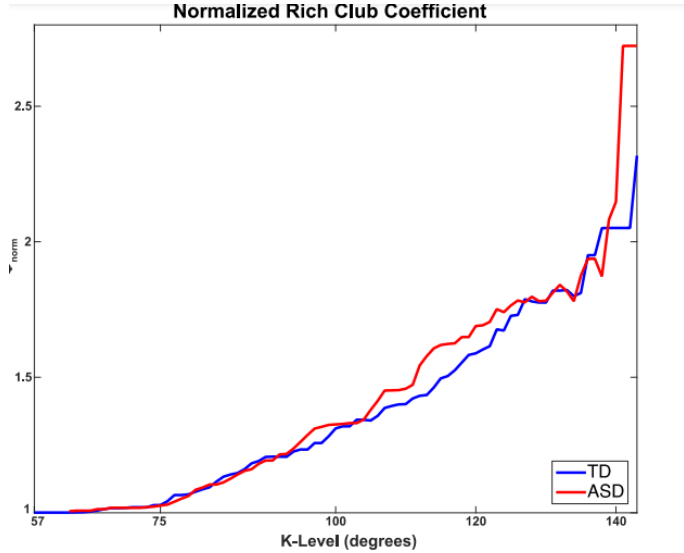
Biol Psychiatry Cogn Neurosci Neuroimaging. 2017 January ; 2(1): 66–75. doi:10.1016/j.bpsc.2016.07.008.

Network organization is globally atypical in autism: A graph theory study of intrinsic functional connectivity

Christopher L. Keown[a,b], **Michael C. Datko**[a,b], **Colleen P. Chen**[a,c], **José Omar Maximo**[a], **Afrooz Jahedi**[a,d], and **Ralph-Axel Müller**[a,*]



OSB zengin klüp bağlantısallık artış

SCIENTIFIC REPORTS

OPEN

Age-associated changes in rich-club organisation in autistic and neurotypical human brains

Received: 23 June 2015
Accepted: 05 October 2015

Takamitsu Watanabe[1] & Geraint Rees[1,2]

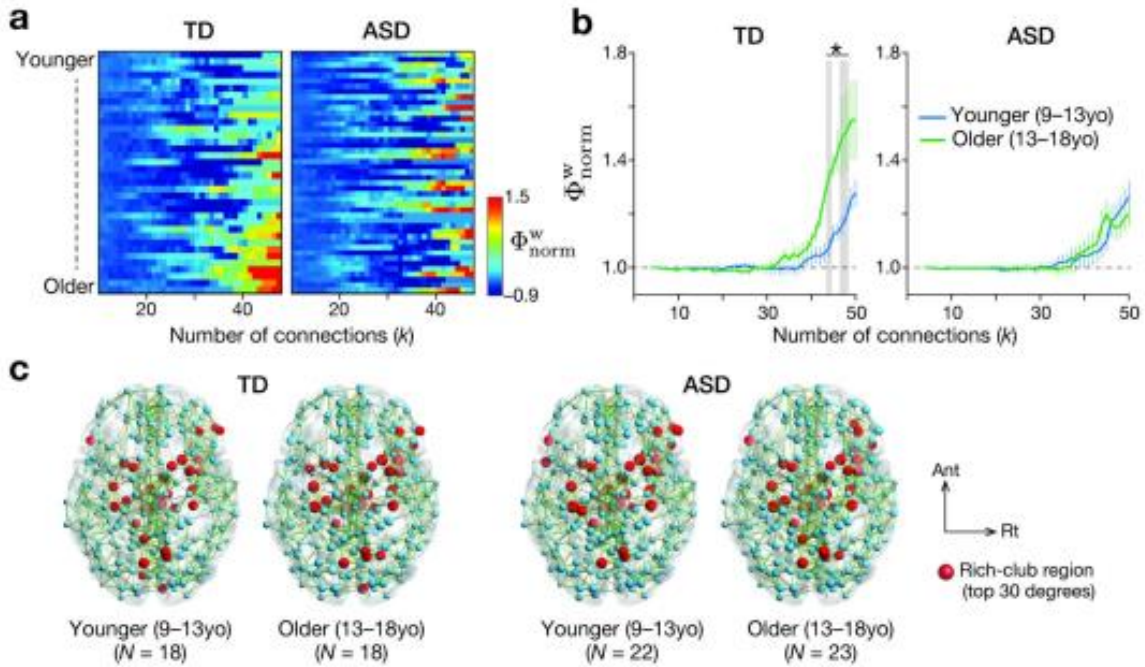


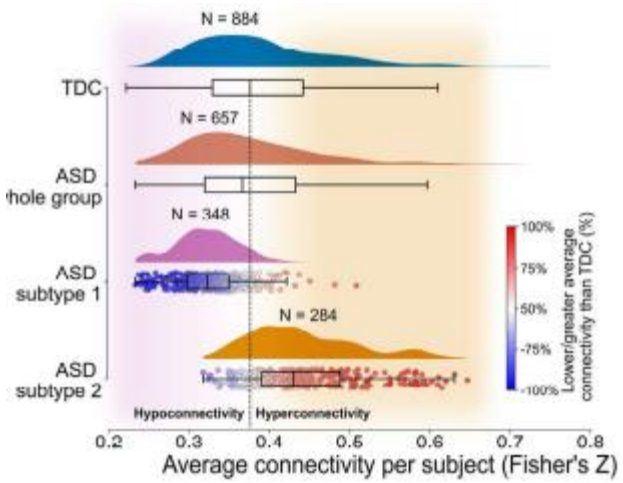
Figure 1. (a) Normalised rich-club coefficients for all individuals were sorted by age. TD, typically

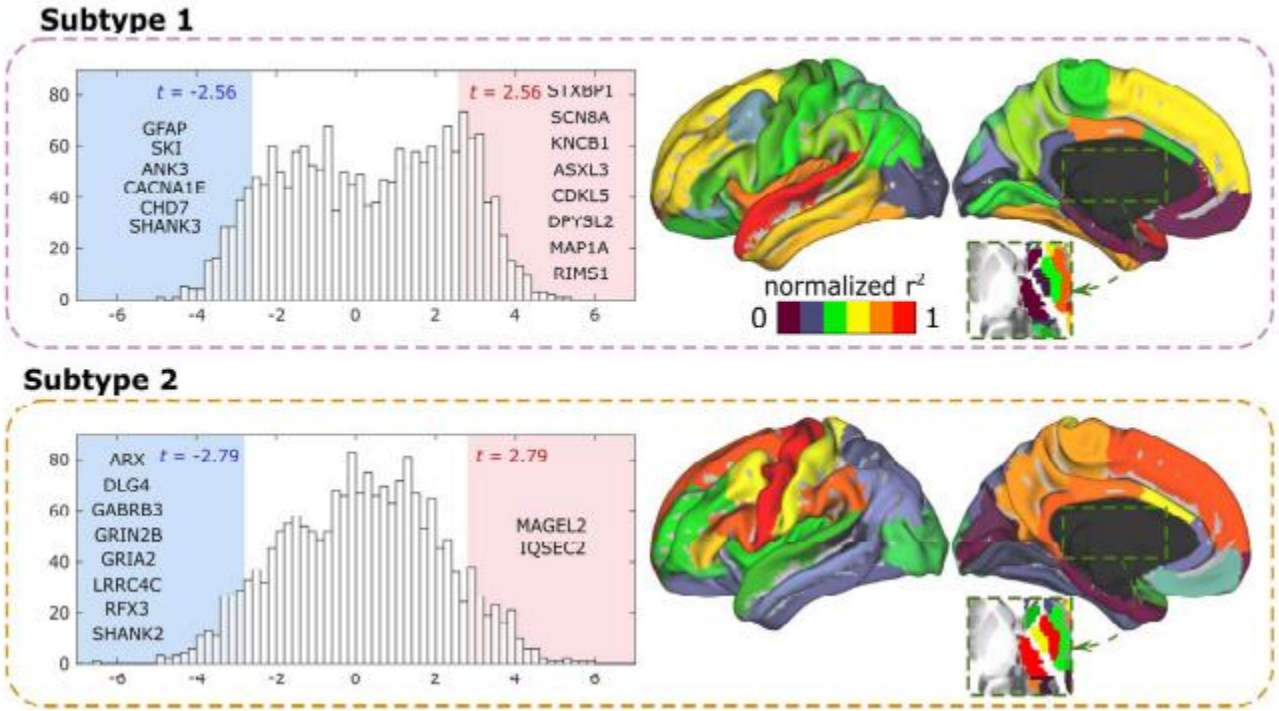
Biological Psychiatry

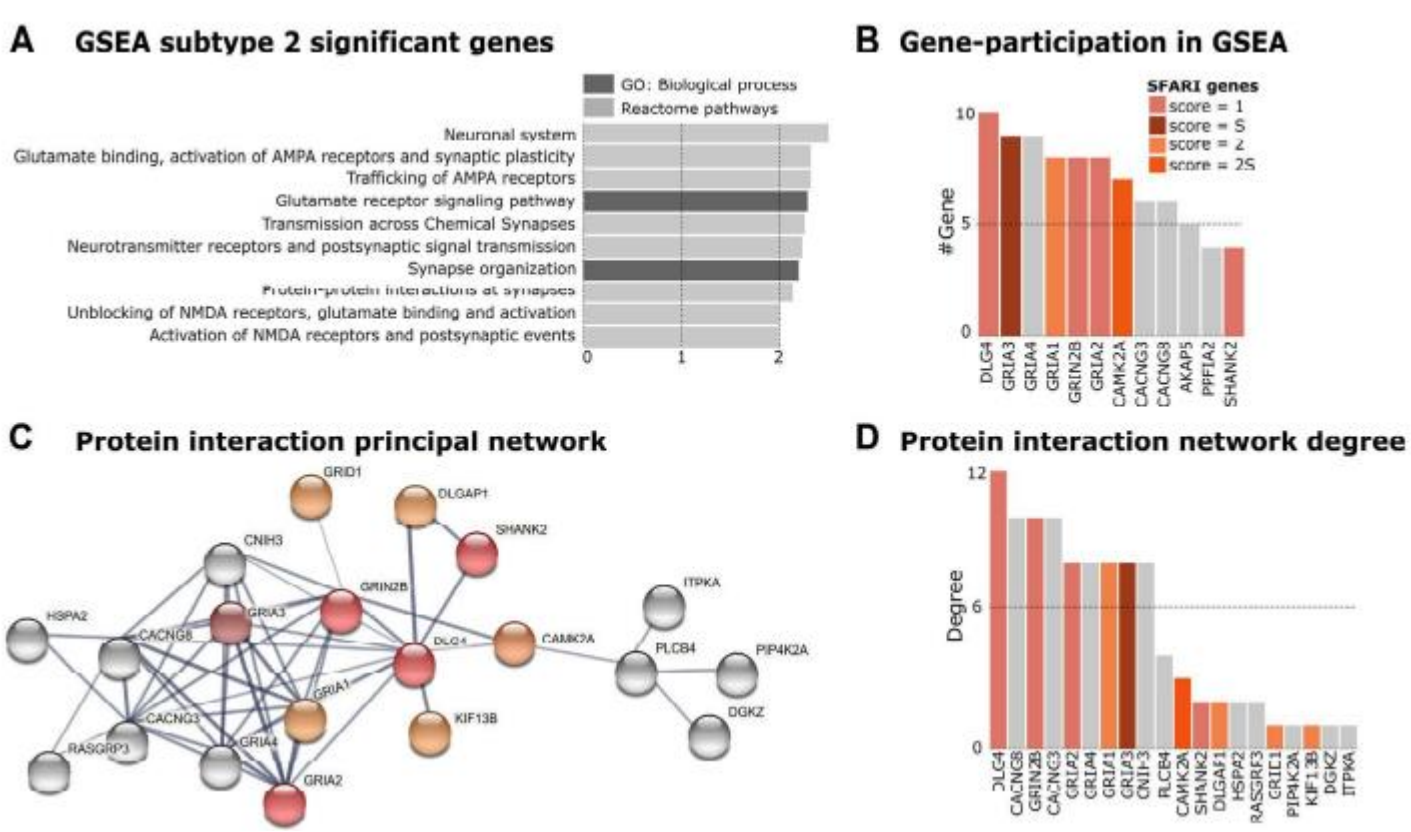
Archival Report

The Neurogenetics of Functional Connectivity Alterations in Autism: Insights From Subtyping in 657 Individuals

Javier Rasero, Antonio Jimenez-Marin, Ibai Diez, Roberto Toro, Mazahir T. Hasan, and Jesus M. Cortes






A
GSEA subtype 2 significant genes
GO: Biological process
Reactome pathways
Neuronal system
Glutamate binding, activation of AMPA receptors and synaptic plasticity
Trafficking of AMPA receptors
Glutamate receptor signaling pathway
Transmission across Chemical Synapses
Neurotransmitter receptors and postsynaptic signal transmission
Synapse organization
Protein-protein interactions at synapses
Unblocking of NMDA receptors, glutamate binding and activation
Activation of NMDA receptors and postsynaptic events
0
1
2
B
Gene-participation in GSEA
SFARI genes
score = 1
score = S
score = 2
score = 2S
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10
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DLG4
GRIA3
GRIA4
GRIA1
GRIN2B
GRIA2
CAMK2A
CACNG3
CACNG8
AKAP5
PPFIA2
SHANK2
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Protein interaction principal network
GRID1
DLGAP1
SHANK2
CNIH3
GRIN2B
ITPKA
HSPA2
GRIA3
CACNG8
DLG4
CAMK2A
PLCB4
PIP4K2A
GRIA1
CACNG3
KIF13B
DGKZ
RASGRP1
GRIA4
GRIA2
D
Protein interaction network degree
Degree
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6
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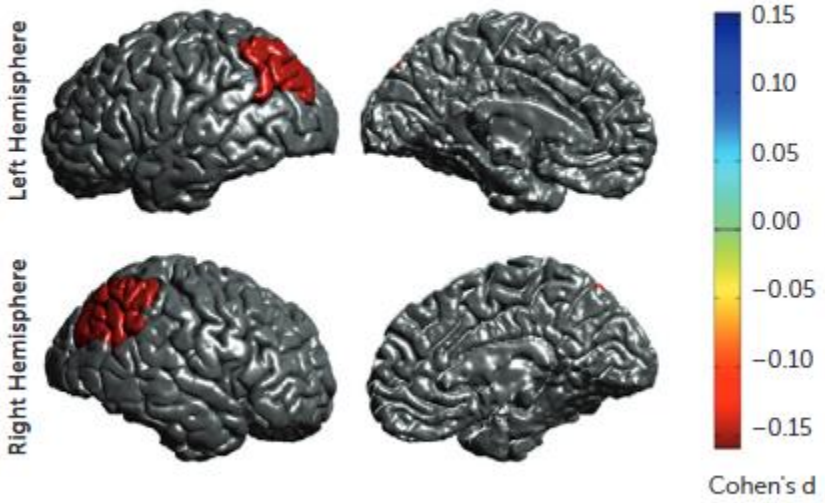
DLG4
CACNG8
GRIN2B
CACNG3
GRIA2
GRIA4
GRIA1
GRIA3
CNIH3
PLCB4
CAMK2A
SHANK2
DLGAP1
HSPA2
RASGRF3
GRID1
PIP4K2A
KIF13B
DGKZ
ITPKA

Cortical Abnormalities Associated With Pediatric and Adult Obsessive-Compulsive Disorder: Findings From the ENIGMA Obsessive-Compulsive Disorder Working Group

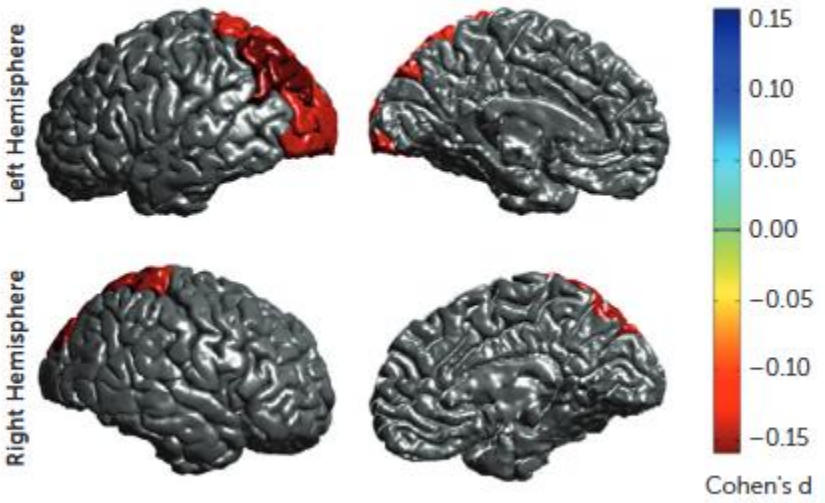
Premika S.W. Boedhoe, M.Sc., Lianne Schmaal, Ph.D., Yoshinari Abe, M.D., Pino Alonso, M.D., Ph.D.,

FIGURE 1. Mega-Analysis Effect Sizes for Regions That Showed a Significant (q<0.05) difference in Cortical Thickness Between Adult OCD Patients and Healthy Controls[a]



[a] Negative effect sizes (shown in red) indicate thinner cortices in OCD patients compared with controls.

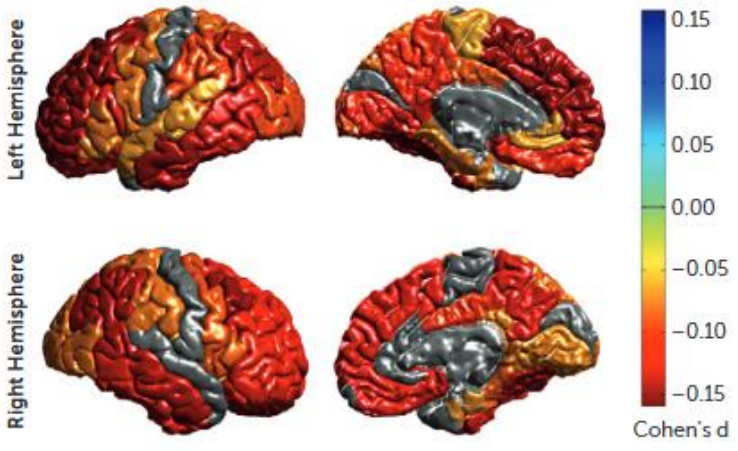
FIGURE 2. Mega-Analysis Effect Sizes for Regions That Showed a Significant (q<0.05) difference in Cortical Thickness Between Pediatric OCD Patients and Healthy Controls[a]



[a] Negative effect sizes (shown in red) indicate thinner cortices in OCD patients compared with controls.

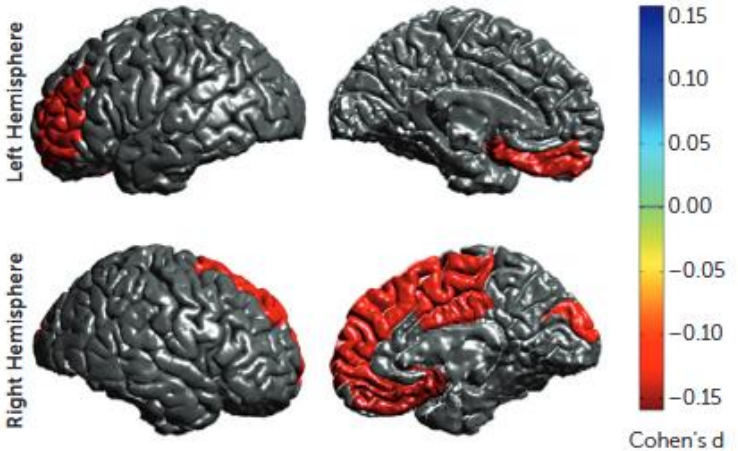
Parietal korteks kalınlık azalma

FIGURE 3. Mega-Analysis Effect Sizes for Regions That Showed a Significant (q<0.05) Difference in Cortical Thickness Between Medicated Adult OCD Patients and Healthy Controls[a]



[a] Negative effect sizes (shown in red, orange, and yellow) indicate thinner cortices in OCD patients compared with controls.

FIGURE 4. Mega-Analysis Effect Sizes for Regions That Showed a Significant (q<0.05) Difference in Cortical Surface Area Between Medicated Pediatric OCD Patients and Healthy Controls[a]



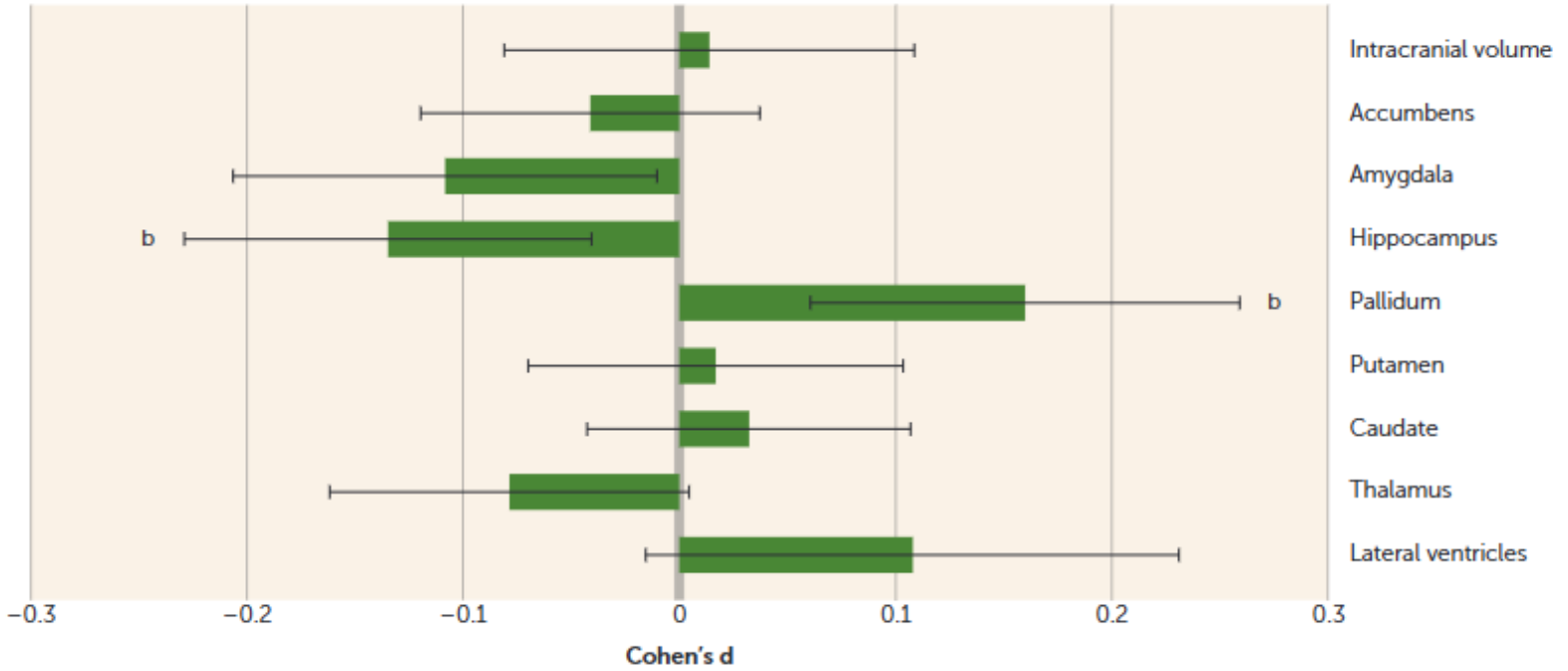
[a] Negative effect sizes (shown in red) indicate reduced cortical surface area in OCD patients compared with controls.

Tedavi alan hastalarda daha yaygın kortikal kalınlık azalma, AD+AP daha da çok
Tedavi etkisi?
Klinik şiddet etkisi?

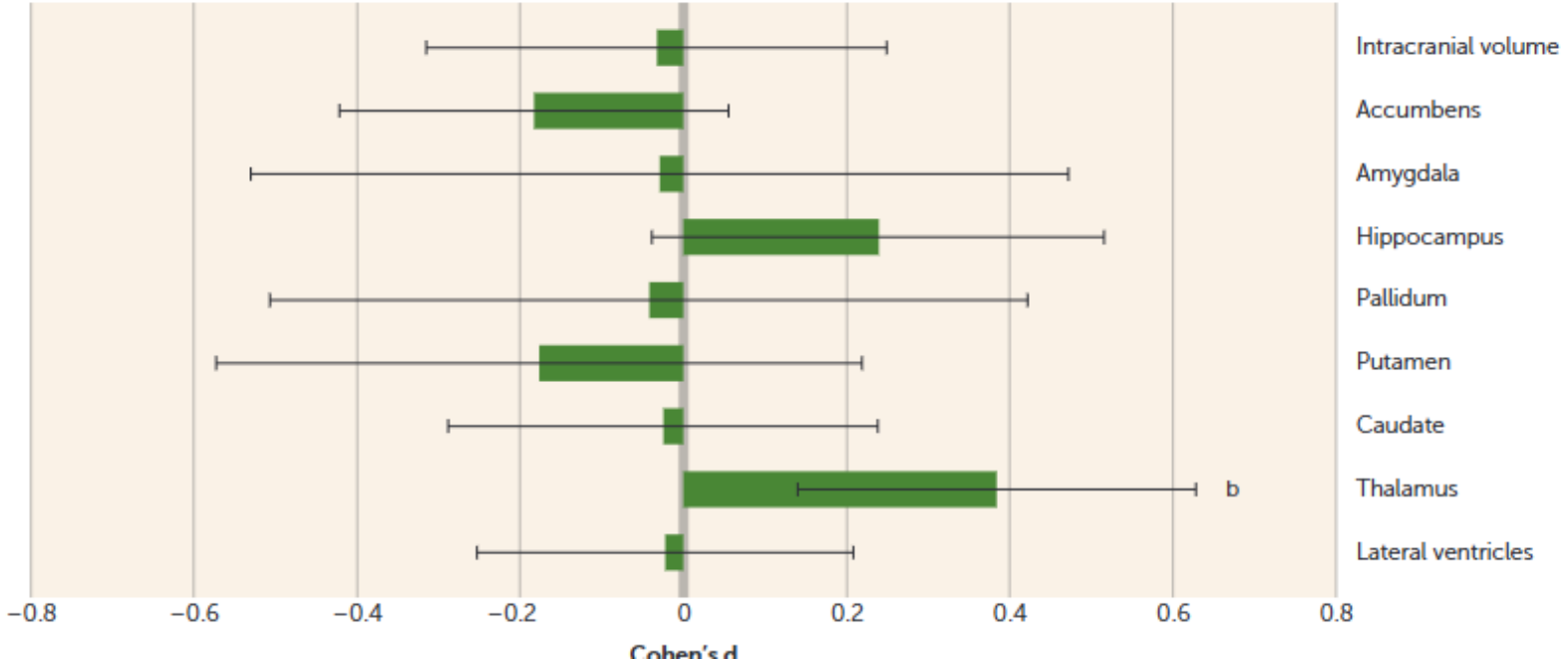
Distinct Subcortical Volume Alterations in Pediatric and Adult OCD: A Worldwide Meta- and Mega-Analysis

Premika S.W. Boedhoe, M.Sc., Lianne Schmaal, Ph.D., Yoshinari Abe, M.D., Stephanie H. Ameis, M.D., Paul D. Arnold, M.D., Ph.D., Marcelo C. Batistuzzo, Ph.D., Francesco Benedetti, M.D., Jan C. Beucke, Ph.D.,

A. Subcortical Volume Differences in OCD Patients Compared With Control Subjects



B. Subcortical Volume Differences in Unmedicated Pediatric OCD Patients Compared With Pediatric Control Subjects



Biological Psychiatry

Archival Report

Mapping Cortical and Subcortical Asymmetry in Obsessive-Compulsive Disorder: Findings From the ENIGMA Consortium

Xiang-Zhen Kong, Premika S.W. Boedhoe, Yoshinari Abe, Pino Alonso, Stephanie H. Ameis,

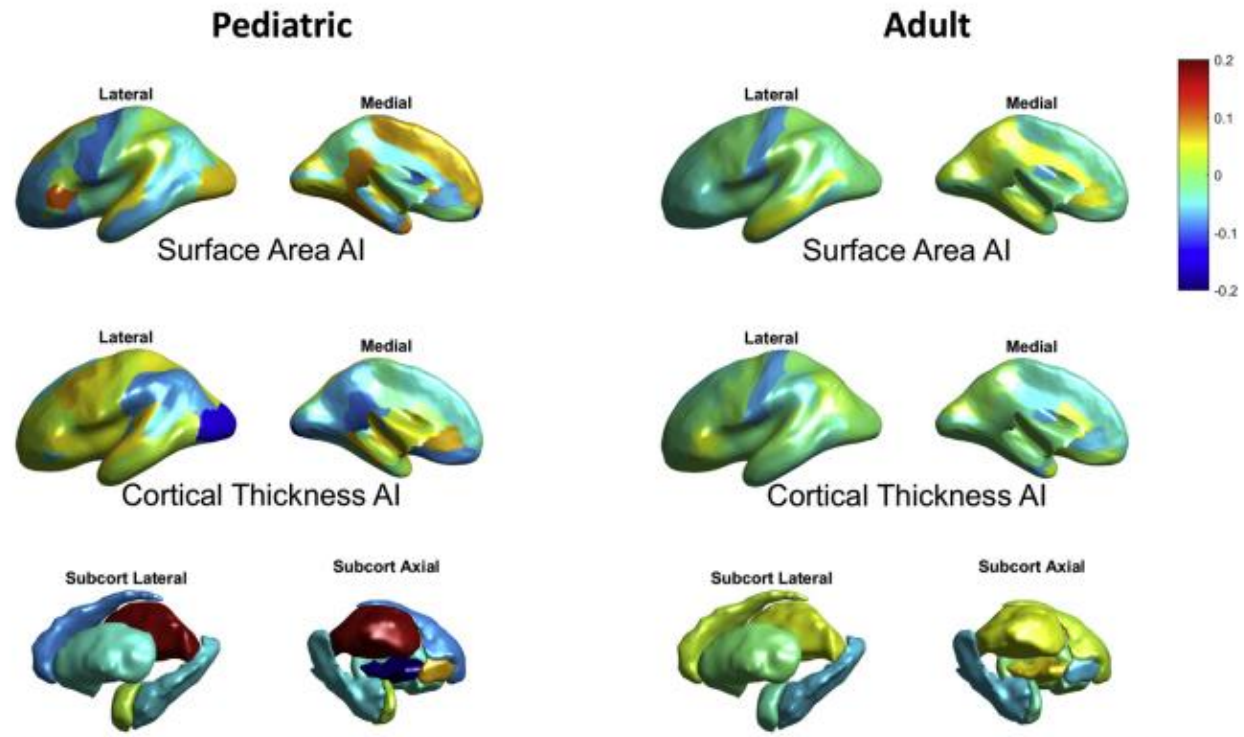


Figure 1. Effect size (Cohen's *d*) distributions for diagnosis on regional asymmetry indices (AIs) in the pediatric (left panel) and adult (right panel) data. Subcort, subcortical.

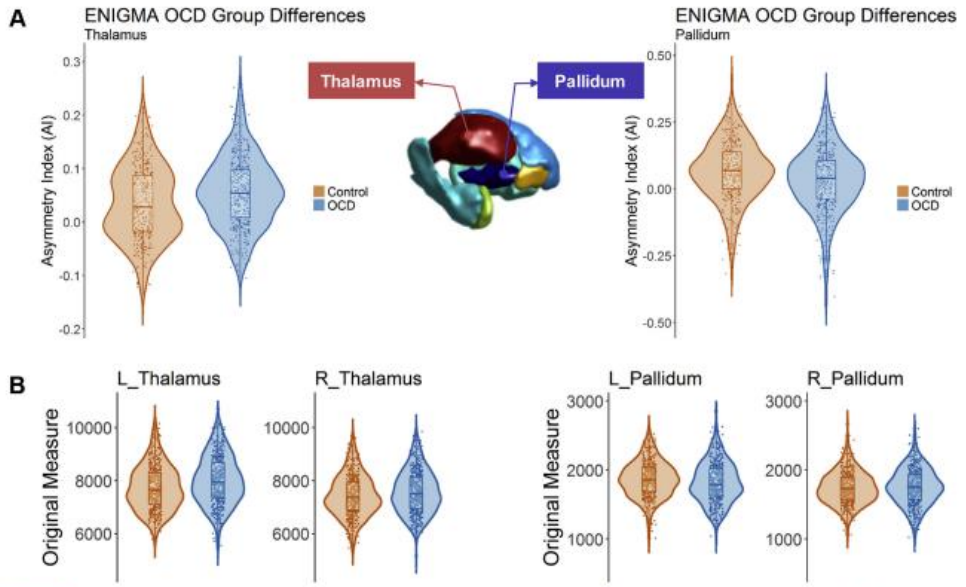


Figure 2. Subcortical structures showing altered volumetric asymmetry in pediatric patients with obsessive-compulsive disorder (OCD): the thalamus and

Pediatrik OKB de Talamusta solda artmış palidum da azalmış asimetri.
Nörogelişimsel?

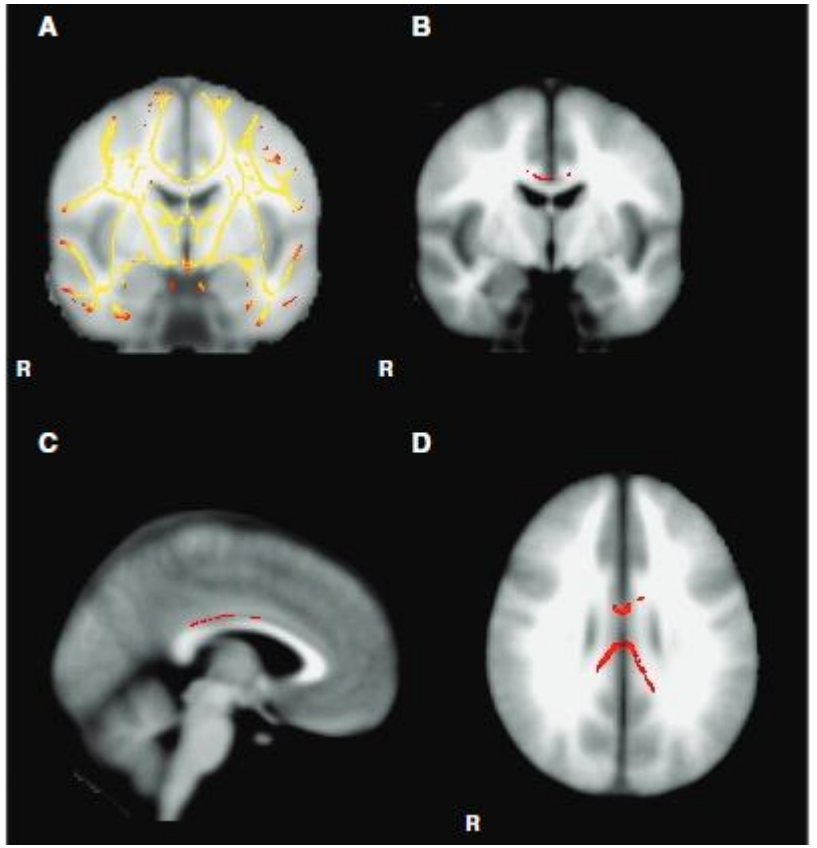
Research Paper

White matter microstructure in patients with obsessive–compulsive disorder

Emre Bora, MD; Ben J. Harrison, PhD; Alex Fornito, PhD; Luca Cocchi, PhD; Jesus Pujol, MD; Leonardo F. Fontenelle, MD; Dennis Velakoulis, MD; Christos Pantelis, MD; Murat Yücel, PhD, MAPS

Bora, Harrison, Fornito, Cocchi, Velakoulis, Pantelis, Yücel — Melbourne Neuropsychiatry Centre, Department of Psychiatry,

J Psychiatry Neurosci 2011;36(1)



CC body
2 hemisferin Parietal assosiasyon alanlarını bağlayan

Piras et al. *Translational Psychiatry* (2021)11:173
https://doi.org/10.1038/s41398-021-01276-z

Translational Psychiatry

ARTICLE Open Access

White matter microstructure and its relation to clinical features of obsessive–compulsive disorder: findings from the ENIGMA OCD Working Group

Abstract

Microstructural alterations in cortico-subcortical connections are thought to be present in obsessive–compulsive disorder (OCD). However, prior studies have yielded inconsistent findings, perhaps because small sample sizes provided insufficient power to detect subtle abnormalities. Here we investigated microstructural white matter alterations and their relation to clinical features in the largest dataset of adult and pediatric OCD to date. We analyzed diffusion tensor imaging metrics from 700 adult patients and 645 adult controls, as well as 174 pediatric patients and 144 pediatric controls across 19 sites participating in the ENIGMA OCD Working Group, in a cross-sectional case-control magnetic resonance study. We extracted measures of fractional anisotropy (FA) as main outcome, and mean diffusivity, radial diffusivity, and axial diffusivity as secondary outcomes for 25 white matter regions. We meta-analyzed patient-control group differences (Cohen's *d*) across sites, after adjusting for age and sex, and investigated associations with clinical characteristics. Adult OCD patients showed significant FA reduction in the sagittal stratum ($d = -0.21$, $z = -3.21$, $p = 0.001$) and posterior thalamic radiation ($d = -0.26$, $z = -4.57$, $p < 0.0001$). In the sagittal stratum, lower FA was associated with a younger age of onset ($z = 2.71$, $p = 0.006$), longer duration of illness ($z = -2.086$, $p = 0.036$), and a higher percentage of medicated patients in the cohorts studied ($z = -1.98$, $p = 0.047$). No significant association with symptom severity was found. Pediatric OCD patients did not show any detectable microstructural abnormalities compared to controls. Our findings of microstructural alterations in projection and association fibers to posterior brain regions in OCD are consistent with models emphasizing deficits in connectivity as an important feature of this disorder.

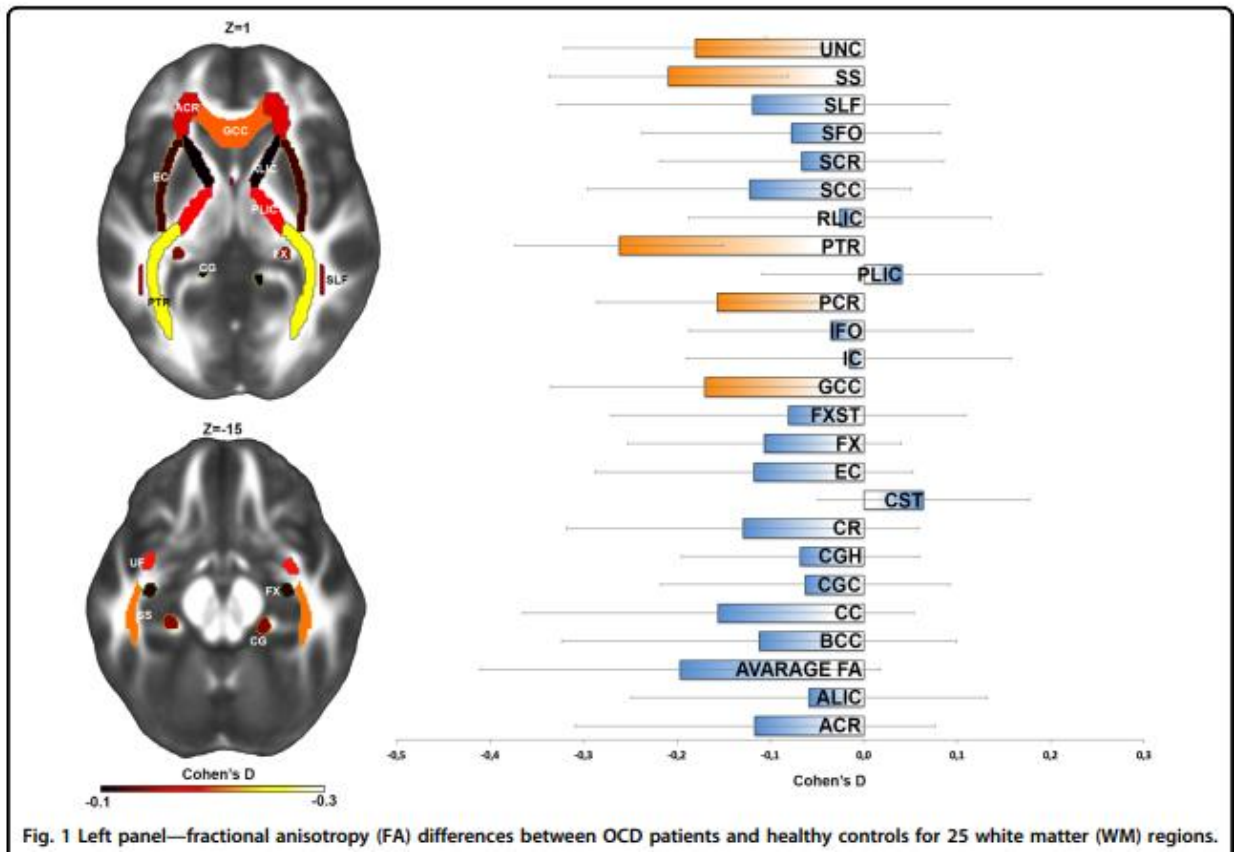


Fig. 1 Left panel—fractional anisotropy (FA) differences between OCD patients and healthy controls for 25 white matter (WM) regions.

Brain functional connectivity during induced sadness in patients with obsessive–compulsive disorder

Leonardo F. Fontenelle, MD, PhD; Ben J. Harrison, PhD; Jesus Pujol, MD;
Christopher G. Davey, MD, PhD; Alex Fornito, PhD; Emre Bora, MD; Christos Pantelis, MD;
Murat Yücel, PhD, MAPS

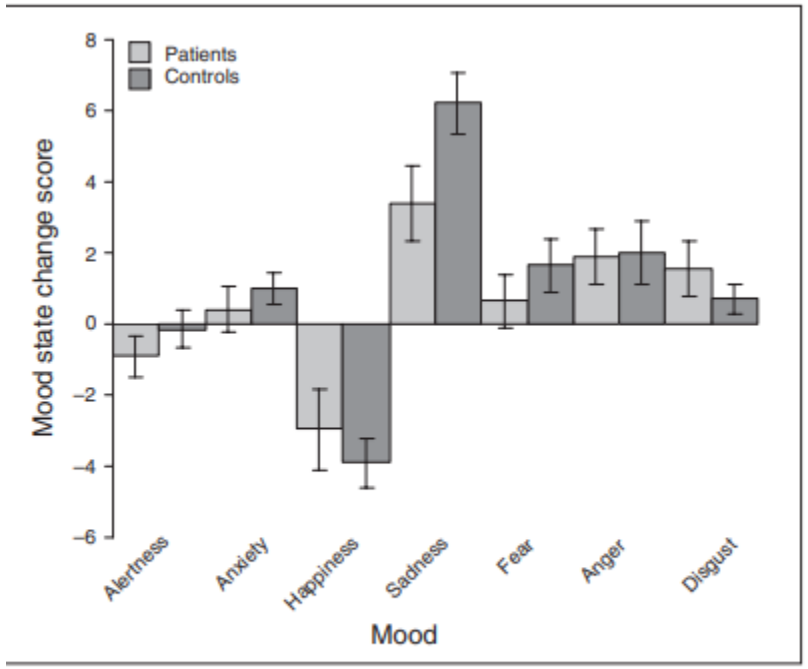


Fig. 1: Direction and magnitude of self-report change scores between the neutral and sad mood recall conditions for each group. Results are given as means and standard errors of the mean.

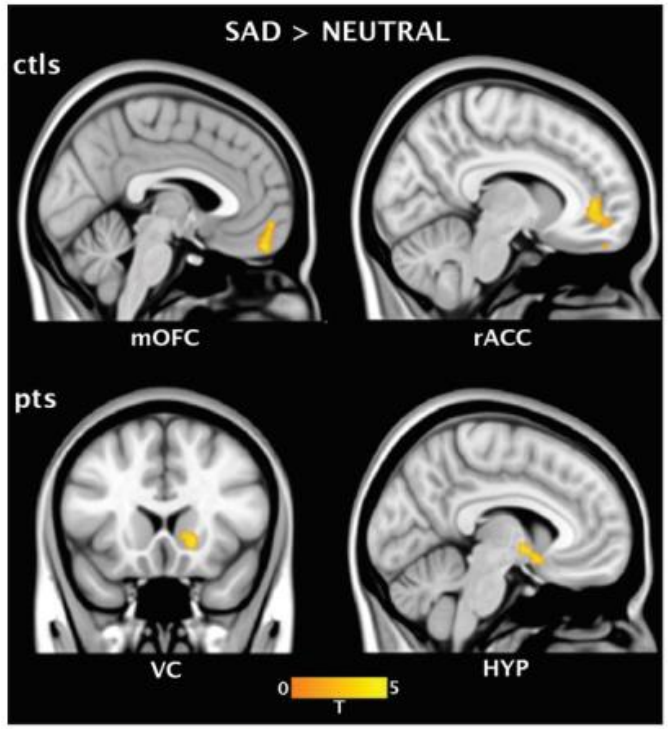


Fig. 3: Areas of increased functional connectivity with the subgenual anterior cingulate cortex during the sad compared with neutral mood induction conditions in controls and patients with obsessive–compulsive disorder. HYP = hypothalamus; mOFC = medial orbitofrontal cortex; rACC = rostral anterior cingulate cortex; VC = ventral caudate.

Molecular Psychiatry

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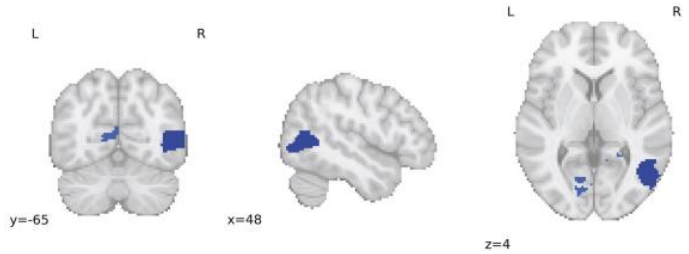
ARTICLE OPEN

Check for updates

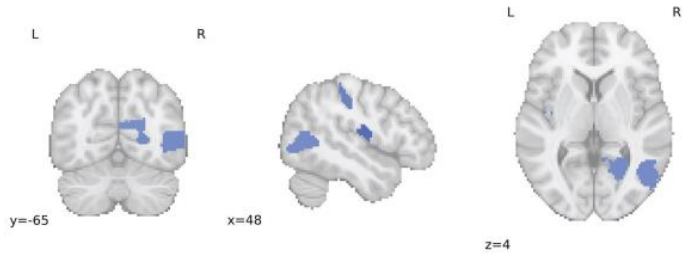
The functional connectome in obsessive-compulsive disorder: resting-state mega-analysis and machine learning classification for the ENIGMA-OCD consortium

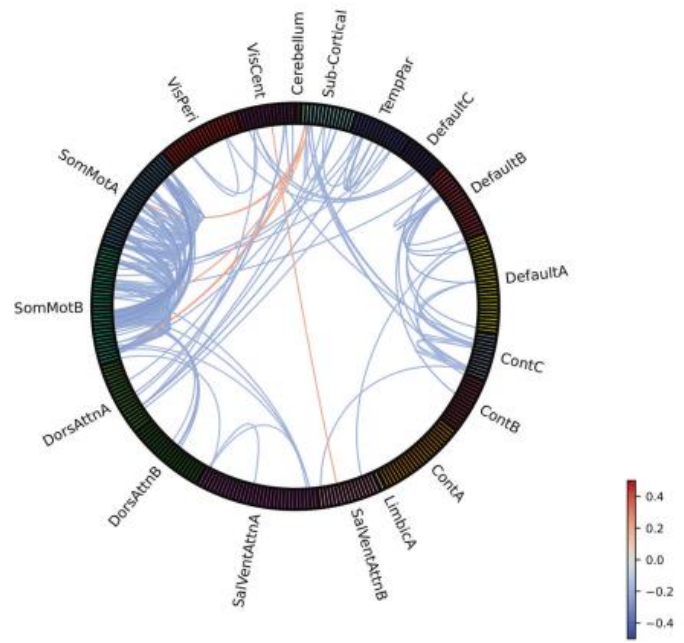
Willem B. Bruin[1,2✉], Yoshinari Abe[3], Pino Alonso[4,5,6,7], Alan Anticevic[8], Lea L. Backhausen[9], Srinivas Balachander[10], Nuria Bargallo[7,11,12], Marcelo C. Batistuzzo[13,14], Francesco Benedetti[15,16], Sara Bertolin Triquell[17], Silvia Brem[18,19], Federico Calesella[15,16], Beatriz Couto[20,21,22], Damiaan A. J. P. Denys[1,2], Marco A. N. Echevarria[13], Goi Khia Eng[23,24], Sónia Ferreira[20,21,22], Jamie D. Feusner[25,26,27], Rachael G. Grazioplene[8], Patricia Gruner[8], Joyce Y. Guo[28], Kristen Hagen[29,30,31], Bjarne Hansen[30,32], Yoshiyuki Hirano[33], Marcelo Q. Hoexter[13], Neda Jahanshad[34], Fern Jaspers-Fayer[35], Selina Kasprzak[36,37], Minah Kim[38,39], Kathrin Koch[40], Yoo Bin Kwak[41], Jun Soo Kwon[38,39,41], Luisa Lazaro[7,12,42,43], Chiang-Shan R. Li[8], Christine Lochner[44], Rachel Marsh[45], Ignacio Martínez-Zalacaín[17,46], Jose M. Menchon[7,17,46], Pedro S. Moreira[20,21,47], Pedro Morgado[20,21,22], Akiko Nakagawa[33], Tomohiro Nakao[48], Janardhanan C. Narayanaswamy[49,50], Erika L. Nurmi[51], Jose C. Pariente Zorrilla[12], John Piacentini[52], Maria Picó-Pérez[20,21,53], Fabrizio Piras[54], Federica Piras[54], Christopher Pittenger[8], Janardhan Y. C. Reddy[10], Daniela Rodriguez-Manrique[55,56,57], Yuki Sakai[3,58], Eiji Shimizu[33,59,60], Venkataram Shivakumar[61], Blair H. Simpson[19,45], Carles Soriano-Mas[7,17,62], Nuno Sousa[21,22], Gianfranco Spalletta[54,63], Emily R. Stern[23,24], S. Evelyn Stewart[35,64,65], Philip R. Szeszko[66,67], Jinsong Tang[68], Sophia I. Thomopoulos[34], Anders L. Thorsen[30,32], Tokiko Yoshida[33], Hirofumi Tomiyama[48], Benedetta Vai[16], Ilya M. Veer[69], Ganesan Venkatasubramanian[10], Nora C. Vetter[9,70], Chris Vriend[36,37,71,72], Susanne Walitza[18], Lea Waller[73], Zhen Wang[74], Anri Watanabe[3], Nicole Wolff[9], Je-Yeon Yun[39,75], Qing Zhao[74], Wieke A. van Leeuwen[1,2], Hein J. F. van Marle[36,76], Laurens A. van de Mortel[1,2], Anouk van der Straten[1,2], Ysbrand D. van der Werf[37,71,72], ENIGMA-OCD Working Group*, Paul M. Thompson[34], Dan J. Stein[77], Odile A. van den Heuvel[36,37,71] and Guido A. van Wingen[1,2✉]

status at the individual level using machine learning analysis. The mega-analyses revealed widespread abnormalities in functional connectivity in OCD, with global hypo-connectivity (Cohen's *d*: -0.27 to -0.13) and few hyper-connections, mainly with the thalamus (Cohen's *d*: 0.19 to 0.22). Most hypo-connections were located within the sensorimotor network and no fronto-striatal abnormalities were found. Overall, classification performances were poor, with area-under-the-receiver-operating-characteristic curve (AUC) scores ranging between 0.567 and 0.673, with better classification for medicated (AUC = 0.702) than unmedicated (AUC = 0.608) patients versus healthy controls. These findings provide partial support for existing pathophysiological models of OCD and highlight the important role of the sensorimotor network in OCD. However, resting-state connectivity does not so far provide an accurate biomarker for identifying patients at the individual level.



B





Neuroscience and Biobehavioral Reviews 131 (2021) 964–987

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

A systematic review and meta-analysis of altered electrophysiological markers of performance monitoring in Obsessive-Compulsive Disorder (OCD), Gilles de la Tourette Syndrome (GTS), Attention-Deficit/ Hyperactivity disorder (ADHD) and Autism

Alessio Bellato [a,b], Luke Norman [c], Iman Idrees [b], Carolina Y. Ogawa [d], Alice Waitt [b], Pedro F. Zuccolo [d], Charlotte Tye [a], Joaquim Radua [a,e,f], Madeleine J. Groom [b], Elizabeth Shephard [a,d,*]

97 çalışma
ERN (Error related negativity)
-OKB (g=0.54) ve GTS (g=0.99) artmış
-OSB (g=-0.61) ve DEHB (g=-0.47) azalmış

OSB Akrabalarında
Nöropsikolojik ve Beyin Görüntüleme Bulguları

Psychiatry and Clinical Neurosciences 2013; **67**: 20–27 doi:10.1111/pcn.12005

Regular Article

Broader autistic phenotype in parents of children with autism: Autism Spectrum Quotient–Turkish version

Sezen Kose, MD,[1*] Emre Bora, MD,[2] Serpil Erermiş, MD,[1] Burcu Özbaran, MD,[1] Tezan Bildik, MD[1] and Cahide Aydın, MD[1]

Table 2. AQ-TR mean scores

	AD parents ($n = 100$)		TDC parents ($n = 100$)		Group effect		Gender effect		Group × Gender interaction	
AQ scores	Fathers (n:47) mean ± SD	Mothers (n:53) mean ± SD	Fathers (n:48) mean ± SD	Mothers (n:52) mean ± SD	F	$P^{\dagger}$	F	$P^{\dagger}$	F	$P^{\dagger}$
Total score	19.08 ± 4.4	18.85 ± 6.5	17.5 ± 4.8	17.15 ± 5.7	4.49	**0.035**	0.12	0.72	0.01	0.90
Social skills	4.04 ± 1.8	4.00 ± 1.9	3.23 ± 1.9	3.43 ± 1.9	6.80	**0.010**	0.15	0.70	0.16	0.69
Attention switching	4.02 ± 1.8	3.81 ± 1.8	3.44 ± 1.7	4.1 ± 2.0	0.42	0.52	0.88	0.35	2.51	0.12
Attention to detail	4.89 ± 2.2	4.67 ± 2.2	4.98 ± 2.0	4.39 ± 1.7	0.42	0.84	1.63	0.20	0.25	0.62
Communication	2.36 ± 1.5	2.71 ± 1.9	2.08 ± 1.3	1.69 ± 1.3	7.83	**0.006**	0.06	0.81	2.36	0.13
Imagination	3.76 ± 1.6	3.65 ± 2.1	3.81 ± 1.9	3.51 ± 2.0	0.11	0.75	0.53	0.47	0.25	0.62

Published in final edited form as:
J Child Fam Stud. 2018 June ; 27(6): 1705–1720. doi:10.1007/s10826-018-1026-3.

Broader autism phenotype in parents of children with autism: a systematic review of percentage estimates

Eric Rubenstein[1,2] and **Devika Chawla**[1]

Published in final edited form as:
Biol Psychiatry. 2021 March 01; 89(5): 476–485. doi:10.1016/j.biopsych.2020.08.029.

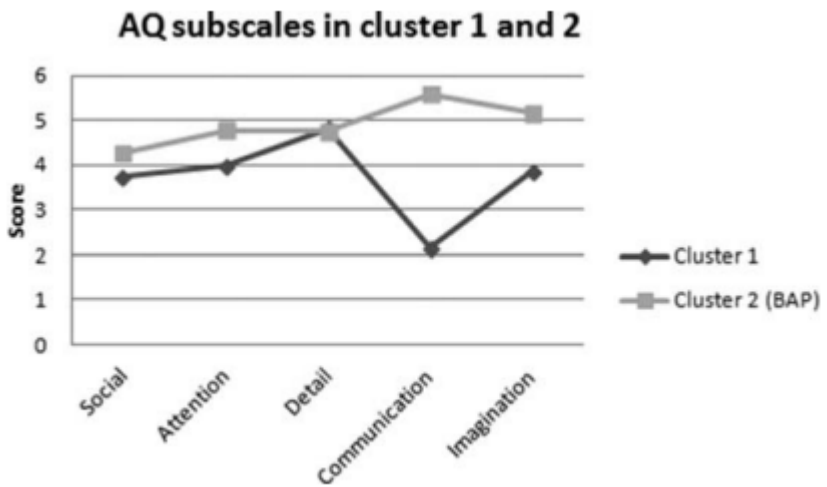
Elevated Polygenic Burden for Autism Spectrum Disorder Is Associated With the Broad Autism Phenotype in Mothers of Individuals With Autism Spectrum Disorder

Kritika Nayar[#], **Julia M. Sealock**[#], **Nell Maltman**, **Lauren Bush**, **Edwin H. Cook**, **Lea K. Davis**[#], **Molly Losh**[#]

Heterogeneity of Subclinical Autistic Traits Among Parents of Children With Autism Spectrum Disorder: Identifying the Broader Autism Phenotype with a Data-Driven Method

Emre Bora, Aydan Aydın, Tuğba Saraç, Muhammed Tayyib Kadak, and Sezen Köse

Clinical diagnosis of autism spectrum disorder (ASD) can be conceptualized as the extreme end of the distribution of subclinical autistic traits related to genetic susceptibility factors (broad autism phenotype (BAP)) in the general population. Subclinical autistic traits are significantly more common among unaffected first-degree relatives of probands with autism. However, there is a significant heterogeneity of autistic traits in family members of individuals with ASD and severity of autistic traits are not significantly different from controls in the majority of these relatives. The current study investigated the heterogeneity of autistic traits using latent class analysis (LCA) of the Autism Spectrum Quotient (AQ) ratings of 673 parents of children with ASD and 147 parents of typically developing children. Two distinct subgroups, including a "low-scoring" and a "high-scorer (BAP)" groups, were found. In comparison to control parents, a significantly larger proportion (21.1% vs. 7.5%) of parents of ASD were members of BAP group. Communication subscale made a distinctive contribution to the separation of high and low-scoring groups ($d = 2.77$). Further studies investigating neurobiological and genetic biomarkers and stability of these two subgroups over time are important for understanding the nature of autistic traits in the general population. ***Autism Res*** *2016, 00: 000–000.*



Psychiatric Disorder and the Broad Autism Phenotype: Evidence From a Family Study of Multiple-Incidence Autism Families

Joseph Piven, M.D., and Pat Palmer, Ph.D.

Objective: Several studies have shown familial aggregation of some axis I psychiatric disorders in families ascertained through a single autistic proband. In this study the authors examined the rate of axis I psychiatric disorders in nonautistic relatives from multiple-incidence autism families and the possible relationship of these disorders to the broad autism phenotype. **Method:** The rates of axis I psychiatric disorders, assessed by using semistructured and family history interviews, were compared in parents, grandparents, and aunts and uncles ascertained through 25 families of multiple-incidence autism probands and 30 families of probands with Down's syndrome. The possible association between selected psychiatric disorders and the broad autism phenotype, assessed directly through semistructured interviews and observational rating measures, was also examined in the two groups of parents. **Results:** The parents of the autistic probands had significantly higher rates of major depressive disorder and social phobia than the parents of the Down's syndrome probands. The high rate of depression in the parents of the autistic probands was consistent with the high rates of depression and anxiety detected in the grandparents and aunts and uncles in the autism families by family history. There was no evidence of an association, within individuals, between either depression or social phobia and the broad autism phenotype. **Conclusions:** Relatives of autistic individuals have high rates of major depression and social phobia that are not associated with the broad autism phenotype and cannot be explained by the increased stress associated with raising an autistic child. Alternative mechanisms and the scientific and clinical implications of these findings are discussed.

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Original Article

Annals of Neurosciences
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Broad Autism Phenotype and Gait in Parents of Children With and Without Autism Spectrum Disorder

Umer Jon Ganai[1], Braj Bhushan[1] and K. S. Venkatesh[2]

Abstract

Background: Autism spectrum disorder (ASD) is a highly heritable neurodevelopmental disorder. Research has shown that parents and relatives of children with ASD often exhibit subthreshold ASD-like characteristics known as broad autism phenotype (BAP) as well as impairments in motor behaviours.
Purpose: The current study aimed to examine the BAP traits and motor behaviours, that is, gait in 44 parents of children with ASD and in 48 parents of typically developing children (TD).
Methods: The BAP traits were measured using the broad autism phenotype questionnaire (BAPQ), and a low-cost computer vision-based framework was utilised to quantify the gait in children with ASD and their parents and TD children and their parents.
Results: The parents of children with ASD consistently displayed significantly higher scores on rigid personality and pragmatic language, however, there were no significant differences between the two group of parents on aloof personality of BAP traits. On gait parameters, the parents of children with ASD had a reduced gait speed in comparison to parents of TD children. There were no meaningful similarities in gait parameters of children with ASD and their parents.
Conclusions: These findings support the presence of ASD-like traits in the parents of children with ASD and gait speed as a putative motor endophenotype of ASD.

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https://doi.org/10.1007/s00787-024-02575-8

REVIEW

Prevalence of mental disorders among family members of individuals on the autism spectrum: systematic review and meta-analysis

Daniel H. Lins-Silva[1] · Igor D. Bandeira[2] · Daniela Faria-Guimarães[1] · Ingrid Dorea-Bandeira[1] · Arthur Tolentino[3] · Ângela Miranda-Scippa[4,5] · Daniel F. Hermens[6] · Rita Lucena[4,5]

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Abstract

Parenting a child on the autism spectrum presents particular challenges that can lead to increased stress, anxiety, and depression among family members. Therefore, we aimed to investigate the prevalence of mental disorders in first-degree relatives of individuals on the autism spectrum. This article adheres to the Preferred Reporting Items for Systematic Review and Meta-Analyses Protocols (PRISMA-P) guidelines, including studies indexed in PubMed/Medline, Embase, PsycINFO, Biblioteca Virtual em Saúde (BVS), and SciELO. Nineteen articles met eligibility criteria for the systematic review. Using a random-effects model (N = 93,876), we found a pooled prevalence of affective disorders of 13% in mothers of people on the autism spectrum (95% CI 7–21%; $I^2 = 99\%$, $p < 0.01$). Additionally, another random-effects model pointed out that first-degree relatives of people on the autism spectrum (N = 93,263) were more likely to present affective disorders than relatives of people with neurotypical development (N = 152,455) (pooled OR: 2.17; 95% CI 1.81–2.61). Careful assessment for mental disorders in parents and siblings of individuals on the autism spectrum is crucial to ensure appropriate treatment for these family members. This approach can also contribute to optimizing care for the individuals on the autism spectrum.

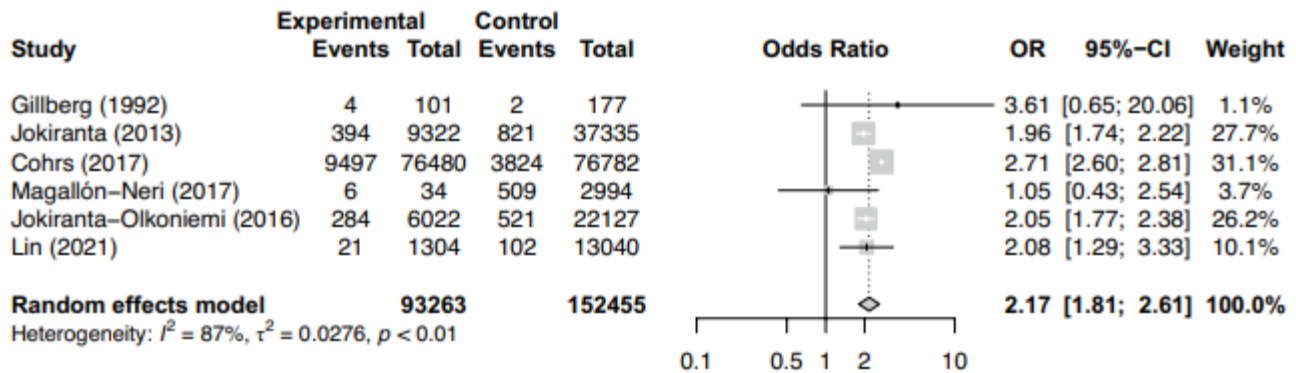

Fig. 2 Pooled prevalence of affective disorders in first-degree relatives of people on the autism spectrum

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Theory of mind and verbal working memory deficits in parents of autistic children

Sezen Gokcen [a], Emre Bora [b,*], Serpil Erermis [a], Hande Kesikci [a], Cahide Aydin [a]

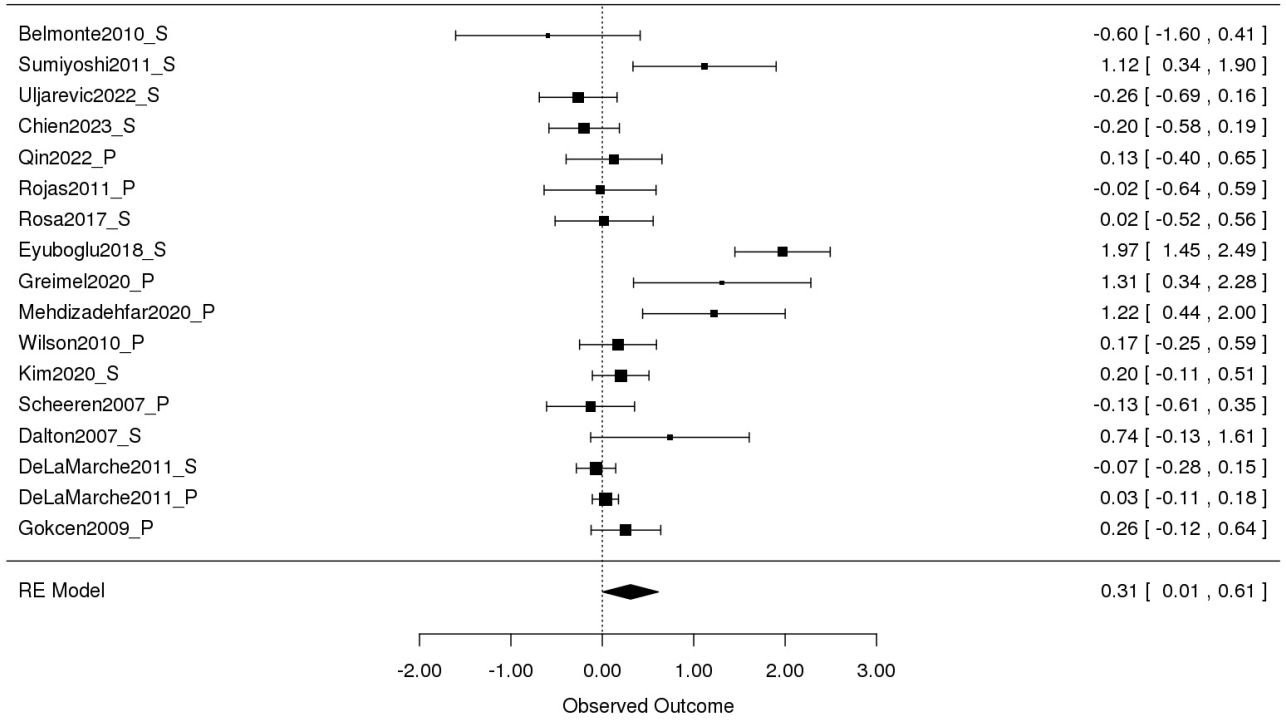
Gender–diagnosis interactions for the tasks in which a between-gender difference was observed

	Index		Controls		Gender		Gender–diagnosis interaction	
	Mother	Father	Mother	Father				
	Mean (S.D.)	Mean (S.D.)	Mean (S.D.)	Mean (S.D.)	*F*	*P*	*F*	*P*
Benton	43.2 (4.2)	41.3 (4.0)	43.5 (4.6)	40.9 (4.7)	6.8	0.01	0.3	0.60
Faces	7.4 (1.4)	6.8 (1.4)	8.0 (1.1)	7.0 (1.8)	8.6	0.004	0.3	0.60
Eyes	16.5 (3.2)	16.3 (3.4)	18.7 (3.3)	16.7 (3.4)	2.9	0.09	2.6	0.11

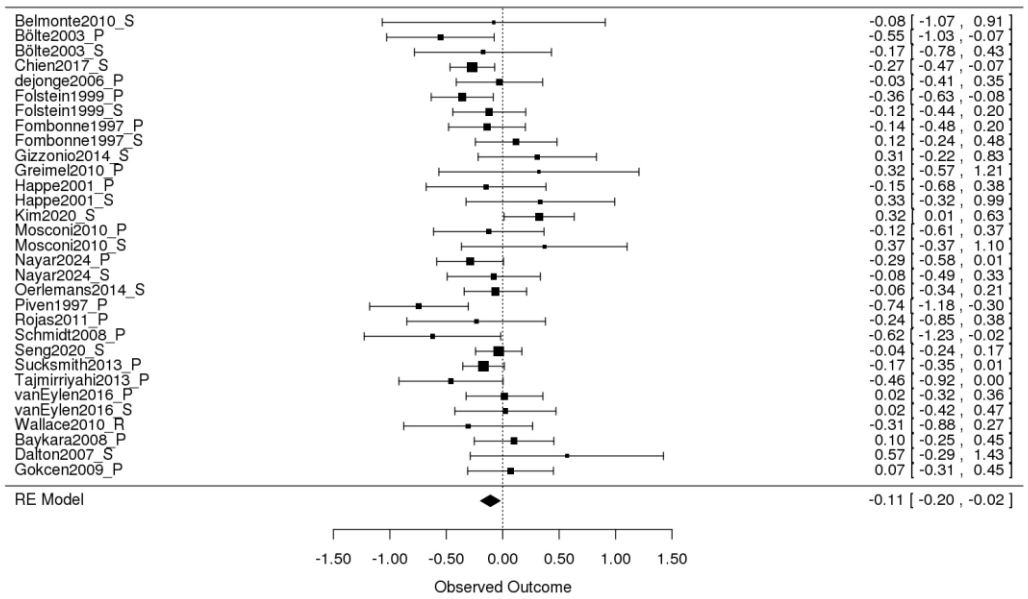
Between-group comparisons for demographic, clinical and EF variables

	Index parents (n=76)		Control parents (n=41)		Diagnosis		
	Mean	S.D.	Mean	S.D.	*F*	*P*	ES
Gender m:f	38:38		20:21				
Age	36.9	6.5	36.6	6.4	0.06	0.81	
Education (years)	12.3	3.5	12.5	3.9	0.06	0.81	
BPRS anxiety	2.6	2.2	2.0	1.8	1.5	0.14	
BPRS depression	1.1	1.4	0.5	1.3	2.7	0.007	
Cognitive tasks							
IQ	97.8	9.1	98.8	15.7	0.12	0.73	0.09
Benton face recognition	42.2	4.2	42.3	4.7	0.01	0.97	0.02
Executive dysfunction							
ACT total	34.0	5.5	36.9	4.4	8.41	0.004*	0.55
Stroop interference	42.0	17.2	38.7	13.9	1.07	0.30	0.20
Fluency	33.8	12.1	37.2	14.7	1.63	0.21	0.26
Social cognition							
Decoding							
Faces	16.1	1.9	16.5	2.2	1.57	0.20	0.23
Eyes	16.4	3.2	17.8	3.5	4.2	0.04	0.43
Reasoning							
UOT	14.7	3.4	16.7	3.2	8.61	0.004*	0.58
Hinting	7.4	1.1	7.6	1.0	1.93[a]	0.06	0.36

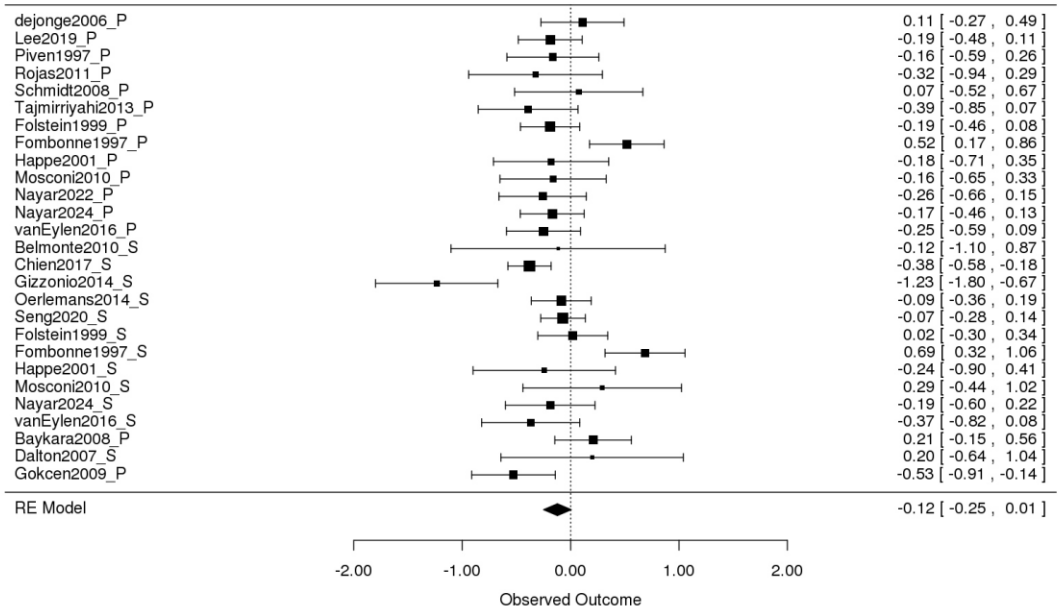
OSB akrabalarında otistik özellikler



OSB Akrabaları- IQ

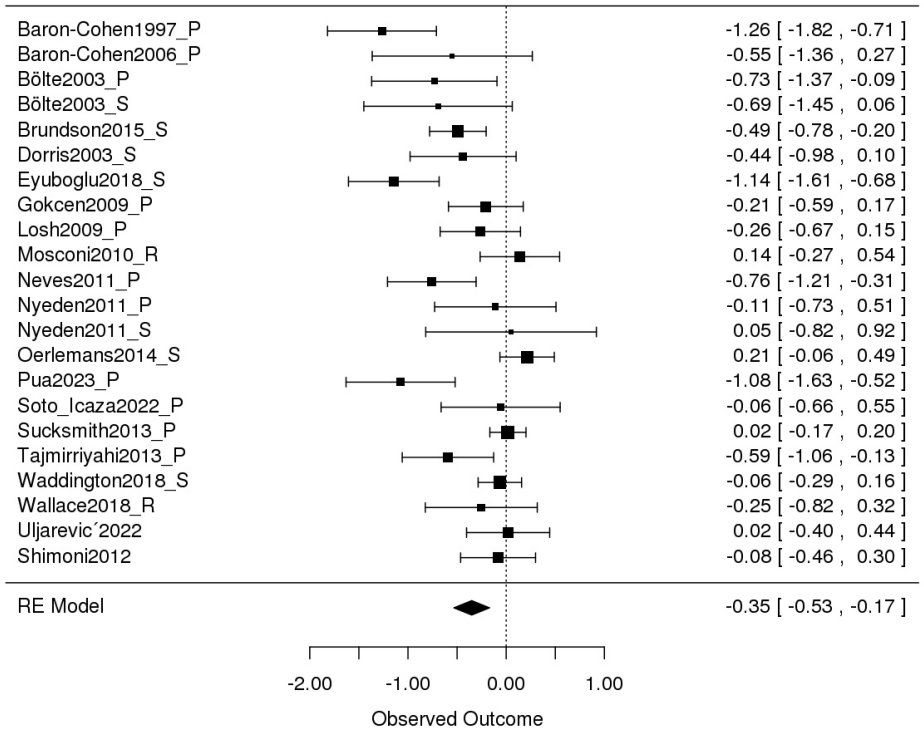


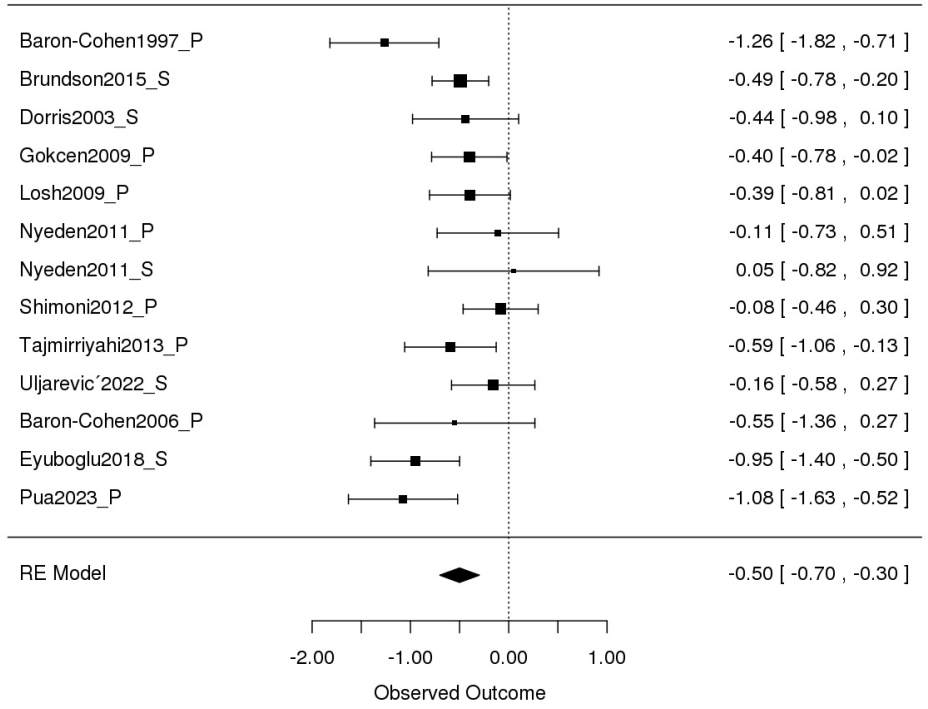
Performans IQ



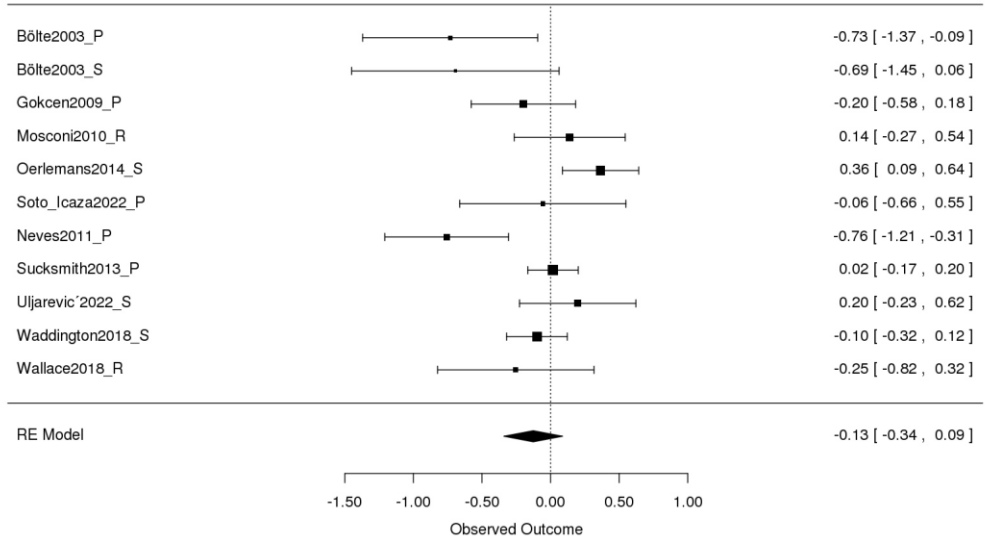
Sözel IQ

OSB Akrabalarında sosyal biliş



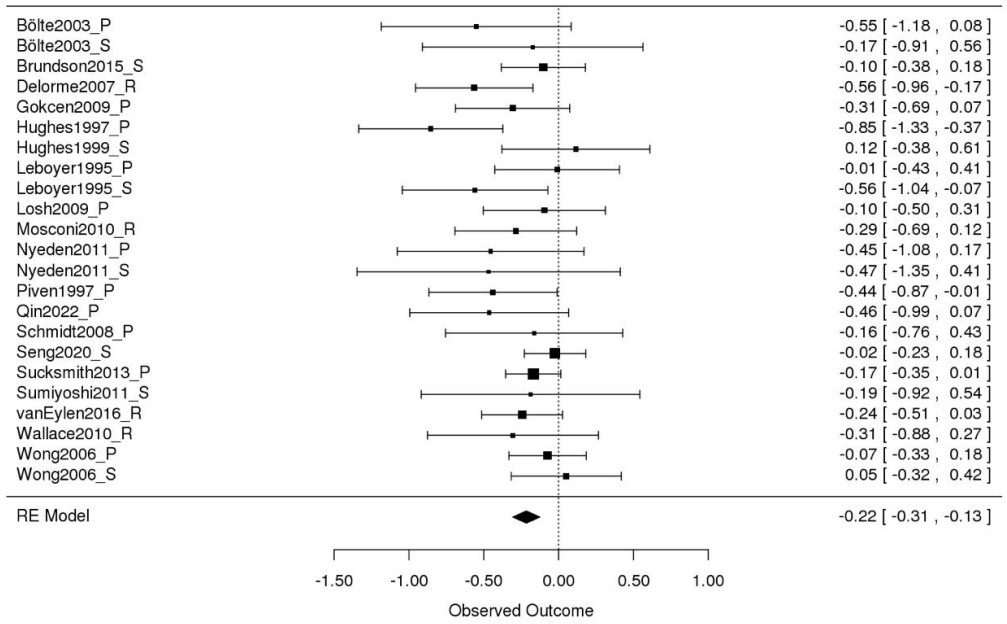


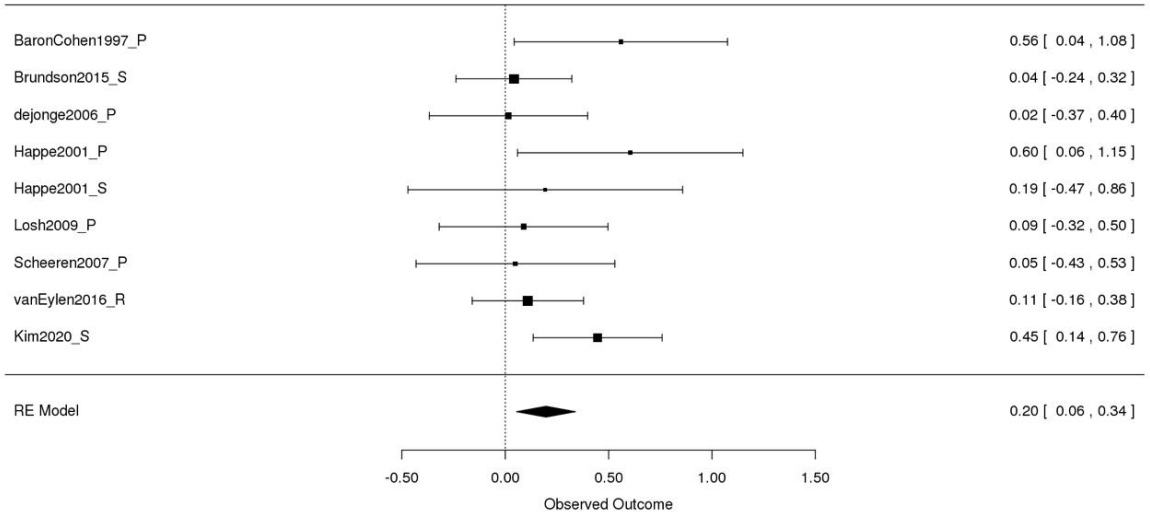
Zihin kuramı



Emosyon tanıma

OSB akrabaları yürütücü işlevler ve merkezi koherans





Psychological Medicine

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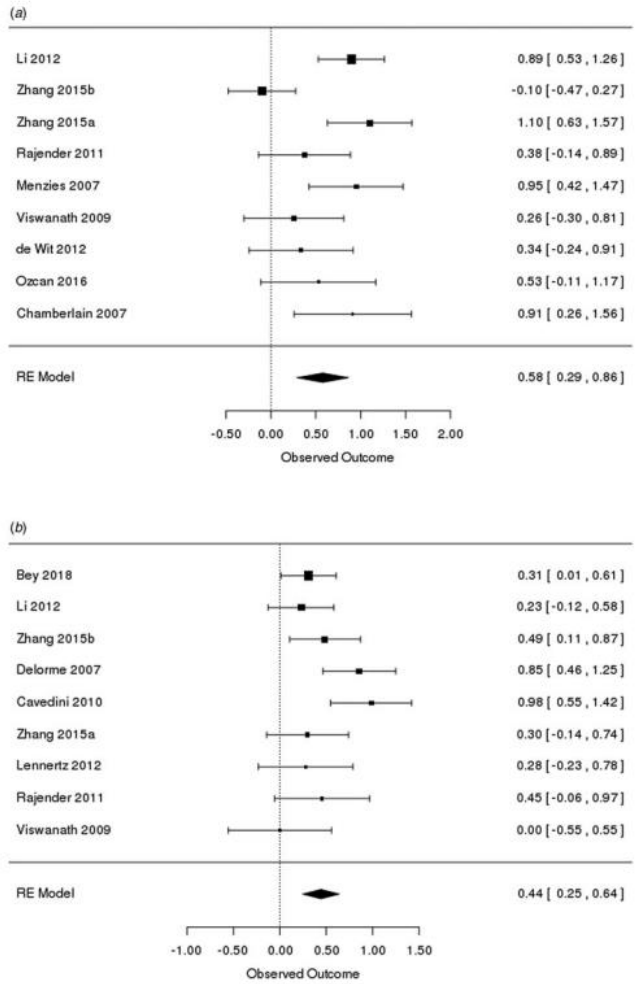
Meta-analysis of neurocognitive deficits in unaffected relatives of obsessive-compulsive disorder (OCD): comparison with healthy controls and patients with OCD

Emre Bora[1,2,3]

Review Article

Cite this article: Bora E (2020). Meta-analysis



g. 1. Forest plot of the meta-analysis of group differences in inhibition and planning/problem solving between OCDrel and healthy controls; (*a*) inhibition; (*b*) anning/problem solving

Table 2. Mean weighted effect sizes for differences between unaffected first-degree relatives of individuals with OCD and healthy controls

Test	Study (k)	OCDrel	HC	*d*	95% CI	*Z*	*p*	*Q*	*Q* (p)	τ^2	Bias (p)	I^2 (%)
Executive functions												
Planning	9	403	417	**0.44**	0.25–0.64	4.4	<0.001	10.5	0.16	0.02	0.99	33
Set-shifting	8	271	309	**0.37**	0.04–0.69	2.1	0.03	27.5	0.003	0.16	0.20	73
WCST per errors[a]	7	251	289	0.26	−0.06 to 0.58	1.6	0.11	19.4	0.004	0.12		69
WCST categories[a]	4	131	173	0.25	−0.11 to 0.62	1.3	0.18	6.8	0.08	0.08		56
TMT B[a]	6	250	275	0.09	−0.19 to 0.37	0.6	0.54	12.6	0.03	0.07		60
Inhibition	9	284	325	**0.58**	0.29–0.86	3.9	<0.001	25.6	0.001	0.12	0.42	66
Stroop[b]	5	198	237	**0.46**	0.04–0.88	2.1	0.03	17.6	0.001	0.18		77
SSRT[b]	3	68	88	**0.73**	0.31–1.16	3.4	<0.001	3.1	0.21	0.05		36
Decision-making	5	148	158	**0.58**	0.19–0.98	3.8	<0.001	10.9	0.03	0.13	0.09	65
Working memory	5	168	173	0.19	−0.02 to 0.41	1.8	0.07	0.5	0.98	0	0.14	0
Visual memory	7	216	260	**0.28**	0.08–0.49	2.8	0.006	6.9	0.33	0	0.25	13
Verbal memory	7	216	258	0.20	−0.12 to 0.53	1.2	0.22	17.9	0.007	0.13	0.40	66
Processing Speed	9	291	333	0.09	−0.12 to 0.30	0.9	0.39	13.0	0.11	0.05	0.67	38
TMT A[c]	5	186	228	0.02	−0.26 to 0.30	0.1	0.88	7.6	0.11	0.05		47
Fluency	6	240	265	0.09	−0.09 to 0.27	1.1	0.29	2.3	0.81	0	0.37	0
Letter fluency[d]	5	192	178	0.11	−0.10 to 0.31	1.0	0.30	3.2	0.52	0		0
Category fluency[d]	3	121	163	0.07	−0.17 to 0.30	0.6	0.58	0.4	0.82	0		0

Table 4. Mean weighted effect sizes for differences between OCDpt and unaffected first-degree relatives of individuals with OCD

Test	Study (k)	OCD	OCrel	*d*	95% CI	*Z*	*p*	*Q*	*Q* (p)	τ^2	Bias (p)	I^2 (%)
Executive functions												
Planning	6	302	284	0.06	−0.13 to 0.26	0.6	0.52	6.7	0.25	0.01	0.86	25
Set-shifting	6	223	216	**0.23**	0.04–0.42	2.4	0.02	4.0	0.54	0	0.70	0
Inhibition	7	260	229	0.10	−0.09 to 0.29	1.0	0.32	6.7	0.35	0.01	0.38	11
Decision-making	4	133	123	0.13	−0.12 to 0.38	1.0	0.30	0.1	0.99	0	0.77	0
Working memory	5	199	160	0.29	−0.06 to 0.64	1.6	0.10	9.9	0.04	0.09	0.03	60
Visual memory	5	168	161	**0.45**	0.22–0.67	3.9	<0.001	1.4	0.85	0	0.38	0
Verbal memory	5	168	161	0.36	−0.05 to 0.76	1.7	0.08	13.0	0.01	0.15	0.37	69
Processing speed	8	274	267	0.18	−0.05 to 0.40	1.5	0.12	11.8	0.11	0.04	0.13	41
Fluency	4	158	151	0.14	−0.07 to 0.37	1.3	0.22	2.0	0.57	0	0.63	0

OCDpt, OCD patients, OCDrel, unaffected relatives of individuals with OCD, *d*, Cohen's *d*. Bold values: Significant underperformance of OCDpt compared to OCDrel.

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Articles

Social brain dysfunctionality in individuals with autism spectrum disorder and their first-degree relatives: An activation likelihood estimation meta-analysis

Ziwen Peng[a,b,1,*], Jierong Chen[c,1], Lili Jin[a], Hongying Han[d], Chenjie Dong[a], Ya Guo[a], Xuejun Kong[e], Guobin Wan[c], Zhen Wei[c,**]

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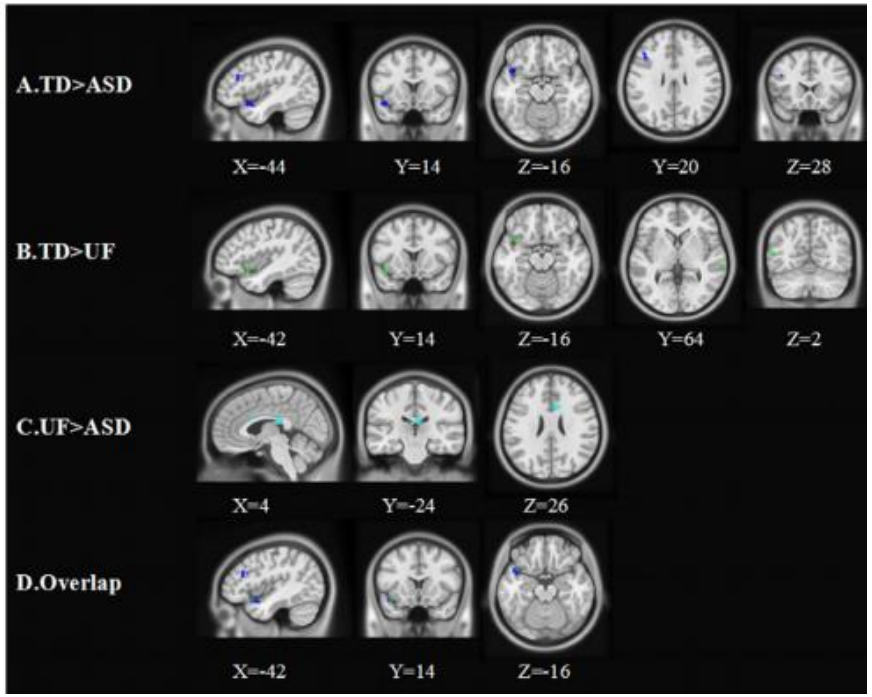
ARTICLE INFO

Keywords:
Autism spectrum disorder (ASD)
Unaffected family members
Functional magnetic resonance Imaging (fMRI)
Amygdala, Meta-analysis

ABSTRACT

The social brain hypothesis is regarded as a powerful theory to understand social cognition. Individuals with autism spectrum disorder (ASD) have specific deficits in social and communicative behavior, but the exact relationship between these deficits and abnormalities in the social brain remains unclear. The high heritability of this disorder makes it important to focus on the first-degree relatives of those affected. Research focusing on genetically at-risk (yet healthy) relatives of patients with ASD is critical to the study of neuroimaging endophenotypes. We conducted a voxel-wise activation likelihood estimation (ALE) meta-analysis of 9 functional neuroimaging studies published during the period from 2006 to 2018. These studies included 200 individuals with ASD, 216 unaffected family members (UF), and 235 typical development controls (TD). The voxel-wise significance threshold was $p < 0.01$ (uncorrected $p = 0.001$).The ALE meta-analyses showed hyperactivation in the inferior frontal gyrus (IFG) and superior temporal gyrus (STG) among individuals with ASD and UF, compared with TD individuals. Group comparisons showed greater likelihood of hyperactivation in the amygdala for ASD, compared with UF and TD.



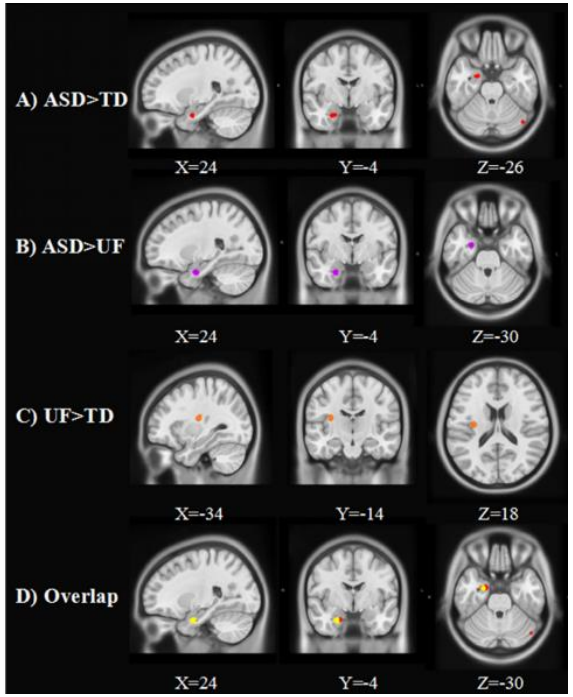


Table 2

Meta-analysis results of comparisons: TD > ASD and TD > UF.

Brain regions	BA	Volume (mm^3)	MNI x	y	z	Extrema Value
TD > ASD						
Inferior Frontal Gyrus	47	528	−44	14	−16	0.015
Superior Temporal Gyrus	38		−44	6	−20	0.010
Middle Frontal Gyrus	9	104	−42	20	28	0.010
TD > UF						
Inferior Frontal Gyrus	47	152	−42	14	−16	0.011
Middle Temporal Gyrus	37	120	−54	−64	10	0.010
Superior Temporal Gyrus	22	80	66	−28	2	0.010

Meta-analysis results of comparisons: UF > ASD and UF > TD.

Brain regions	BA	Volume (mm^3)	MNI x	y	z	Extrema Value
UF > ASD						
Superior Temporal Gyrus	39	584	52	−52	12	0.009
Fusiform		528	−34	−68	−10	0.008
Cingulate Gyrus	24	504	8	18	26	0.008
Posterior Cingulate	23	488	4	−24	22	0.008
UF > TD						
Insula	13	496	−34	−14	18	0.015

Meta-analysis results of comparisons: ASD > TD and ASD > UF.

Brain regions	BA	Volume (mm^3)	MNI x	y	z	Extrema Value
ASD > TD						
Amygdala		504	24	−4	−30	0.009
Cerebellum		272	−46	−72	−24	0.008
ASD > UF						
Amygdala		328	24.6	−3.8	−29.2	0.008

Journal of Affective Disorders 384 (2025) 180–207

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Research paper

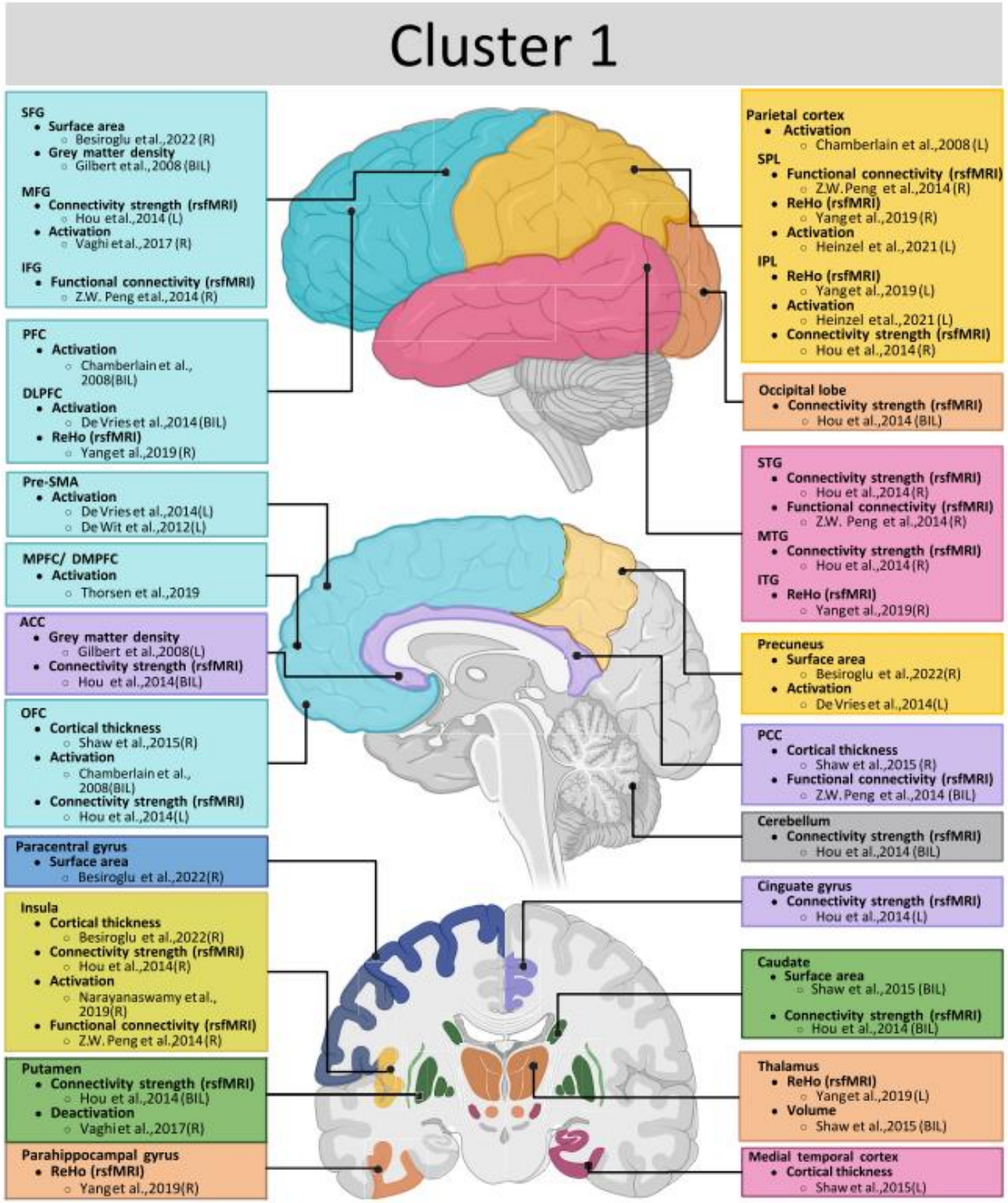
Neuroimaging alterations in relatives of patients with obsessive-compulsive disorder: A review of magnetic resonance imaging studies☆

Payam Jannatdoust [a], Parya Valizadeh [a], Sara Bagherieh [b], Giulia Cattarinussi [c,d,e], Fabio Sambataro [c,d], Luisa Cirella [f], Giuseppe Delvecchio [f,*]

OKB akraba çalışmaları en sık örüntü, hastalara benzer bulgular (Küme 1) insula, talamus, dorsolateral ve ventromedial prefrontal korteks, parietal korteks sık

Bulgular çok heterojen

Küme 2: Akrabalarda daha belirgin bulgu frontoparietal, koruyucu, küme 3: hasta akraba zıt yönde (kompensaturar), küme 4 sadece hastalarda anormallik



OSB ve Eşlik Eden Psikiyatrik Tanılar

Neuroscience and Biobehavioral Reviews 155 (2023) 105436

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Neuroscience and Biobehavioral Reviews

journal homepage: www.elsevier.com/locate/neubiorev

Prevalence of co-occurring conditions in children and adults with autism spectrum disorder: A systematic review and meta-analysis

Martina Micai [a], Laura Maria Fatta [a], Letizia Gila [a], Angela Caruso [a], Tommaso Salvitti [a], Francesca Fulceri [a], Antonio Ciaramella [a], Roberto D'Amico [b,c], Cinzia Del Giovane [b,d], Marco Bertelli [e], Giovanna Romano [f], Holger Jens Schünemann [g,h], Maria Luisa Scattoni [a,*]

Co-occurring condition prevalence type	*Prevalence in autistic population*	*I^2% (Q Test p-value)*
Mental health and psychiatric		
ADHD N = 72 n = 208,789	37% (28–46%)	99.93 (<0.0001)
Affective Disorder N = 22 n = 16,195	19% (11–28%)	99.37 (<0.0001)
Anxiety Disorder N = 60 n = 45,978	35% (30–39%)	98.54 (<0.0001)
Bipolar Disorder N = 25 n = 108,224	7% (4–9%)	98.85 (<0.0001)
Depressive Disorder N = 55 n = 41,923	18% (15–21%)	97.60 (<0.0001)
Developmental Coordination Disorder N = 2 n = 11,857	87% (87–88%)	0
Disruptive Behavior N = 24 n = 18,842	28% (21–36%)	98.94 (<0.0001)
Disruptive Impulse Control Disorder N = 42 n = 163,225	17% (13–22%)	99.73 (<0.0001)
Feeding and Eating Disorder N = 32 n = 19,233 Anorexia nervosa, bulimia nervosa, binge eating disorder N = 14 n = 12,721	32% (20–46%) 5% (2–10%)	99.72 (<0.0001) 98.55 (<0.0001)
Gender Identity Disorder N = 1 n = 34	3% (1–15%)	NA
Intellectual Disability N = 27 n = 162,997	33% (26–41%)	99.87 (<0.0001)
Language Disorder N = 4 n = 8979	16% (0–53%)	99.93 (<0.0001)
Obsessive Compulsive Disorder N = 44 n = 36,467	9% (7–10%)	
Personality Disorder (any kind) N = 14 n = 8306	7% (4–10%)	
Schizophrenia N = 29 n = 65,841	10% (7–13%)	
Sleep-Wake Disorder N = 29 n = 203,287	25% (18–34%)	
Specific Learning Disorder N = 1 n = 122	13% (8–20%)	
Somatic Symptom and related disorders N = 4 n = 186	28% (5–58%)	
Substance Use Disorder N = 18 n = 20,311	5% (2–8%)	
Tic Disorder N = 38 n = 36,749	10% (8–13%)	
Trauma Stress Related Disorder N = 12 n = 21,162	4% (0–10%)	

Medical and neurological	
Celiac Disease N = 6 n = 8419	4% (1–9%)
Chromosome abnormality N = 9 n = 56,118	2% (1–4%)
Genetic Disorder N = 12 n = 19,692	3% (1–6%)
Down Syndrome N = 16 n = 74,916	1% (1–1%)
Elimination Disorder N = 27 n = 2246)	29% (19–40%)
Epilepsy N = 84 n = 250,800	16% (14–18%)
Food Intolerance N = 7 n = 7094	13% (3–29%)
Gastrointestinal Disorder N = 18 n = 27,249	21% (11–33%)
Gastrointestinal Problem N = 43 n = 23,908	39% (32–46%)
Gluten Intolerance N = 2 n = 338	1% (0–3%)
Hearing Disorder N = 14 n = 22,213	4% (2–7%)
Metabolic Disorder N = 5 n = 5014	3% (1–5%)
Motor Problem N = 12 n = 25,945	40% (19–64%)
Neurocutaneous Disorder N = 12 n = 21,682	0% (0–1%)
Organic Nutrition Disorder N = 3 n = 6038	22% (0–76%)
Overweight/Obesity N = 26 n = 70598	33% (25–41%)
Rett Syndrome N = 1 n = 92	2% (1–8%)
Fragile X Syndrome N = 20 n = 20,802	2% (1–3%)

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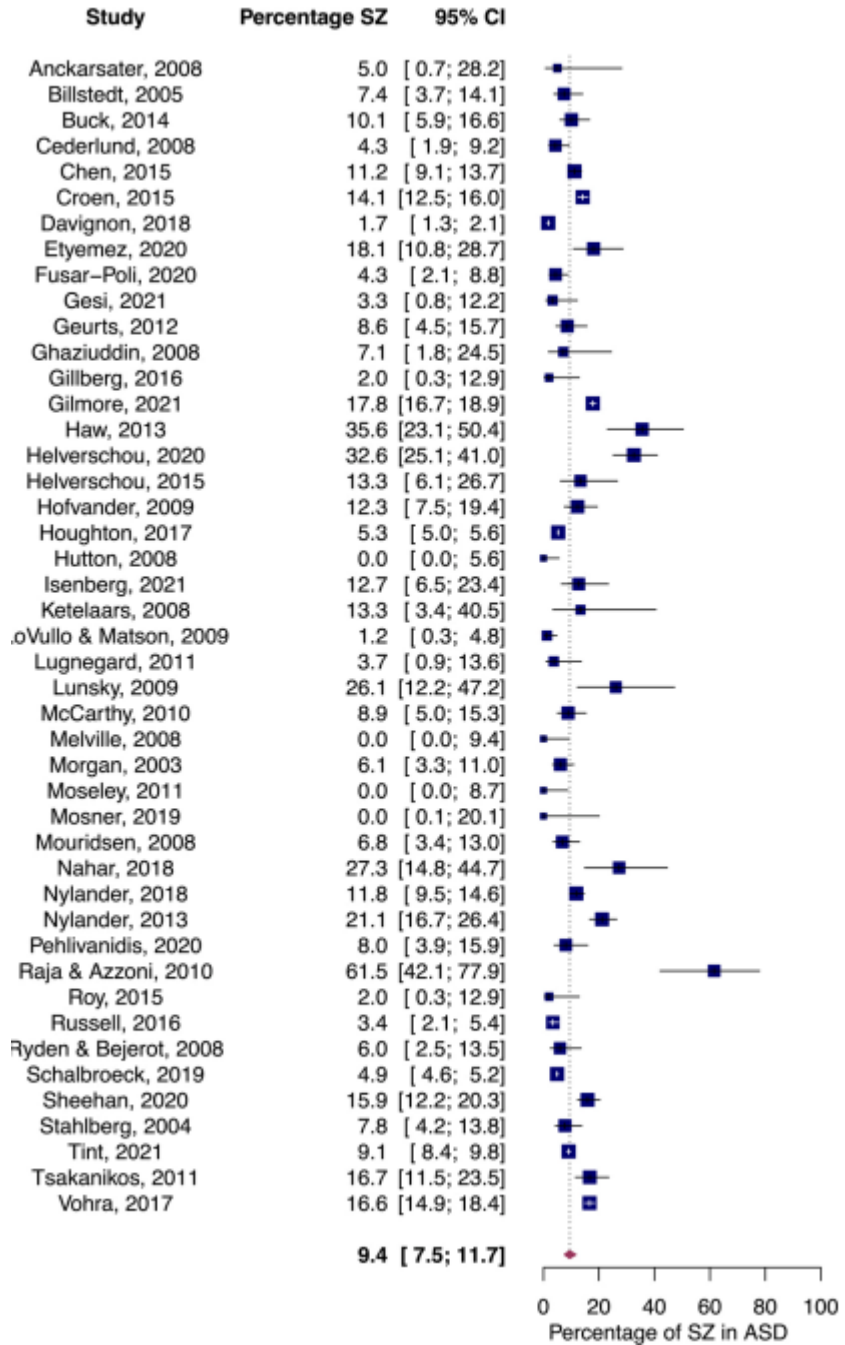
Occurrence of psychosis and bipolar disorder in adults with autism: A systematic review and meta-analysis

Kandice J. Varcin [a,b], Sarah E. Herniman [c,d,e], Ashleigh Lin [b], Yanyu Chen [b], Yael Perry [b], Charlotte Pugh [b], Katharine Chisholm [f,g], Andrew J.O. Whitehouse [b], Stephen J. Wood [c,d,g,*]

OSB tanılı erkeklerde psikoz kadınlarda bipolar bozukluk görece çok

OSB ve psikotik bipolar bozukluğa yanlış şizofreni tanısı riski artmış (Vannucchi ve ark 2014)


Study
Percentage SZ
95% CI
Anckarsater, 2008 5.0 [0.7; 28.2]
Billstedt, 2005 7.4 [3.7; 14.1]
Buck, 2014 10.1 [5.9; 16.6]
Cederlund, 2008 4.3 [1.9; 9.2]
Chen, 2015 11.2 [9.1; 13.7]
Croen, 2015 14.1 [12.5; 16.0]
Davignon, 2018 1.7 [1.3; 2.1]
Etyemez, 2020 18.1 [10.8; 28.7]
Fusar−Poli, 2020 4.3 [2.1; 8.8]
Gesi, 2021 3.3 [0.8; 12.2]
Geurts, 2012 8.6 [4.5; 15.7]
Ghaziuddin, 2008 7.1 [1.8; 24.5]
Gillberg, 2016 2.0 [0.3; 12.9]
Gilmore, 2021 17.8 [16.7; 18.9]
Haw, 2013 35.6 [23.1; 50.4]
Helverschou, 2020 32.6 [25.1; 41.0]
Helverschou, 2015 13.3 [6.1; 26.7]
Hofvander, 2009 12.3 [7.5; 19.4]
Houghton, 2017 5.3 [5.0; 5.6]
Hutton, 2008 0.0 [0.0; 5.6]
Isenberg, 2021 12.7 [6.5; 23.4]
Ketelaars, 2008 13.3 [3.4; 40.5]
oVullo & Matson, 2009 1.2 [0.3; 4.8]
Lugnegard, 2011 3.7 [0.9; 13.6]
Lunsky, 2009 26.1 [12.2; 47.2]
McCarthy, 2010 8.9 [5.0; 15.3]
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Morgan, 2003 6.1 [3.3; 11.0]
Moseley, 2011 0.0 [0.0; 8.7]
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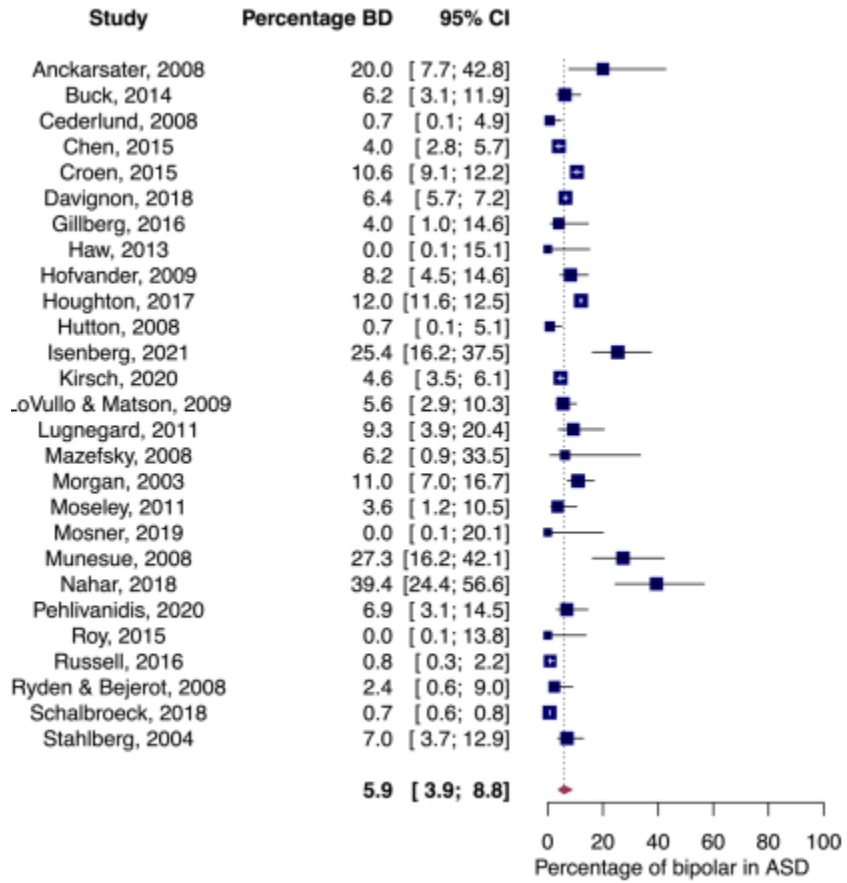
Mouridsen, 2008 6.8 [3.4; 13.0]
Nahar, 2018 27.3 [14.8; 44.7]
Nylander, 2018 11.8 [9.5; 14.6]
Nylander, 2013 21.1 [16.7; 26.4]
Pehlivanidis, 2020 8.0 [3.9; 15.9]
Raja & Azzoni, 2010 61.5 [42.1; 77.9]
Roy, 2015 2.0 [0.3; 12.9]
Russell, 2016 3.4 [2.1; 5.4]
Ryden & Bejerot, 2008 6.0 [2.5; 13.5]
Schalbroeck, 2019 4.9 [4.6; 5.2]
Sheehan, 2020 15.9 [12.2; 20.3]
Stahlberg, 2004 7.8 [4.2; 13.8]
Tint, 2021 9.1 [8.4; 9.8]
Tsakanikos, 2011 16.7 [11.5; 23.5]
Vohra, 2017 16.6 [14.9; 18.4]
9.4 [7.5; 11.7]
0 20 40 60 80 100
Percentage of SZ in ASD


Study
Percentage BD
95% CI
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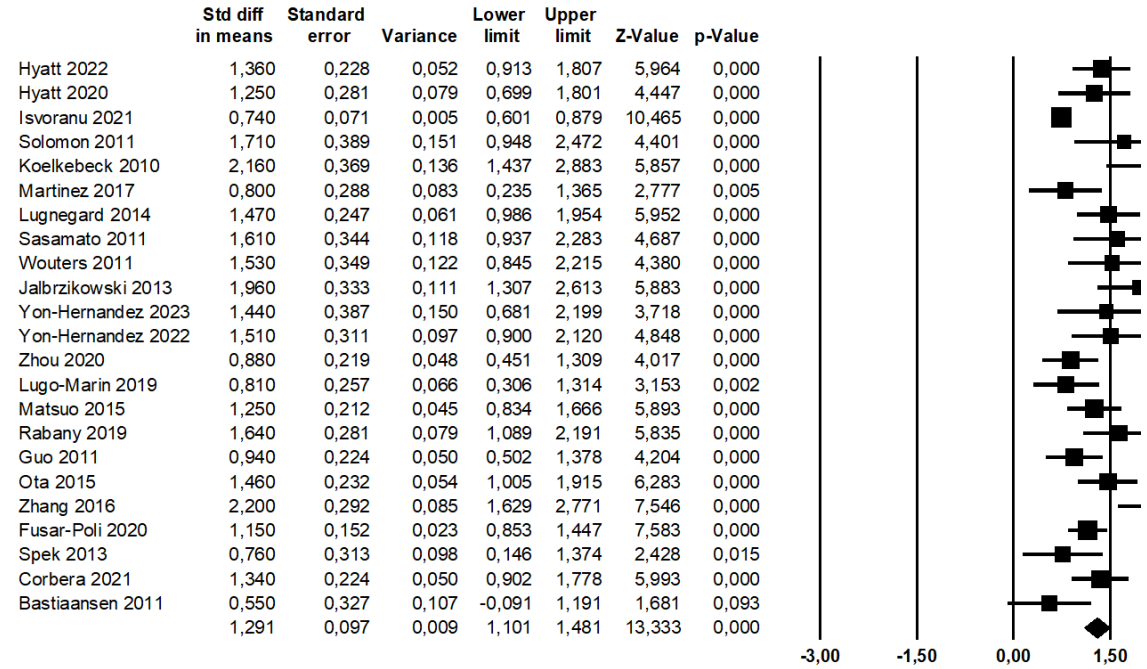
Buck, 2014 6.2 [3.1; 11.9]
Cederlund, 2008 0.7 [0.1; 4.9]
Chen, 2015 4.0 [2.8; 5.7]
Croen, 2015 10.6 [9.1; 12.2]
Davignon, 2018 6.4 [5.7; 7.2]
Gillberg, 2016 4.0 [1.0; 14.6]
Haw, 2013 0.0 [0.1; 15.1]
Hofvander, 2009 8.2 [4.5; 14.6]
Houghton, 2017 12.0 [11.6; 12.5]
Hutton, 2008 0.7 [0.1; 5.1]
Isenberg, 2021 25.4 [16.2; 37.5]
Kirsch, 2020 4.6 [3.5; 6.1]
oVullo & Matson, 2009 5.6 [2.9; 10.3]
Lugnegard, 2011 9.3 [3.9; 20.4]
Mazefsky, 2008 6.2 [0.9; 33.5]
Morgan, 2003 11.0 [7.0; 16.7]
Moseley, 2011 3.6 [1.2; 10.5]
Mosner, 2019 0.0 [0.1; 20.1]
Munesue, 2008 27.3 [16.2; 42.1]
Nahar, 2018 39.4 [24.4; 56.6]
Pehlivanidis, 2020 6.9 [3.1; 14.5]
Roy, 2015 0.0 [0.1; 13.8]
Russell, 2016 0.8 [0.3; 2.2]
Ryden & Bejerot, 2008 2.4 [0.6; 9.0]
Schalbroeck, 2018 0.7 [0.6; 0.8]
Stahlberg, 2004 7.0 [3.7; 12.9]
5.9 [3.9; 8.8]
0 20 40 60 80 100
Percentage of bipolar in ASD

Şizofreni de otistik özellikler (Bora ve ark, hazırlanmakta)

Tablo-1 Şizofreni hastalarıyla sağlıklı kontrollerde otistik özellikleri (OÖ) karşılaştıran çalışmaların meta-analiz sonuçları

Değişken	k	Şz	SK	g	CI	Z	p	I^2
OÖ	24	1169	1606	1.29	1.11-1.48	14.2	<0.001	71.2
OÖ-Klinisyen	9	285	374	1.43	1.26-1.61	16.4	<0.001	0
OÖ-Özbildirim	16	900	1252	1.22	0.99-1.45	10.6	<0.001	73.5
OÖ-Sosyal iletişimsel	18	1025	1335	1.13	0.97-1.29	14.1	<0.001	54.8
OÖ-Tekrarlayıcı kısıtlayıcı	14	875	1165	0.90	0.69-1.11	8.3	<0.001	69.6



Şekil 3 Şizofreni-sağlıklı kontrol gruplarında otistik özellikleri karşılaştıran orman grafiği

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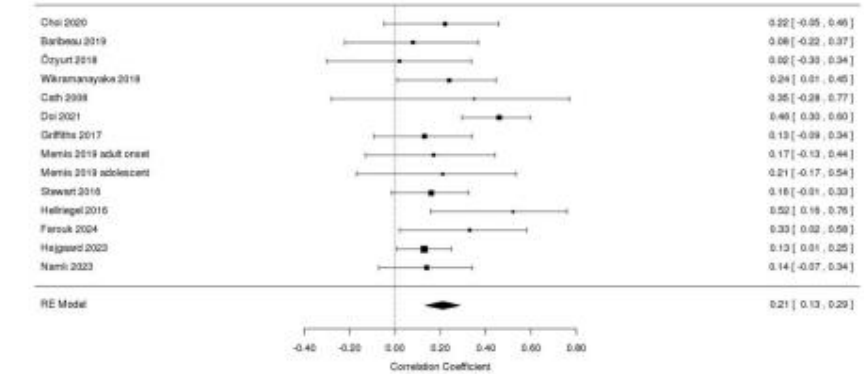
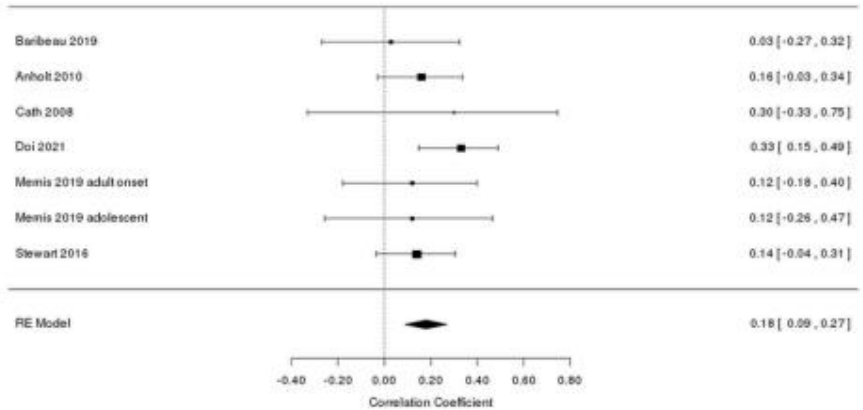
Autistic traits in obsessive compulsive disorder: A systematic review and meta-analysis

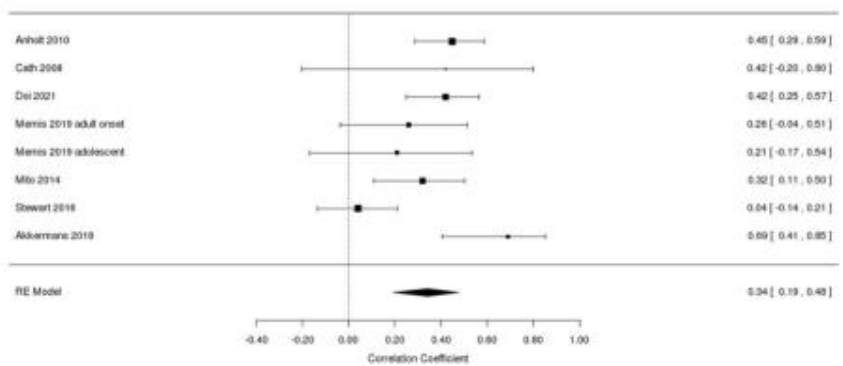
Sıla Derin [a], Melike Tetik [b], Emre Bora [a,b,c,*]

Mean weighted effect sizes for autistic traits between OCD and HC.

Test	*k*	OCD	HC	*g*	95 % *CI*	*Z*	*P*	*Q-test p*	I^2	Bias
Autistic traits total	14	783	1048	1.27	1.02–1.53	9.75	<0.0001	<0.0001	82.6	0.26
-Social-communication domain	10	553	726	0.98	0.66–1.31	5.97	<0.0001	<0.0001	85.21	0.78
-RRBs domain	13	563	820	1.65	1.27–2.04	8.45	<0.0001	<0.0001	88.33	0.21
AQ total score	9	474	704	1.41	1.1–1.72	8.89	<0.0001	<0.0001	80.1	0.43
AQ social skills	7	404	574	1.12	0.84–1.4	7.82	<0.0001	<0.001	72.4	0.63
AQ attention switching	7	404	574	1.47	0.87–2.05	4.86	<0.0001	<0.0001	93.1	0.61
AQ attention to detail	7	404	574	0.47	0.23–0.71	3.79	0.0001	0.002	66.87	0.55
AQ communication	7	404	574	1.03	0.7–1.35	6.19	<0.0001	<0.0001	79.8	0.67
AQ imagination	7	404	574	0.62	0.36–0.88	4.61	<0.0001	0.001	71.33	0.44
SCQ	5	292	328	0.82	0.66–0.99	9.75	<0.0001	0.72	0	0.40

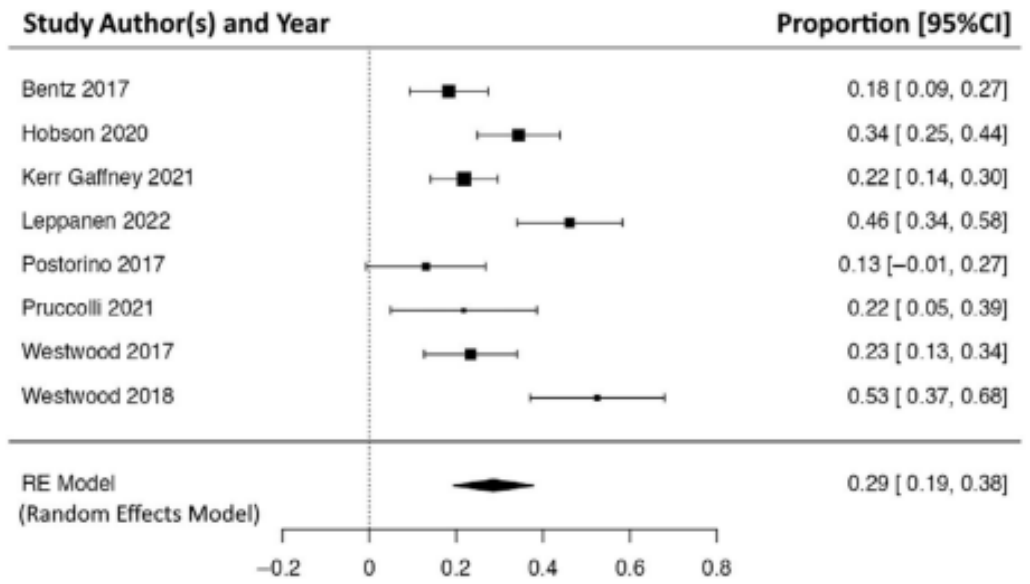






YBOCS skorları Otistik özellikler özelikle RRB ile ilişkili

OKB tanılı bireylerin bir alt grubu ek OSB tanısı?



Forest plot for ratio meta-analysis for the proportion of individuals with AN scoring above the ADOS for a possible ASD diagnosis.

International Journal of Eating Disorders

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Investigating the Presence of Autistic Traits and Prevalence of Autism Spectrum Disorder Symptoms in Anorexia Nervosa: A Systematic Review and Meta-Analysis

Ipek Inal-Kaleli[1] | Nurhak Dogan[2] | Sezen Kose[1] | Emre Bora[3,4,5]

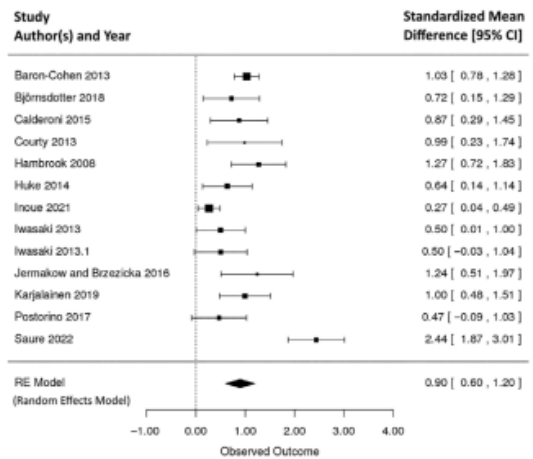
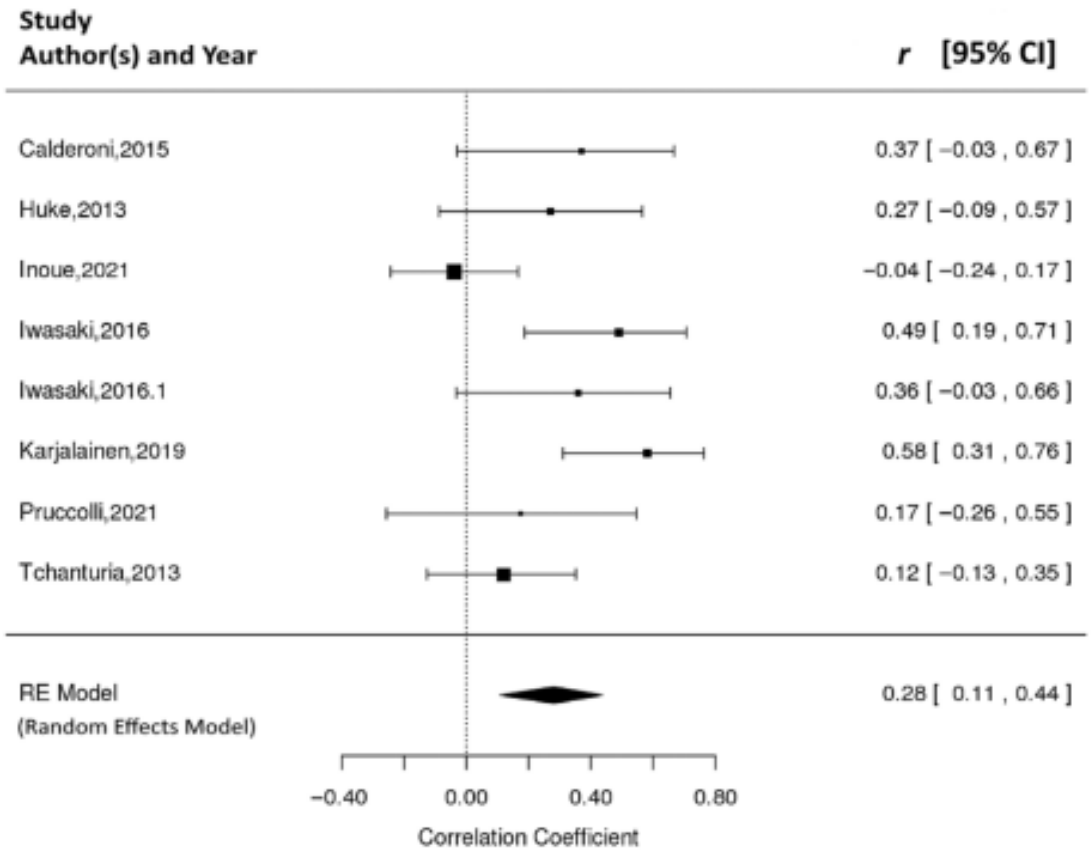


FIGURE 3 | Forest plot of standardized mean differences from studies comparing AQ total scores for AN and HC groups (positive effect indicating greater autistic traits in the AN group).

Test	*k*	AN	HC	*g*	95% CI	*Z*	*p*	*Q*-test *p*	I^2	Bias
AQ total/AQ 10 pooled	17	817	2706	0.88	0.65–1.12	7.32	<0.0001*	<0.0001	81.12	0.18
AQ total score	13	450	2437	0.90	0.60–1.20	5.85	<0.0001*	<0.0001*	81.70	0.17
AQ attention to detail	9	281	733	0.28	0.13–0.42	3.66	0.0003*	0.723	0	0.99
AQ attention switching	9	281	733	0.53	0.28–0.78	4.34	<0.0001	0.0406	51.54	0.27
AQ imagination	9	281	733	0.26	−0.05–0.57	1.66	0.967	<0.0001	71.1	0.01*
AQ communication	9	281	733	0.48	0.22–0.74	3.65	0.0003*	0.0247	58.72	0.36
AQ social skills	9	281	733	0.70	0.43–0.97	5.15	<0.0001*	0.0045*	59.65	0.03*

Yeme bozukluğunun şiddeti otistik özellikler anlamlı düzeyde ilişkili



Sonuç

- Genetik, CNV-OSB ilişkisi çoğu OKB de yok (olası küçük alt grup), poligenik kalıtımda ortaklıklar görece daha fazla
- Sosyal biliş, Yürütücü işlevler ve Merkezi koherans anormallikleri OSB hasta ve akrabalarda belirgin. OKB yürütücü işlevler için benzer kanıt, sosyal biliş etkilenme hafif, CC veri yetersiz, bu 2 boyutta akraba verisi yok
- Beyin görüntüleme, gri cevher OSB ayrıksı örüntü OKB ve diğer psikiyatrik hastalıklara göre, beyaz cevher mikroyapısı ve işlevsel bazı ortak noktalar, bazı farklı noktalar.
- Elektrofizyolojik verilerden Hata ile ilişkili potansiyeller zıt uçta bulgular
- GOF, OKB ye de renk vermekte
- Boyutsal daha belirgin ilişkiler (örneğin RRB nin nörobiyoloji ile)?
- OKB ve Bazı psikiyatrik bozuklukların GOF eşlik eden alt tipleri. Farklı özelliklerini inceleyen çalışmalar gerekli
- Alt tiplerin keşfine yönelik veriye dayalı çalışmaların önemi