Addiction and ADHD:

Aetiology, Prevention, and Treatment

Wim van den Brink, MD PhD

Em Prof of Psychiatry and Addiction

Amsterdam University Medical Centers, location Academic Medical Center

Amsterdam, The Netherlands

Jubiläumssymposium Prof Franz Moggi: Sucht, Comorbidität und Behandling

Bern, 28 April 2023

UNIVERSITÄRE PSYCHIATRISCHE DIENSTE BERN

Disclosures

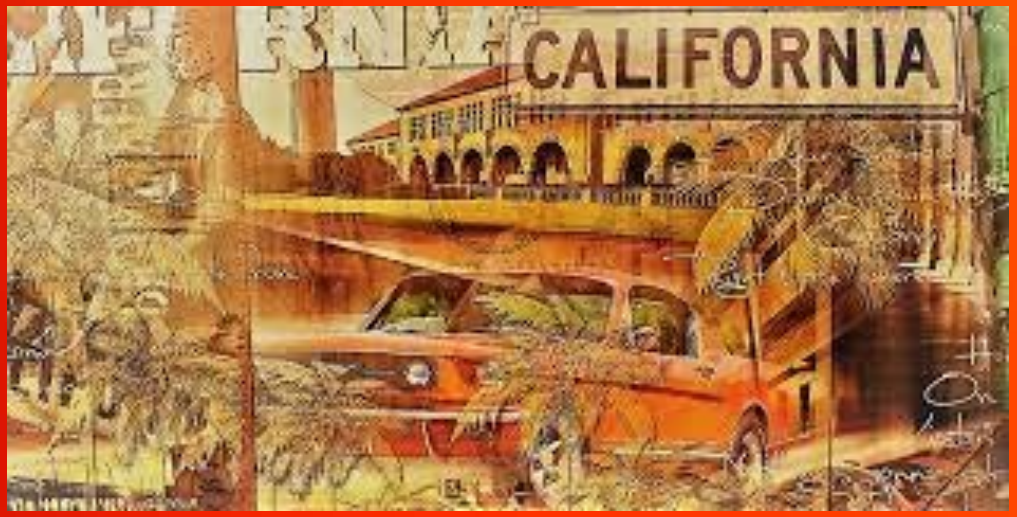
Interest	Name of the Organization
Subsidies	NeuroSearch, Alkermes
Honoraria	Lundbeck, Reckitt Benckiser/Indivior, Pfizer, Eli Lilly, Merck Serono, Recordati, Angelini, Takeda-Acino
Consultancies	Lundbeck, Reckitt Benckiser/Indivior, Novartis, Teva, Mundipharma, Bioproject, D&A Pharma, Kinnov Therapeutics, Opiant Pharmaceuticals, Takeda, Camurus, Clearmind Medicine, Novo Nordisk, Adial Pharmaceuticals

Franz Moggi

Congratulations!!!

- Great scientist
  (>200 papers, HI=28)
- Perfect host and organizer
- Great colleague
- Fellow traveller in ICASA
  (board member and 16 shared papers)

Program



Program

- **ADHD and Substance Use Disorders (SUD)**

  * How many SUD patients also have ADHD?

  * What are the responsible mechanisms for this comorbidity?

  * Is it possible to prevent development of SUD in ADHD patients?

  * Do we need special interventions to treat SUD+ADHD?

- **Conclusions**

Epidemiology of Addiction and ADHD

How many SUD patients also have ADHD?

Cross-national prevalence and correlates of adult attention-deficit hyperactivity disorder

J. FAYYAD, R. DE GRAAF, R. KESSLER, J. ALONSO, M. ANGERMEYER, K. DEMYTTENAERE, G. DE GIROLAMO, J. M. HARO, E. G. KARAM, C. LARA, J.-P. LÉPINE, J. ORMEL, J. POSADA-VILLA, A. M. ZASLAVSKY and R. JIN

BJP, 2007

The descriptive epidemiology of DSM-IV Adult ADHD in the World Health Organization World Mental Health Surveys

John Fayyad[1] · Nancy A. Sampson[2] · Irving Hwang[2] · Tomasz Adamowski[3] · Sergio Aguilar-Gaxiola[4] · Ali Al-Hamzawi[5] · Laura H. S. G. Andrade[6] · Guilherme Borges[7] · Giovanni de Girolamo[8] · Silvia Florescu[9] · Oye Gureje[10] · Josep Maria Haro[11] · Chiyi Hu[12,13] · Elie G. Karam[1,14,15] · Sing Lee[16] · Fernando Navarro-Mateu[17] · Siobhan O'Neill[18] · Beth-Ellen Pennell[19] · Marina Piazza[20,21] · José Posada-Villa[22] · Margreet ten Have[23] · Yolanda Torres[24] · Miguel Xavier[25] · Alan M. Zaslavsky[2] · Ronald C. Kessler[2] · on behalf of the WHO World Mental Health Survey Collaborators

Atten Defic Hyperact Disord. 2017 Mar;9(1):47-65.

Table 2 Multiply imputed prevalence estimates of adult attention-deficit hyperactivity disorder

Country	Prevalence, % (s.e.)	n
Belgium	4.1 (1.5)	486
Colombia	1.9[1] (0.5)	1731
France	7.3[2] (1.8)	727
Germany	3.1 (0.8)	621
Italy	2.8 (0.6)	853
Lebanon	1.8[1] (0.7)	595
Mexico	1.9[1] (0.4)	1736
Netherlands	5.0 (1.6)	516
Spain	1.2[1] (0.6)	960
USA[3]	5.2 (0.6)	3197
Total	3.4 (0.4)	11422

Cross-national community sample:

* LTP adult ADHD: 3.4% (range 1.8-7.3%)
* ADHD in subjects with SUD: 12.5%
* $OR_{ADHD/SUD}$ = 4.0 (95% CI: 2.8-5.8)
* ADHD first: 99.0%
  SUD first: 0.5%
  same year: 0.5%
* ADHD in Tx: USA 13%, rest 0-3%

How is it in Tx seeking SUD patients →

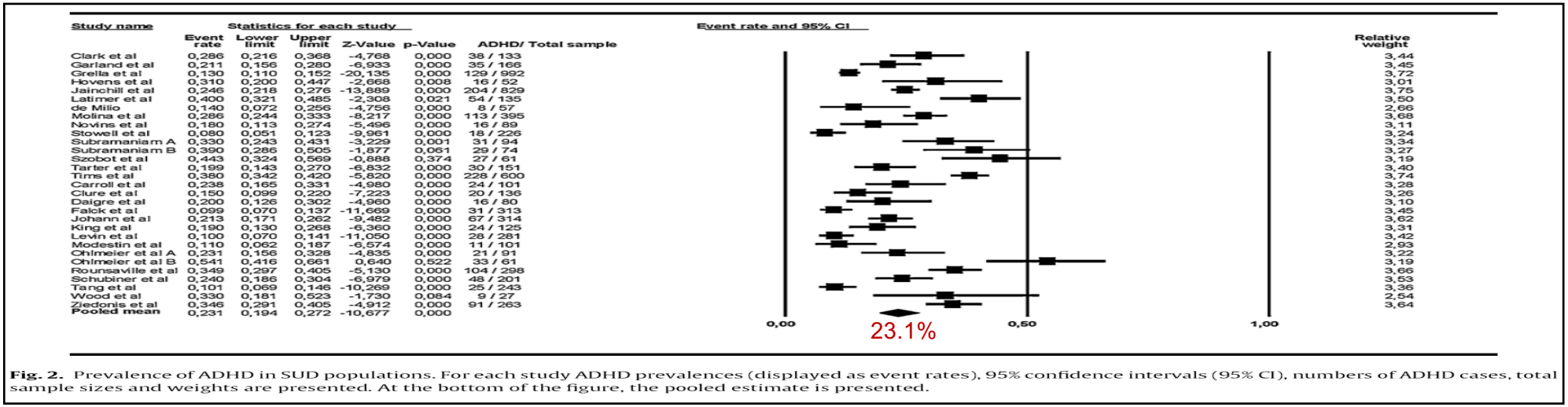
Review

Prevalence of attention-deficit hyperactivity disorder in substance use disorder patients: A meta-analysis and meta-regression analysis

Katelijne van Emmerik-van Oortmerssen[a,b,*], Geurt van de Glind[c], Wim van den Brink[b], Filip Smit[c,d], Cleo L. Crunelle[b,e], Marije Swets[a], Robert A. Schoevers[f,a]

ADHD in SUD patients Cross-Sectional

DAD, 2012



Study name	Event rate	Lower limit	Upper limit	Z-Value	p-Value	ADHD/ Total sample	Relative weight
Clark et al	0,286	0,216	0,368	-4,768	0,000	38 / 133	3,44
Garland et al	0,211	0,156	0,280	-6,933	0,000	35 / 166	3,45
Grella et al	0,130	0,110	0,152	-20,135	0,000	129 / 992	3,72
Hovens et al	0,310	0,200	0,447	-2,668	0,008	16 / 52	3,01
Jainchill et al	0,246	0,218	0,276	-13,689	0,000	204 / 829	3,75
Latimer et al	0,400	0,321	0,485	-2,308	0,021	54 / 135	3,50
de Milio	0,140	0,072	0,256	-4,756	0,000	8 / 57	2,66
Molina et al	0,286	0,244	0,333	-8,217	0,000	113 / 395	3,68
Novins et al	0,180	0,113	0,274	-5,496	0,000	16 / 89	3,11
Stowell et al	0,080	0,051	0,123	-9,961	0,000	18 / 226	3,24
Subramaniam A	0,330	0,243	0,431	-3,229	0,001	31 / 94	3,34
Subramaniam B	0,390	0,286	0,505	-1,877	0,061	29 / 74	3,27
Szobot et al	0,443	0,324	0,569	-0,888	0,374	27 / 61	3,19
Tarter et al	0,199	0,143	0,270	-6,832	0,000	30 / 151	3,40
Tims et al	0,380	0,342	0,420	-5,820	0,000	228 / 600	3,74
Carroll et al	0,238	0,165	0,331	-4,980	0,000	24 / 101	3,28
Clure et al	0,150	0,099	0,220	-7,223	0,000	20 / 136	3,26
Daigre et al	0,200	0,126	0,302	-4,960	0,000	16 / 80	3,10
Falck et al	0,099	0,070	0,137	-11,669	0,000	31 / 313	3,45
Johann et al	0,213	0,171	0,262	-9,482	0,000	67 / 314	3,62
King et al	0,190	0,130	0,268	-6,360	0,000	24 / 125	3,31
Levin et al	0,100	0,070	0,141	-11,050	0,000	28 / 281	3,42
Modestin et al	0,110	0,062	0,187	-6,574	0,000	11 / 101	2,93
Ohlmeier et al A	0,231	0,156	0,328	-4,835	0,000	21 / 91	3,22
Ohlmeier et al B	0,541	0,416	0,661	0,640	0,522	33 / 61	3,19
Rounsaville et al	0,349	0,297	0,405	-5,130	0,000	104 / 298	3,66
Schubiner et al	0,240	0,186	0,304	-6,979	0,000	48 / 201	3,53
Tang et al	0,101	0,069	0,146	-10,269	0,000	25 / 243	3,36
Wood et al	0,330	0,181	0,523	-1,730	0,084	9 / 27	2,54
Ziedonis et al	0,346	0,291	0,405	-4,912	0,000	91 / 263	3,64
Pooled mean	0,231	0,194	0,272	-10,677	0,000		

Fig. 2. Prevalence of ADHD in SUD populations. For each study ADHD prevalences (displayed as event rates), 95% confidence intervals (95% CI), numbers of ADHD cases, total sample sizes and weights are presented. At the bottom of the figure, the pooled estimate is presented.

29 studies: 6,689 subjects (4,054 adolescents and 2,635 adults)

Overall prevalence adult ADHD in SUD patients = 23.1% (95% CI: 19.4 - 27.2%)

Prevalence ADHD in cocaine dependence lower than in other SUDs!!

Article

Age of Methylphenidate Treatment Initiation in Children With ADHD and Later Substance Abuse: Prospective Follow-Up Into Adulthood

Prospective: SUD in ADHD patients

AJP, 2008

Salvatore Mannuzza, Ph.D.

Rachel G. Klein, Ph.D.

Nhan L. Truong, M.A.

John L. Moulton III, Ph.D.

Erica R. Roizen, B.A.

Kathryn H. Howell, B.S.

Francisco X. Castellanos, M.D.

Cohort study: 176 children with ADHD

* Treatment: MPH since age 6-12 yrs

* FU: age 18 and 25 yrs

Incidence SUD:

* N=80 (45%); N=49 AUD; N= 64 DUD

Predictors SUD:

* .....

* .....

Screened Attention Deficit/Hyperactivity Disorder as a Predictor of Substance Use Initiation and Escalation in Early Adulthood and the Role of Self-Reported Conduct Disorder and Sensation Seeking: A 5-Year Longitudinal Study with Young Adult Swiss Men

Franz Moggi[a] Deborah Schorno[a] Leila Maria Soravia[a, b]
Meichun Mohler-Kuo[c, d] Natialia Estévez-Lamorte[d] Joseph Studer[e]
Gerhard Gmel[e, f, g, h]

Eur Addict Res. 2020;26(4-5):233-244.

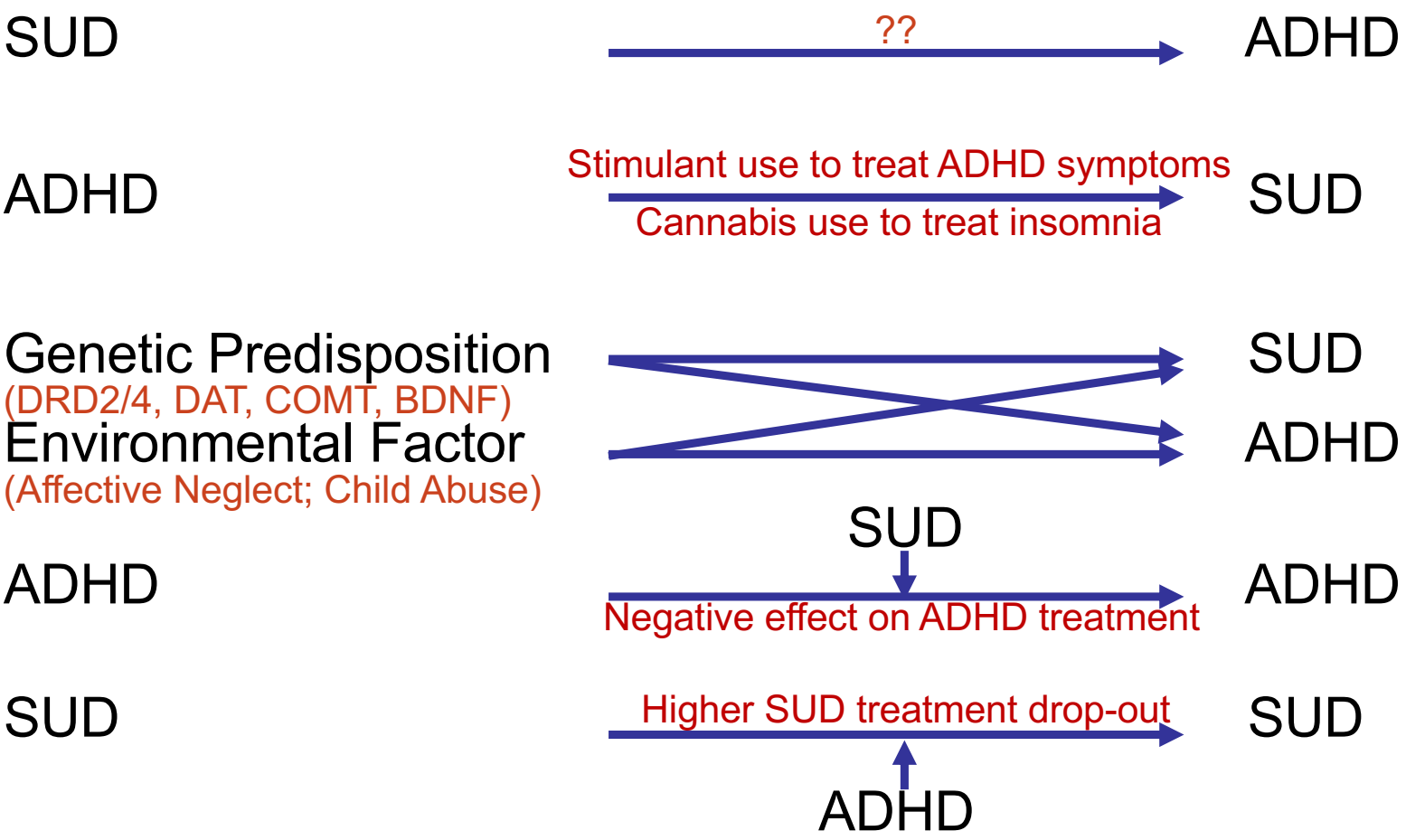
Risk of (relapse in) addiction still present in ADHD screen positives in early childhood (20 → 25 years)

medication, and in case of SS except for sedatives. From a public health perspective, identifying ADHD in childhood or adolescence and continuous treatment and support would be best to lower risks of SU and SUD, but identification of ADHD in early adulthood may be still relevant for that start of specific ADHD and SU interventions.

ADHD and Addiction: A Model

What are mechanisms responsible for SUD+ADHD?

SUD and ADHD: Models



ADHD and Substance Use (Disorders): The Model

Attention-Deficit/Hyperactivity Disorder and Risk of Substance Use Disorder: Developmental Considerations, Potential Pathways, and Opportunities for Research

Brooke S.G. Molina[1] and William E. Pelham Jr.[2]

[1]Departments of Psychiatry and Psychology, University of Pittsburgh, Pittsburgh, Pennsylvania 15213; email: molinab@upmc.edu

[2]Departments of Psychology and Psychiatry, Florida International University, Miami, Florida 33199; email: wpelham@fiu.edu

Annu. Rev. Clin. Psychol. 2014. 10:607–39

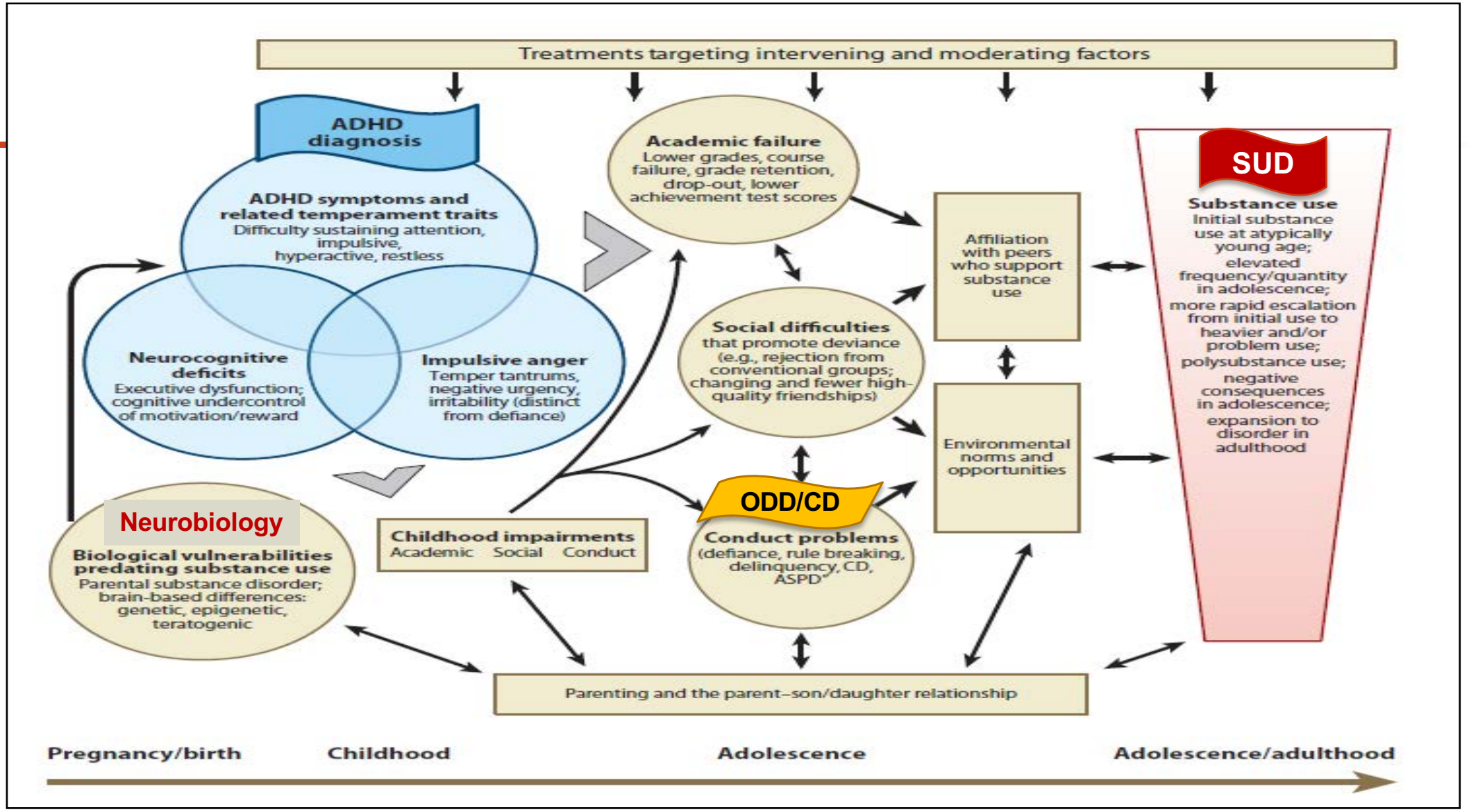
Keywords

attention deficit disorder, adolescent, substance-related disorders, alcoholism, young adult

Abstract

Many opportunities to explain attention-deficit/hyperactivity disorder (ADHD)-related risk of substance use disorder (SUD) remain available for study. We detail these opportunities by considering characteristics of children with ADHD and factors affecting their outcomes side by side with overlapping variables in the developmental literature on SUD etiology. Although serious conduct problems are a known contributor to ADHD-related risk of SUD, few studies have considered their emergence developmentally and in relation to other candidate mediators and moderators that could also explain risk and be intervention targets. Common ADHD-related impairments, such as school difficulties, are in need of research. Heterogeneous social impairments have the potential for predisposing, and buffering, influences. Research on neurocognitive domains should move beyond standard executive function batteries to measure deficits in the interface between cognitive control, reward, and motivation. Ultimately, maximizing prediction will depend, as it has in the SUD literature, on simultaneous consideration of multiple risk factors.

* ADHD Sxx → Personality Traits → SUD
* ADHD → ODD/CD → SUD
* ADHD → Academic/ Vocational Problems → SUD
* ADHD → Social Problems → SUD
* ADHD → Neurocognitive Deficits → SUD
* ADHD → Problems → Low Self-Esteem → MDD → SUD
* ADHD → Failures → Impulsive Aggression → SUD
* ADHD → Positive Expectancies about Drugs → SUD
* ADHD → Deficient Coping Skills → SUD
* ADHD → Deficient Parenting → SUD
* ADHD → Stimulant Tx (?) → SUD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Genetics of Addiction and ADHD

What is the role of genetics in this comorbidity

Alcohol and illicit drug dependence among parents: associations with offspring externalizing disorders

N. R. Marmorstein[1]*, W. G. Iacono[2] and M. McGue[2]

Family Study

Psychol Med 2008

Table 2. *Risk for offspring diagnoses associated with parental alcohol and drug dependence*

	Parental alcohol dependence[a]		Parental drug dependence[b]		Parental cannabis dependence[b]		Parental non-cannabis drug dependence[b]	
Offspring disorder	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
ADHD	2.77*	1.18–6.46	1.65	0.63–4.32	0.88	0.25–3.19	4.01**	1.66–9.72
ODD	2.28***	1.46–3.56	2.02**	1.20–3.39	1.14	0.65–1.99	2.33**	1.29–4.23
CD	1.85**	1.27–2.68	1.70*	1.04–2.78	1.61	0.94–2.76	1.78*	1.00–3.18
AAB	2.25**	1.22–4.14	1.93*	1.01–3.66	1.55	0.79–3.03	2.49**	1.25–4.98
Nicotine dependence	1.96**	1.27–3.05	1.77*	1.05–2.99	1.55	0.87–2.78	2.71***	1.52–4.84
Alcohol dependence	2.18**	1.32–3.61	1.98*	1.14–3.42	1.97*	1.11–3.49	2.06*	1.16–3.65
Drug dependence	2.25*	1.09–4.62	2.97**	1.49–5.88	2.73**	1.38–5.42	4.00**	2.02–7.93

OR, Odds ratio; CI, confidence interval; ADHD, attention deficit hyperactivity disorder; ODD, oppositional defiant disorder; CD, conduct disorder; AAB, adult antisocial behavior.

[a] Adjusting for parental drug dependence.

[b] Adjusting for parental alcohol dependence.

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$.

Parental alcohol and drug addiction are associated with addiction and other externalising disorders (ADHD/CD/ODD) in their children

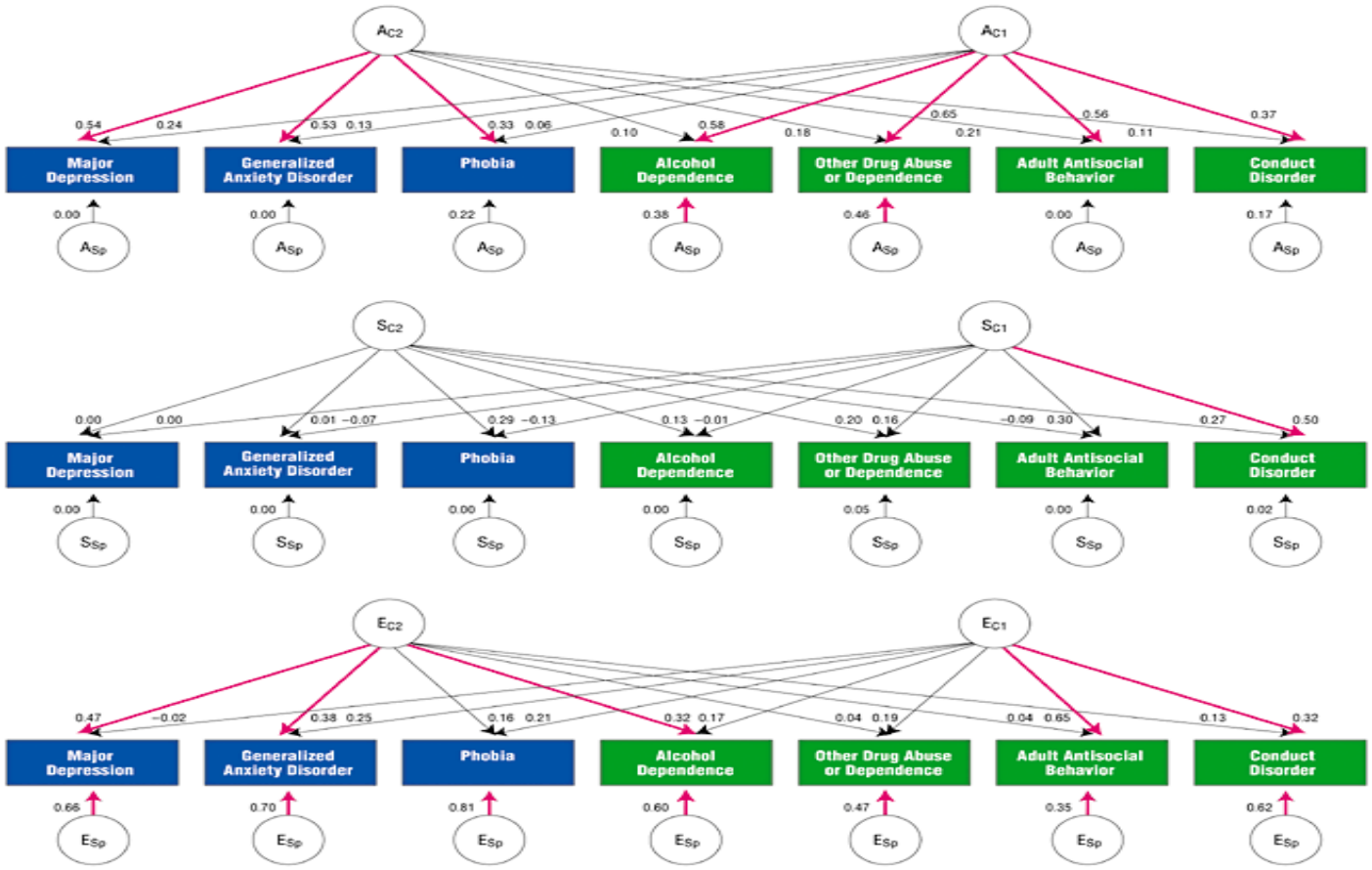
Twin Study CMDs

ORIGINAL ARTICLE

The Structure of Genetic and Environmental Risk Factors for Common Psychiatric and Substance Use Disorders in Men and Women

Kenneth S. Kendler, MD; Carol A. Prescott, PhD; John Myers, MS; Michael C. Neale, PhD

AGP, 2003



Additive Genetic

Common factors

Specific factors

Shared Environment

Common factors

Specific factors

Unique Environment

Common factors

Specific factors

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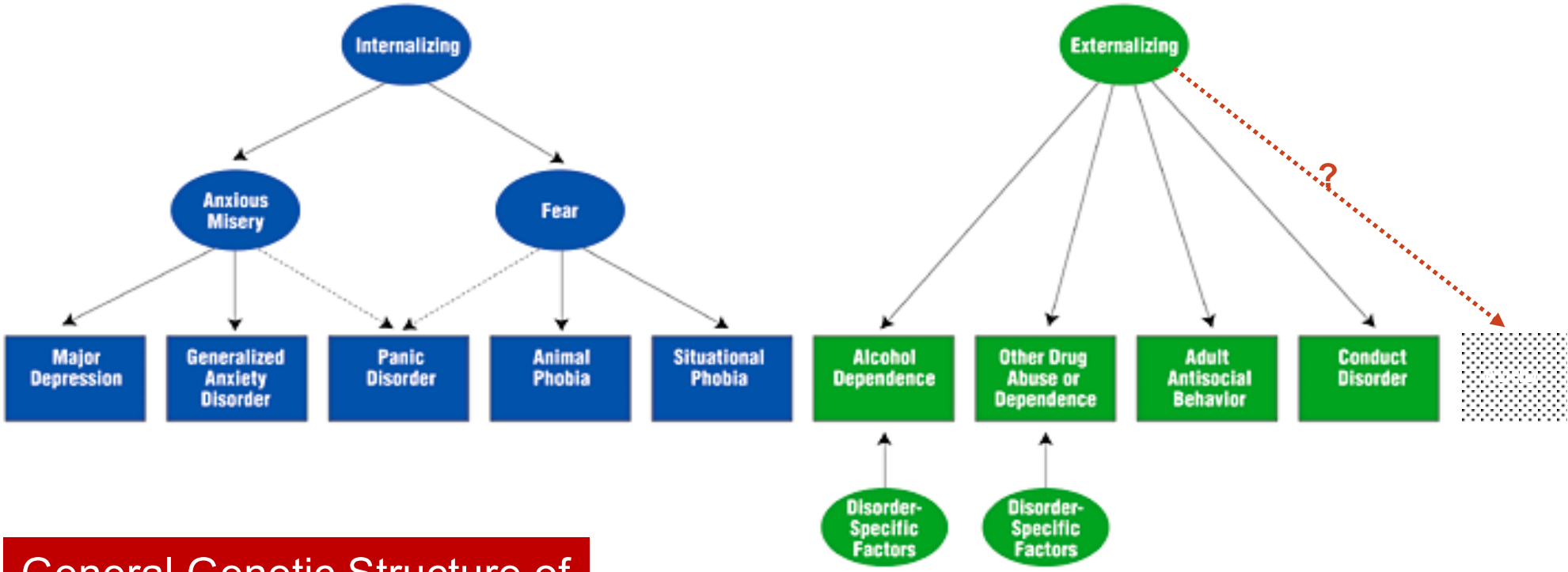


General Genetic Structure of Common Mental Disorders

Genetic and environmental influences on the relation between adult ADHD symptoms and self-reported problem drinking in 6,024 Dutch twins

Derks EM[1*], Vink JM[2,3], Willemsen G[2,4], van den Brink W[1], Boomsma DI[2,3,4]

Twin Study

Psychol Med 2014

ABSTRACT

Background. Both cross-sectional and longitudinal studies have shown an association between ADHD and problematic alcohol use. In adults, a positive correlation between these traits has been reported, but it is yet unknown to what extent this association is explained by genetic and environmental factors.

Methods. Data on ADHD and alcohol consumption were collected in 6,024 adult Dutch twins. ADHD symptoms were assessed using the ADHD-Index of the self-report version of Conners' Adult ADHD Rating Scales (CAARS – S:SV). Problem drinking was defined as meeting at least two self-reported alcohol-related problems on the CAGE questionnaire. Structural equation modelling was applied to the MZ and DZ bivariate data to estimate genetic and environmental influences.

Results. The heritability of the ADHD-index and problem drinking are 38% and 50%, respectively. The genetic correlation, the extent to which genes contributing to individual differences in ADHD symptoms overlap with those in problem drinking, is substantial (r=0.49). The phenotypic correlation (r=0.30) between ADHD-index scores and problem drinking is for 91% explained by genetic influences and for 9% by non-shared environmental influences. No significant gender differences are found.

Conclusions. This study convincingly shows that ADHD symptoms and problem drinking are moderately but significantly correlated in adults; genetic factors are primarily responsible for this correlation. This suggests that early interventions are required to prevent adolescents with ADHD from developing problematic levels of alcohol use. Furthermore, clinicians who treat alcohol dependent patients should be aware that the patient may have a comorbid condition of ADHD; integrated interventions are required.

Heritability:

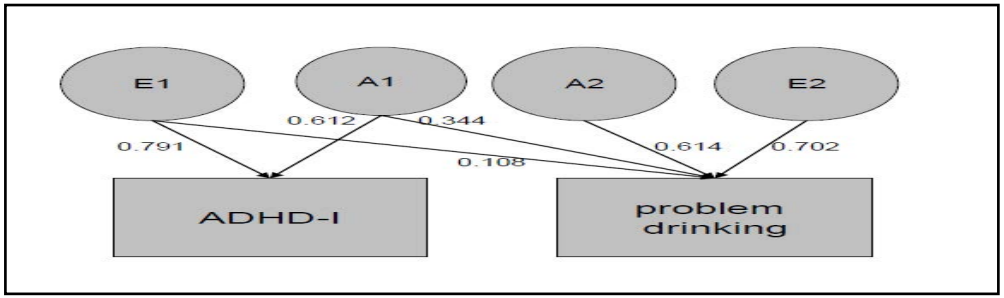
* h^2_{ADHD} = .38, h^2_{AUD} = .50

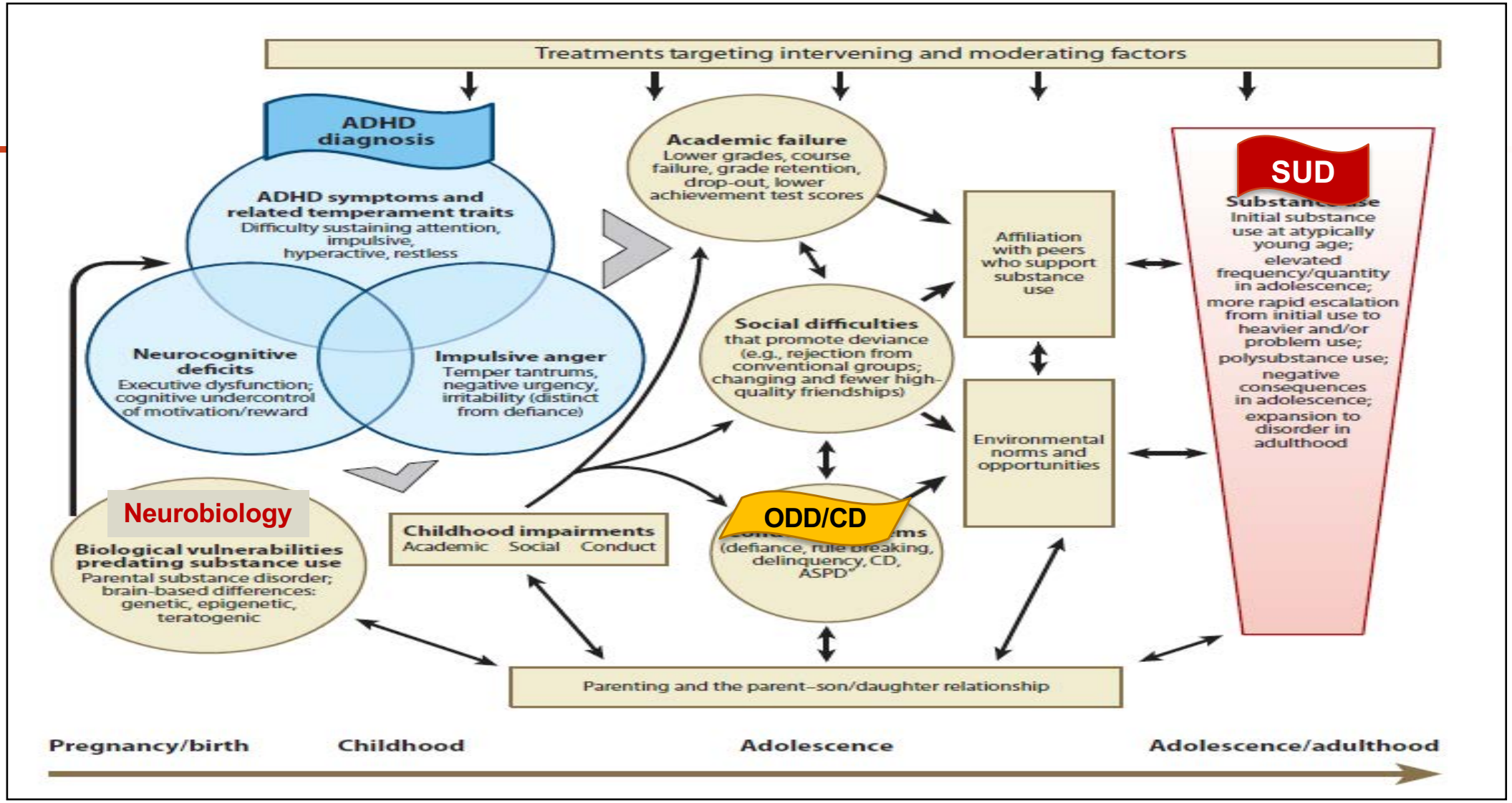
Phenotypic correlation ADHD-AUD:

* r=.30

Causality phenotypic correlation:

* 91% explained by genetic factors
* 9% explained by non-shared environment

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Biological vulnerabilities predating substance use
Parental substance disorder; brain-based differences: genetic, epigenetic, teratogenic
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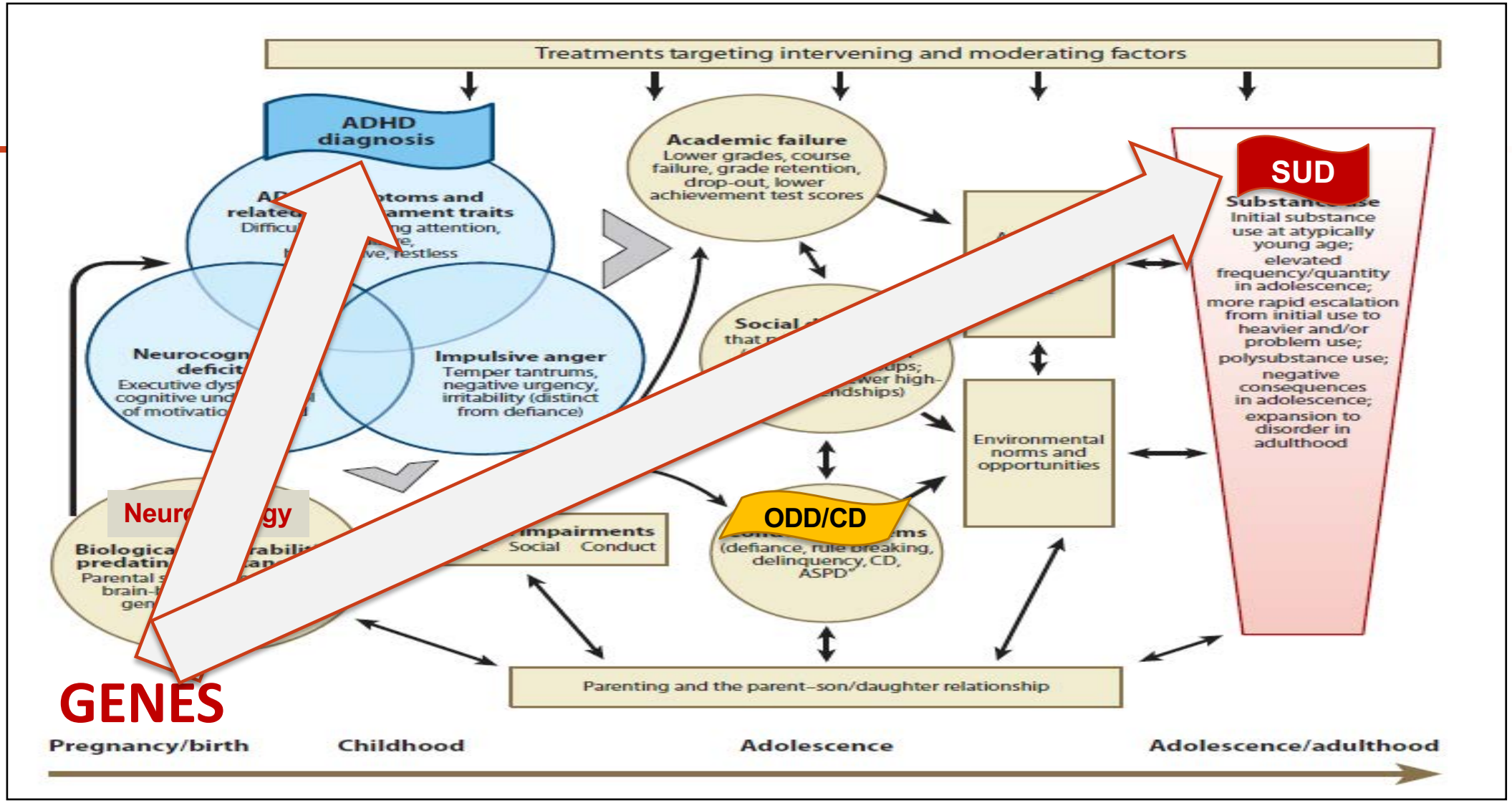
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Heritabilty and GenxEnvironment interactions

Interaction between dopamine D2 receptor genotype and parental rule-setting in adolescent alcohol use: evidence for a gene-parenting interaction

CS van der Zwaluw[1], RCME Engels[1], AA Vermulst[1], B Franke[2,3], J Buitelaar[3], RJ Verkes[3] and RHJ Scholte[1]

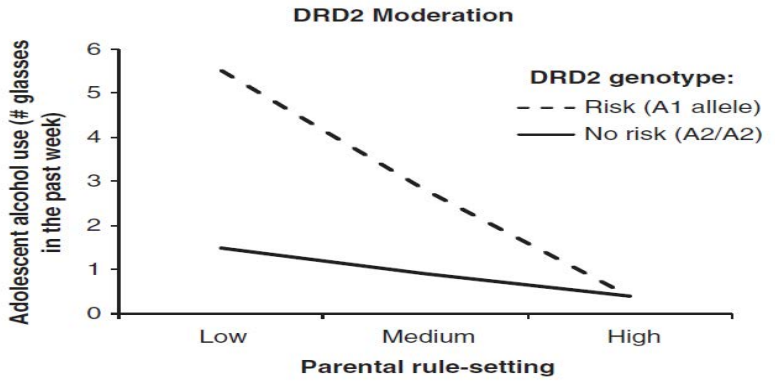
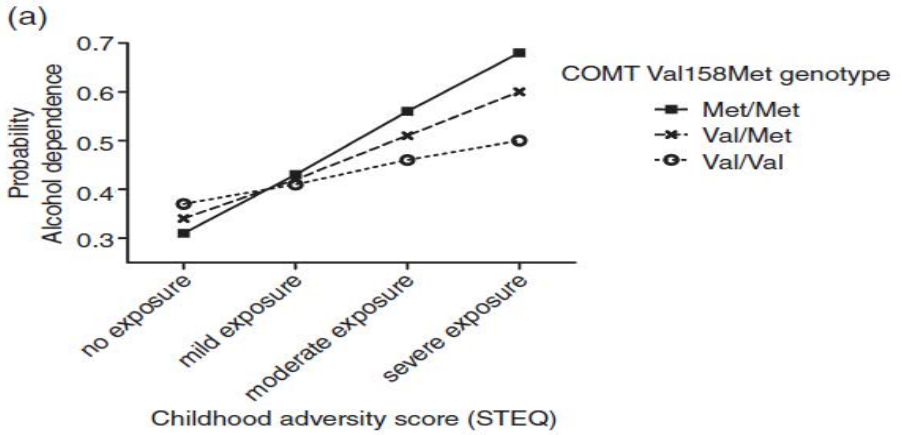


Figure 2 Interaction between *DRD2* genotype and parental rule-setting at T2 on adolescent alcohol use at T3.

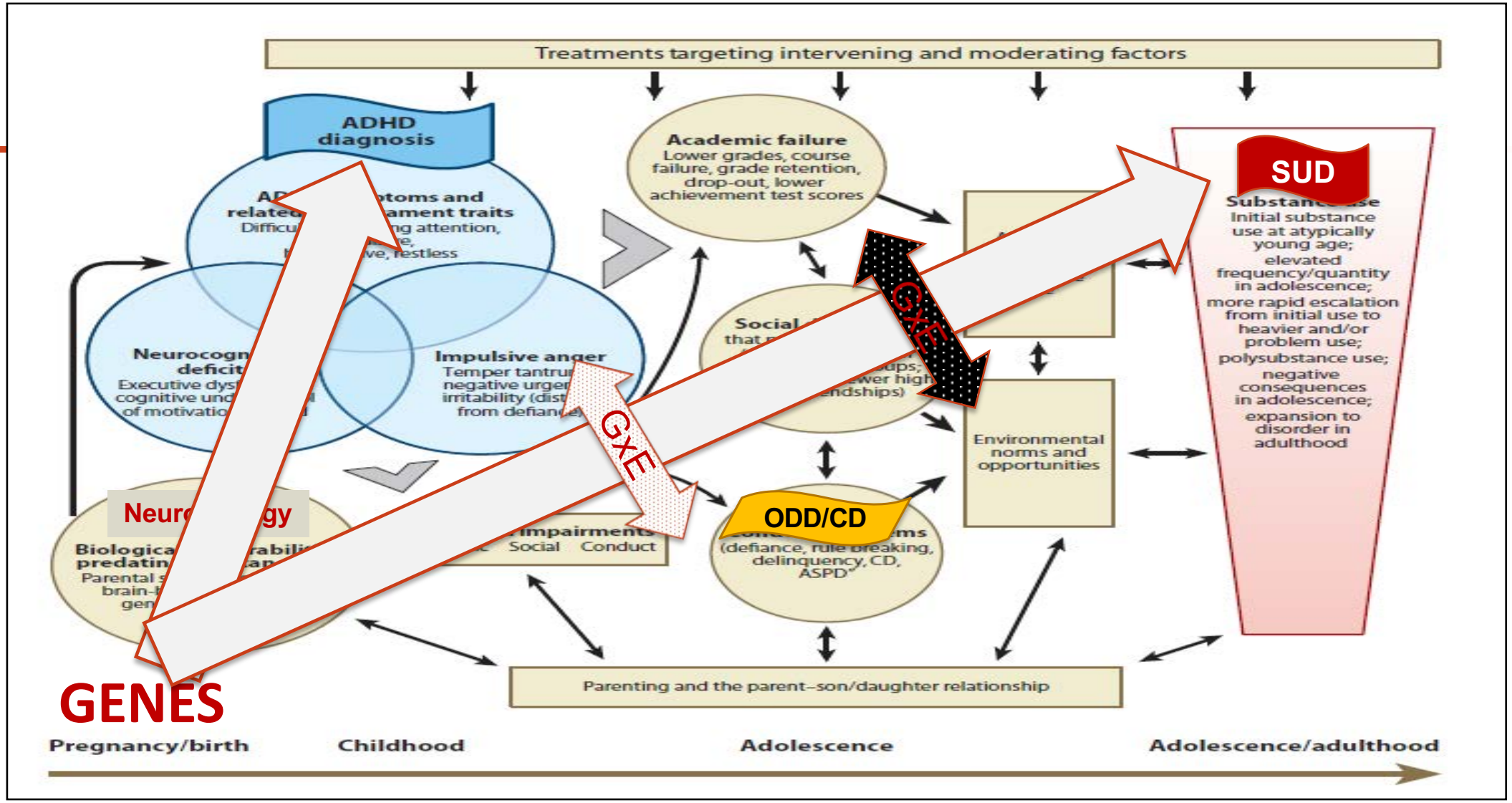
Genetic variation in DRD2-gene moderates effect Of parental behavior on risk of early age drinking

***COMT* Val158Met modulates the effect of childhood adverse experiences on the risk of alcohol dependence**

Arnt F. A. Schellekens[1,2], Barbara Franke[3], Bart Ellenbroek[4], Alexander Cools[1], Cor A. J. de Jong[2], Jan K. Buitelaar[1] & Robbert-Jan Verkes[5]



Genetic variation in COMT-gene moderates effect of early childhood adversity on risk of AD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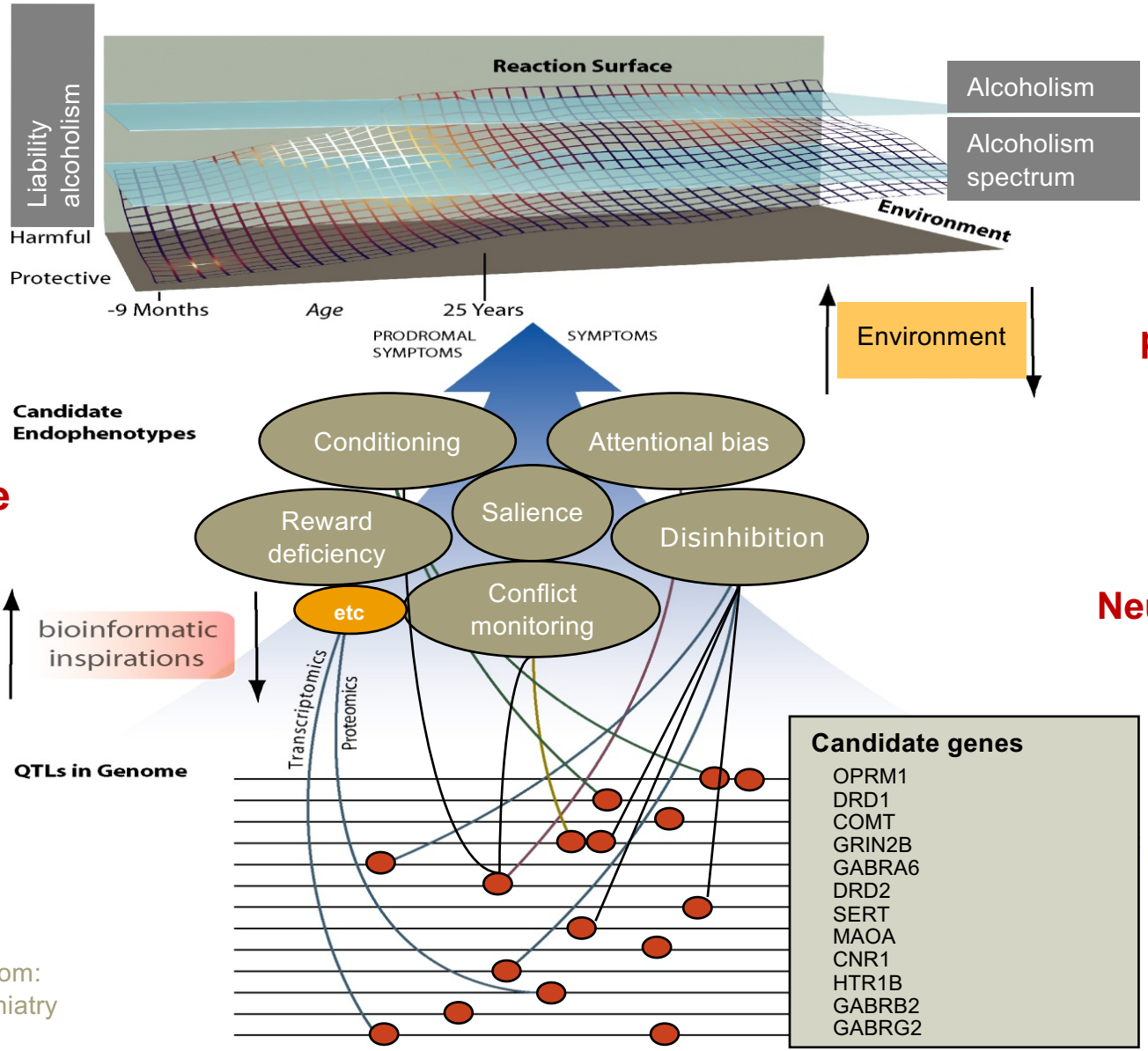
Neurobiology of Addiction-ADHD Comorbidity

Do ADHD and SUD have a similar underlying neurobiology?

Phenotype

Endophenotype

Genotype



Social support

Insight-oriented psychotherapy

CBT

Medication Neuromodulation

Pharmaco-genetics

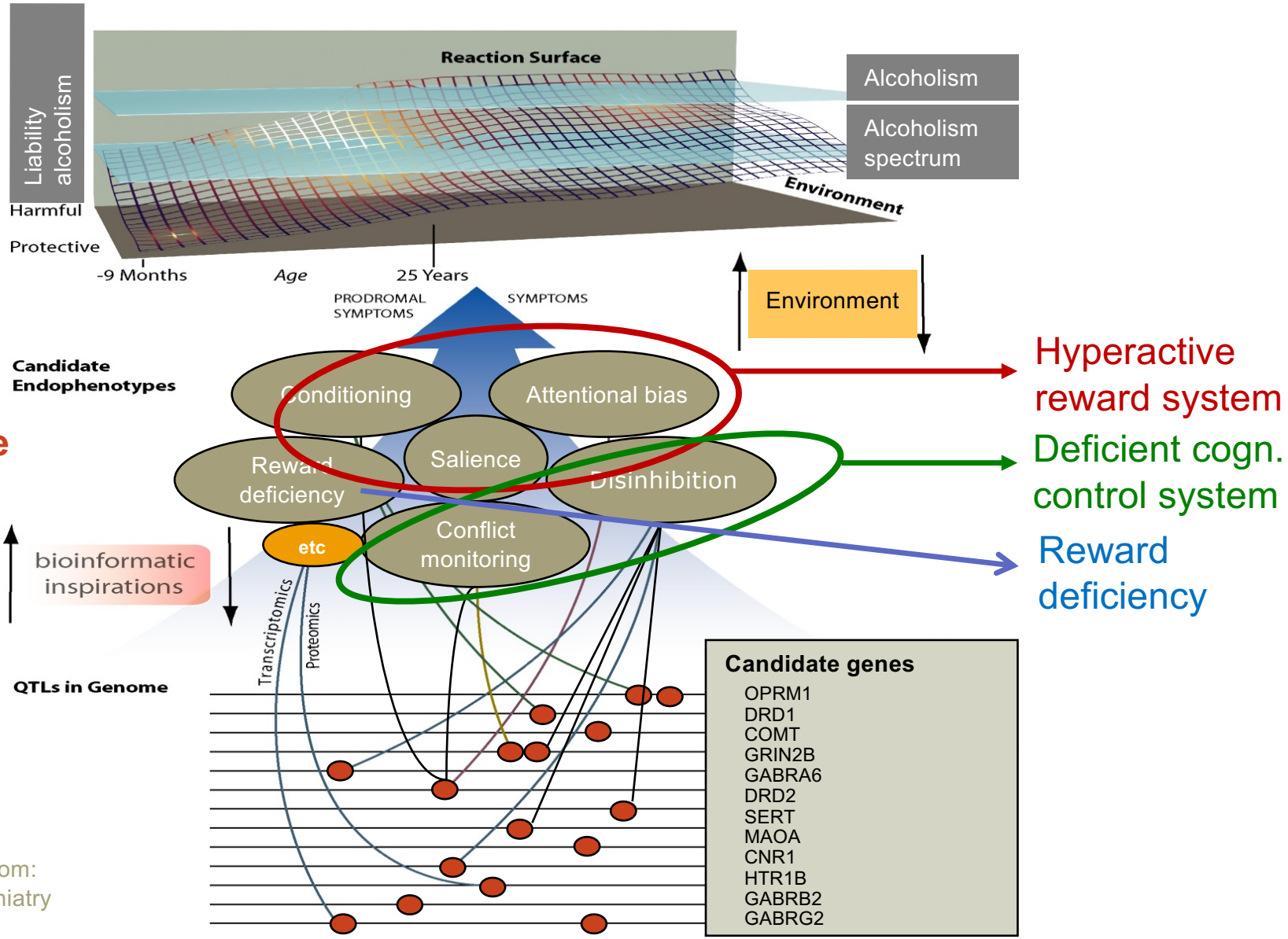
Gene therapy

Ooteman et al (2006) adapted from:
Gottesman & Gould. Am J Psychiatry 2003;160:636–645

Phenotype

Endophenotype

Genotype

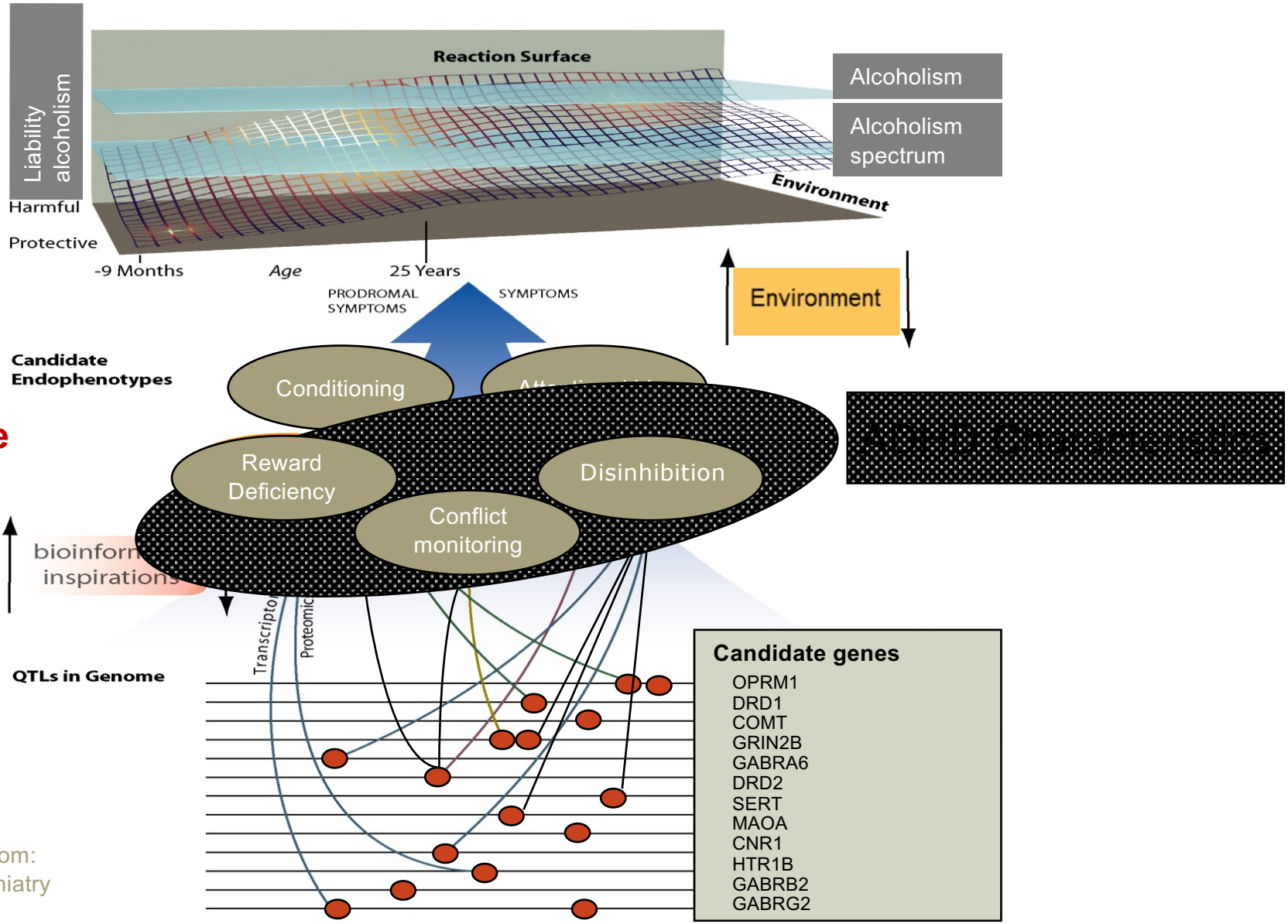


Ooteman et al (2006) adapted from:
Gottesman & Gould. Am J Psychiatry 2003;160:636–645

Phenotype

Endophenotype

Genotype



Ooteman et al (2006) adapted from:
Gottesman & Gould. Am J Psychiatry 2003;160:636–645

Stop What You're Doing! — An fMRI Study on Comparisons of Neural Subprocesses of Response Inhibitio in ADHD and Alcohol Use Disorder

Sarah Gerhardt [1], Mathias Luderer [2], Jan M. Bumb [1], Esther Sobanski [3,4], Franz Moggi [5], Falk Kiefer [1,6,7] and Sabine Vollstädt-Klein [1,8]*

frontiers in Psychiatry

2021

Yes, but ….

Conclusions: Even though deficits in response inhibition are related to both ADHD and AUD, neural activation and recruited networks during response inhibition differ regarding both neuronal subprocesses and examined groups. While a replication of this study is needed in a larger sample, the results suggest that tasks have to be carefully selected when examining neural activation patterns of response inhibition either in research on various psychiatric disorders or transdiagnostic questions.

Neuroimaging the Neural Correlates of Increased Risk for Substance Use Disorders in Attention-Deficit/Hyperactivity Disorder—A Systematic Review

Vitria Adisetiyo, PhD,[1] Kevin M. Gray, MD[2]

The American Journal on Addictions, 26: 99–111, 2017

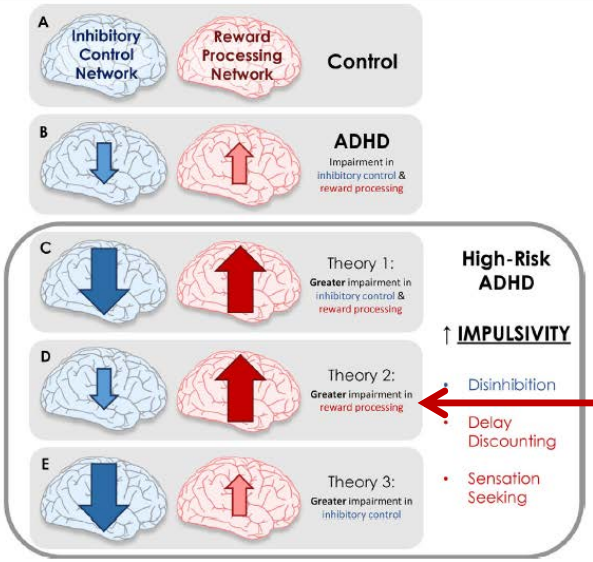
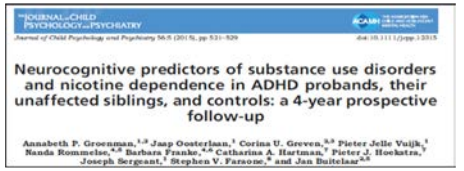


FIGURE 1. Impulsivity-Related Theories of High-Risk ADHD: Exaggerated Imbalance of Brain Networks. (A) Compared to typically developing controls, (B) individuals with low-risk ADHD have impairments in both the inhibitory control and reward processing brain networks: weaker inhibitory control (reduced macro/microstructure; hypoactive at-rest and during associated tasks) and hyperprimed reward processing (greater macro/microstructure; hyperactive at-rest but hypoactive during associated tasks). High impulsivity is implicated as an important mechanism underlying increased SUD risk in ADHD and corresponds to greater disinhibition, delay discounting and sensation seeking. Impulsivity-related theories of high-risk ADHD suggest these impulsive behaviors stem from a greater degree of impairment (C) in both the inhibitory control and reward processing networks compared to low-risk ADHD, (D) in mainly the reward processing network or (E) in mainly the inhibitory control network.

High-Risk ADHD:
Comorbid DBD (ODD/CD), familial SUD and/or early substance use


Neurocognitive predictors of substance use disorders and nicotine dependence in ADHD probands, their unaffected siblings, and controls: a 4-year prospective follow-up

Discussion/Conclusions: An exaggerated imbalance between the inhibitory control network and the motivation-reward processing network is theorized to distinguish individuals with high-risk ADHD. Preliminary findings suggest that an exaggerated aberrant reward processing network may be the driving neural correlate of increased SUD risk in ADHD.

Neuroimaging the Neural Correlates of Increased Risk for Substance Use Disorders in Attention-Deficit/Hyperactivity Disorder—A Systematic Review

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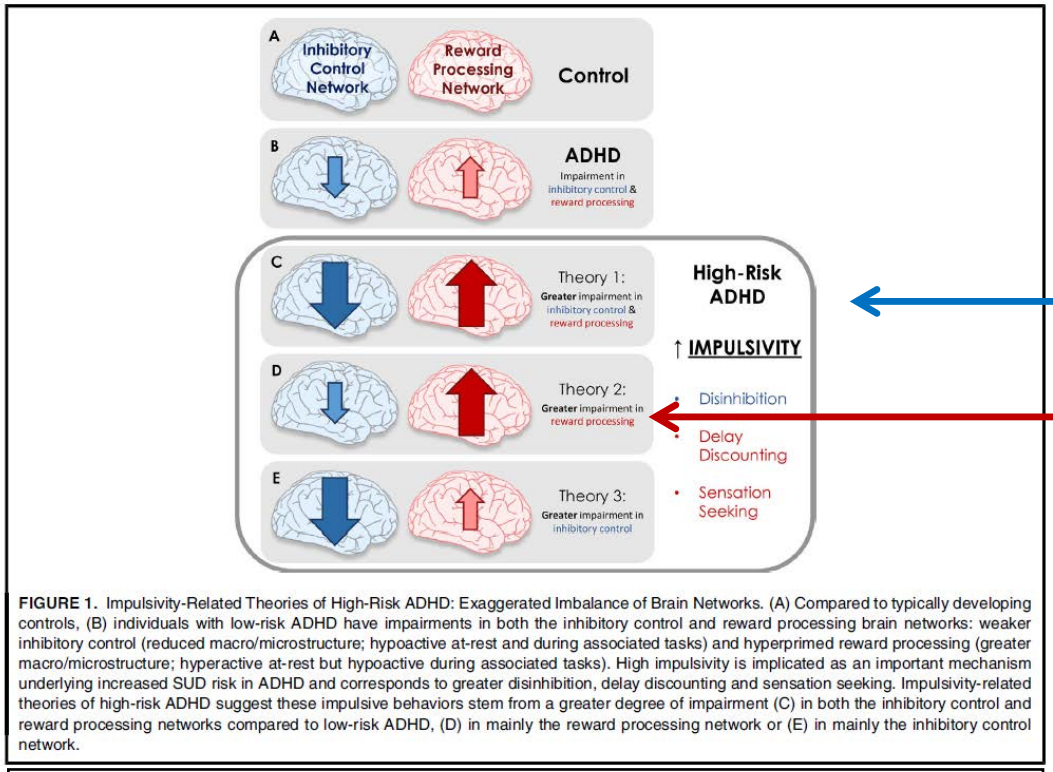


FIGURE 1. Impulsivity-Related Theories of High-Risk ADHD: Exaggerated Imbalance of Brain Networks. (A) Compared to typically developing controls, (B) individuals with low-risk ADHD have impairments in both the inhibitory control and reward processing brain networks: weaker inhibitory control (reduced macro/microstructure; hypoactive at-rest and during associated tasks) and hyperprimed reward processing (greater macro/microstructure; hyperactive at-rest but hypoactive during associated tasks). High impulsivity is implicated as an important mechanism underlying increased SUD risk in ADHD and corresponds to greater disinhibition, delay discounting and sensation seeking. Impulsivity-related theories of high-risk ADHD suggest these impulsive behaviors stem from a greater degree of impairment (C) in both the inhibitory control and reward processing networks compared to low-risk ADHD, (D) in mainly the reward processing network or (E) in mainly the inhibitory control network.

High-Risk ADHD:
Comorbid DBD (ODD/CD), familial SUD and/or early substance use

The indirect effects of childhood attention deficit hyperactivity disorder on alcohol problems in adulthood through unique facets of impulsivity

Sarah L. Pedersen[1], Christine A. P. Walther[2], Seth C. Harty[3], Elizabeth M. Gnagy[4], William E. Pelham[4] & Brooke S. G. Molina[1]

Neurocognitive predictors of substance use disorders and nicotine dependence in ADHD probands, their unaffected siblings, and controls: a 4-year prospective follow-up

Discussion/Conclusions: An exaggerated imbalance between the inhibitory control network and the motivation-reward processing network is theorized to distinguish individuals with high-risk ADHD. Preliminary findings suggest that an exaggerated aberrant reward processing network may be the driving neural correlate of increased SUD risk in ADHD.

ADHD and Addiction: Prevention

Is it possible to prevent the development of addiction in children with ADHD?

Article

Age of Methylphenidate Treatment Initiation in Children With ADHD and Later Substance Abuse: Prospective Follow-Up Into Adulthood

SUD in ADHD patients

AJP, 2008

Salvatore Mannuzza, Ph.D.

Rachel G. Klein, Ph.D.

Nhan L. Truong, M.A.

John L. Moulton III, Ph.D.

Erica R. Roizen, B.A.

Kathryn H. Howell, B.S.

Francisco X. Castellanos, M.D.

Cohort study: 176 children with ADHD

* Treatment: MPH since age 6-12 yrs

* FU: age 18 and 25 yrs

Incidence SUD:

* N=80 (45%); N=49 AUD; N= 64 DUD

Predictors SUD:

* Only age at starting MPH treatment:

The earlier the start, the smaller the risk of SUD

Stimulant Tx ADHD and SUD: A meta-analysis

Original Investigation | META-ANALYSIS

Stimulant Medication and Substance Use Outcomes
A Meta-analysis

Kathryn L. Humphreys, MA, EdM; Timothy Eng, BS; Steve S. Lee, PhD

IMPORTANCE Psychostimulant medication is an efficacious treatment for childhood attention-deficit/hyperactivity disorder, yet controversy remains regarding potential iatrogenic effects of stimulant medication, particularly with respect to increasing susceptibility to later substance use disorders. However, stimulant treatment was previously reported to reduce the risk of substance problems.

OBJECTIVE To meta-analyze the longitudinal association between treatment with stimulant medication during childhood and later substance outcomes (ie, lifetime substance use and substance abuse or dependence).

DATA SOURCES Studies published between January 1980 and February 2012 were identified using review articles, PubMed, and pertinent listservs.

STUDY SELECTION Studies with longitudinal designs in which medication treatment preceded the measurement of substance outcomes.

DATA EXTRACTION AND SYNTHESIS Odds ratios were extracted or provided by the study authors. Odds ratios were obtained for lifetime use (ever used) and abuse or dependence status for alcohol, cocaine, marijuana, nicotine, and nonspecific drugs for 2565 participants from 15 different studies.

MAIN OUTCOMES AND MEASURES Random-effects models estimated the overall association, and potential study moderators were examined.

RESULTS Separate random-effects analyses were conducted for each substance outcome, with the number of studies ranging from 3 to 11 for each outcome. Results suggested comparable outcomes between children with and without medication treatment history for any substance use and abuse or dependence outcome across all substance types.

CONCLUSIONS These results provide an important update and suggest that treatment of attention-deficit/hyperactivity disorder with stimulant medication neither protects nor increases the risk of later substance use disorders.

JAMA Psychiatry. 2013

Question:
Does stimulant treatment of childhood ADHD result in more or in less addiction?

Method:
Meta-analysis of 15 longitudinal studies with 2,565 participants with start medication before assessment of addiction

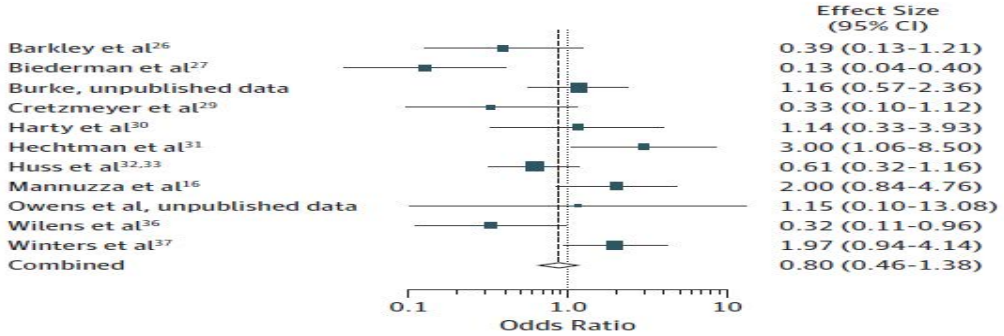
Result: No effect of stimulant treatment on the risk of development of addiction, BUT….

Stimulant Medication and Substance Use Outcomes
A Meta-analysis

Kathryn L. Humphreys, MA, EdM; Timothy Eng, BS; Steve S. Lee, PhD

JAMA Psychiatry. 2013

Figure 1. Alcohol Abuse or Dependence

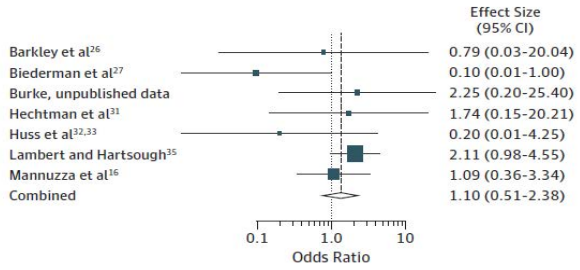


Effect of medication treatment on the risk of alcohol abuse or dependence in children with attention-deficit/hyperactivity disorder.

No overall effect, but lot of heterogeneity explained by:
* percentage stimulant Tx
* time since Dx

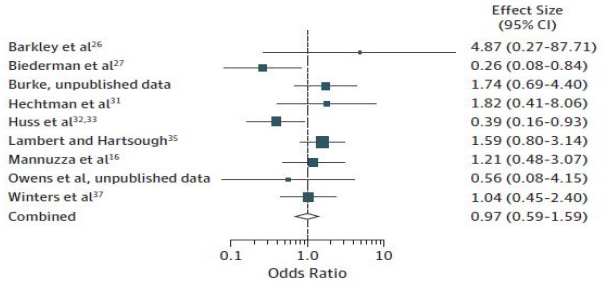
No control for ADHD severity
No control for CD comorbidity

Figure 2. Cocaine Abuse or Dependence



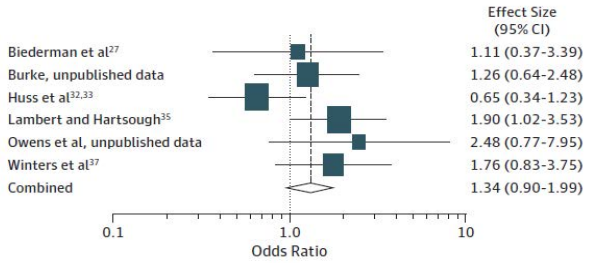
Effect of medication treatment on the risk of cocaine abuse or dependence in children with attention-deficit/hyperactivity disorder.

Figure 3. Marijuana Abuse or Dependence



Effect of medication treatment on the risk of marijuana abuse or dependence in children with attention-deficit/hyperactivity disorder.

Figure 4. Nicotine Dependence



Effect of medication treatment on the risk of nicotine dependence in children with attention-deficit/hyperactivity disorder.

An informative Study from the Netherlands

(with control for ADHD severity en ODD/CD comorbidity)

BJP, 2013

Stimulant treatment for attention-deficit hyperactivity disorder and risk of developing substance use disorder

Annabeth P. Groenman, Jaap Oosterlaan, Nanda N. J. Rommelse, Barbara Franke, Corina U. Greven, Pieter J. Hoekstra, Catharina A. Hartman, Marjolein Luman, Herbert Roeyers, Robert D. Oades, Joseph A. Sergeant, Jan K. Buitelaar* and Stephen V. Faraone*

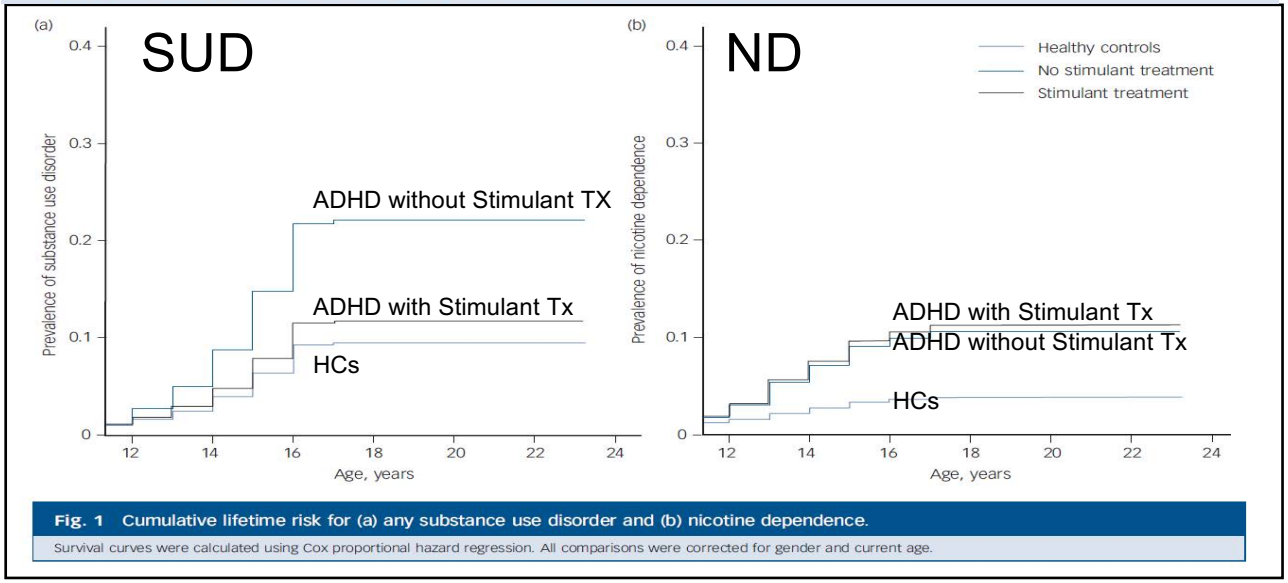


Fig. 1 Cumulative lifetime risk for (a) any substance use disorder and (b) nicotine dependence.

Survival curves were calculated using Cox proportional hazard regression. All comparisons were corrected for gender and current age.

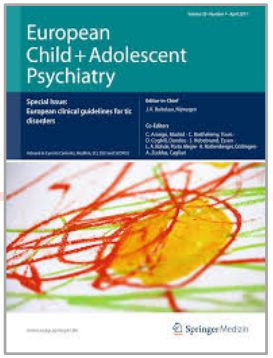
* Stimulant Tx normalizes risk for AUD/DUD until age 17

* Stimulant Tx unfortunately has no effect on risk for ND

*** Stimulant Tx effect is larger if stimulant Tx was started at a younger age!**

Stimulant treatment profiles predicting co-occurring substance use disorders in individuals with attention-deficit/hyperactivity disorder

Annabeth P. Groenman[1,2,13] · Lizanne J. S. Schweren[2,10] · Wouter Weeda[3] · Marjolein Luman[1] · Siri D. S. Noordermeer[1] · Dirk J. Heslenfeld[1] · Barbara Franke[4,5] · Stephen V. Faraone[6,7] · Nanda Rommelse[8,9] · Catharina A. Hartman[2] · Pieter J. Hoekstra[2] · Jan Buitelaar[5,8,9] · Jaap Oosterlaan[1,11,12]



Eur Child Adolesc Psychiatry. 2019 Sep;28(9):1213-1222.

2019

Data from Dutch part of International Multicenter ADHD Genetics (IMAGE) study:

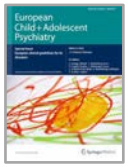
ADHD participants N=303; age at baseline 12 and age at FU = 16.2
HC participants N=2019; age at baseline 12 and age at FU =16.3

Follow-up: 4.2 years

SUD= [either DISC-IV-P + OR AUDIT + OR DAST +]
ND=FTND ≥ 6 OR DISC-IV-P +

Stimulant treatment profiles predicting co-occurring substance use disorders in individuals with attention-deficit/hyperactivity disorder

Annabeth P. Groenman[1,2,13] · Lizanne J. S. Schweren[2,10] · Wouter Weeda[3] · Marjolein Luman[1] · Siri D. S. Noordermeer[1] · Dirk J. Heslenfeld[1] · Barbara Franke[4,5] · Stephen V. Faraone[6,7] · Nanda Rommelse[8,9] · Catharina A. Hartman[2] · Pieter J. Hoekstra[2] · Jan Buitelaar[5,8,9] · Jaap Oosterlaan[1,11,12]



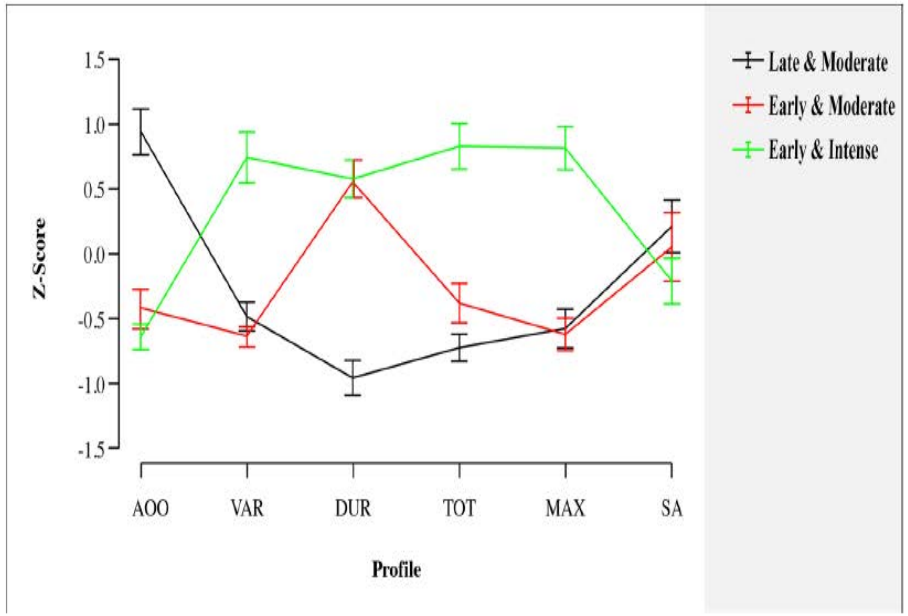


Fig. 2 Community detection outcomes. This figure depicts the three medication subgroups that were defined by the community detection algorithm: (1) a late-and-moderate use group characterized by a late onset of treatment, short duration, and moderate total dose and maximum use, (2) a early-and-moderate use group characterized by a young onset age, a long duration of use, and a late offset of treatment age, and (3) early-and-intense use group characterized by a young onset of treatment age, a variable trajectory of medication use with a long duration, high total dosage, high maximum dosage and early age at treatment offset. *AOO* stimulant medication offset, *VAR* variability of dose (SD), *DUR* duration of use, *TOT* total dose, *MAX* maximum dose, *SA* stop age

Subgroups of medical stimulant use in ADHD participants

Late (11 yrs) & Moderate Dose (24 mg)
N=91

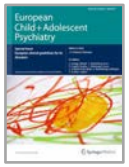
Early (8 yrs) & Moderate Dose (23 mg)
N=51

Early (7yrs) & Intense Dose (53 mg)
N=103

No medical stimulant use
(N=53)

Stimulant treatment profiles predicting co-occurring substance use disorders in individuals with attention-deficit/hyperactivity disorder

Annabeth P. Groenman[1,2,13] · Lizanne J. S. Schweren[2,10] · Wouter Weeda[3] · Marjolein Luman[1] · Siri D. S. Noordermeer[1] · Dirk J. Heslenfeld[1] · Barbara Franke[4,5] · Stephen V. Faraone[6,7] · Nanda Rommelse[8,9] · Catharina A. Hartman[2] · Pieter J. Hoekstra[2] · Jan Buitelaar[5,8,9] · Jaap Oosterlaan[1,11,12]



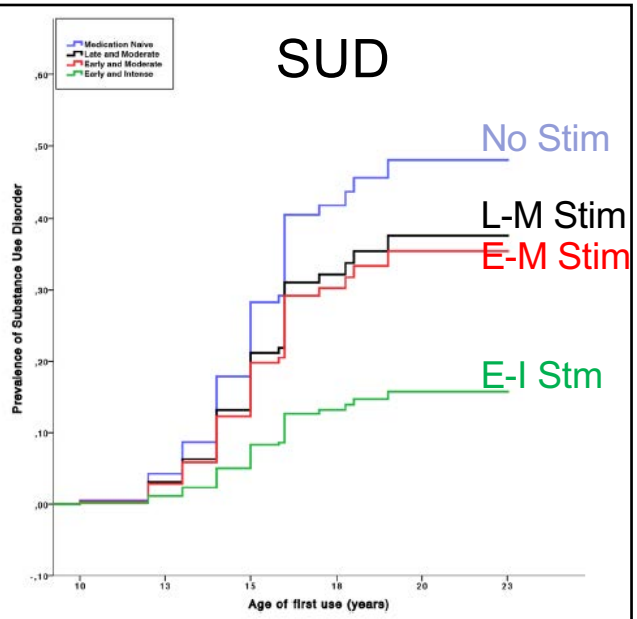


Fig. 3 Cumulative lifetime risk for any substance use disorder. One minus survival curve estimated with cox proportional hazard model for development of SUDs (any alcohol or drug use disorder) in subjects with ADHD with age of first substance use on the *x* axis

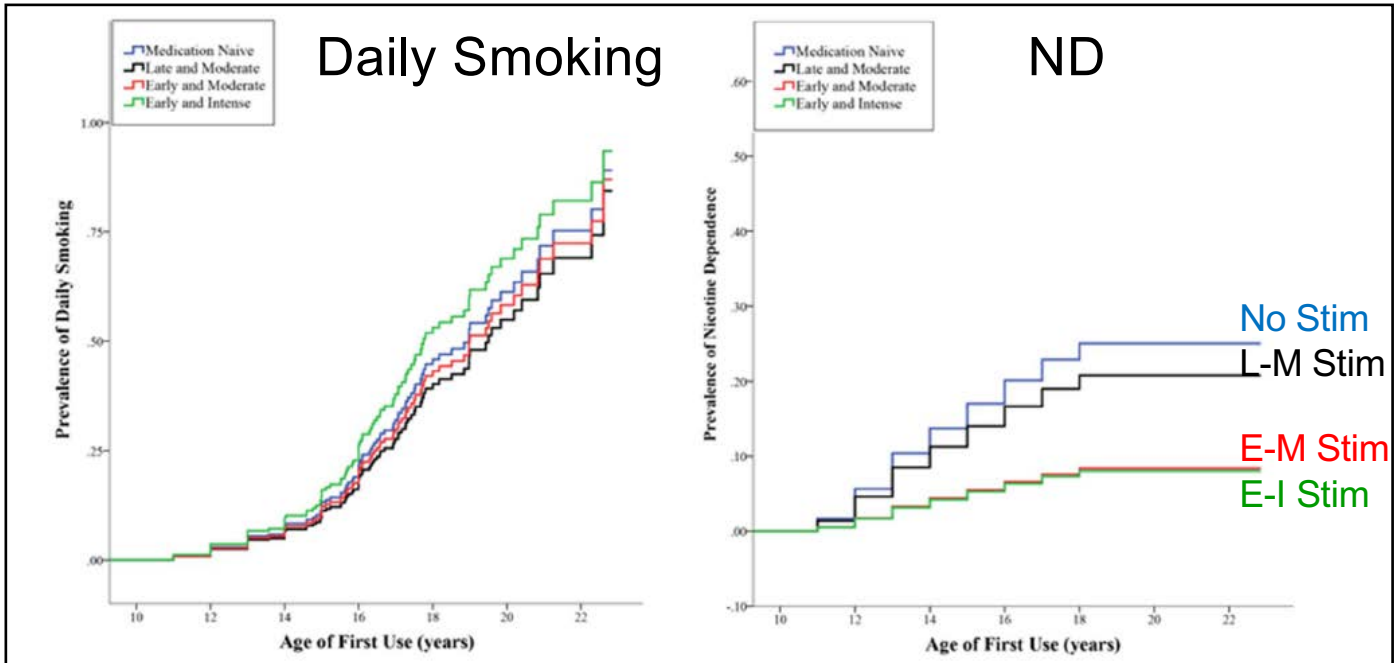


Fig. 4 Cumulative lifetime risk for smoking. One minus survival curve estimated with cox proportional hazard model for development of smoking in subjects with ADHD with age of first cigarette use on the *x* axis. Left panel: daily smoking, right panel: nicotine dependence

SUD: early+intense stimulant use normalizes SUD-risk at least until age 16
ND: early+intense/moderate stimulant use normalizes risk at least until age 16

THE JOURNAL OF CHILD PSYCHOLOGY AND PSYCHIATRY

ACAMH THE ASSOCIATION FOR CHILD AND ADOLESCENT MENTAL HEALTH

Journal of Child Psychology and Psychiatry 55:8 (2014), pp 878–885 doi:10.1111/jcpp.12164

Stimulant ADHD medication and risk for substance abuse

Zheng Chang,[1] Paul Lichtenstein,[1] Linda Halldner,[1,2] Brian D'Onofrio,[3] Eva Serlachius,[4] Seena Fazel,[5] Niklas Långström,[1] and Henrik Larsson[1]

N=28,753

Table 2 Stimulant ADHD medication in 2006 and hazard ratio for substance abuse during 2009

	Hazard ratio for substance abuse during 2009							
	Confounder adjustment				Mediation analysis			
	Model 1: Adjusted for sex, age, and ADHD medication in 2009		Model 2: As in Model 1 + other potential confounders before 2006		Model 3: As in Model 2 + nonsubstance mediators 2006–2008		Model 4: As in Model 3 + substance-related mediators 2006–2008	
Medication	Hazard ratio	95% confidence interval	Hazard ratio	95% confidence interval	Hazard ratio	95% confidence interval	Hazard ratio	95% confidence interval
All patients with an ADHD diagnosis								
Stimulant ADHD medication in January 1, 2006	0.52	0.42–0.66	0.69	0.57–0.84	0.77	0.65–0.93	0.87	0.74–1.03
Duration of treatment with stimulant ADHD medication 2006–2008 (in years)	0.80	0.73–0.88	0.87	0.80–0.94	0.89	0.82–0.96	0.95	0.88–1.02
All patients with an ADHD diagnosis and 15 years or younger on 1 January 2006								
Stimulant ADHD medication in January 1, 2006	0.33	0.20–0.56	0.38	0.23–0.64	0.42	0.26–0.70	0.45	0.27–0.74
Duration of treatment with stimulant ADHD medication 2006–2008 (in years)	0.72	0.61–0.86	0.76	0.63–0.90	0.77	0.65–0.92	0.80	0.68–0.94
All patients with an ADHD diagnosis and 20 years or older on 1 January 2006								
Stimulant ADHD medication in January 1, 2006	0.65	0.46–0.91	0.75	0.58–0.98	0.85	0.67–1.07	0.97	0.78–1.20
Duration of treatment with stimulant ADHD medication 2006–2008 (in years)	0.92	0.82–1.03	0.90	0.91–0.99	0.92	0.84–1.01	0.97	0.89–1.06

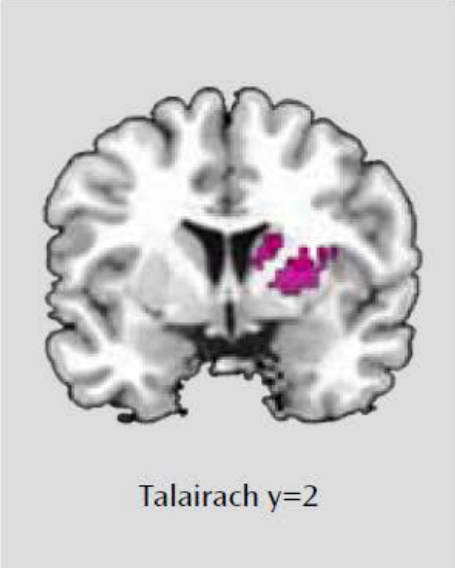
Large Swedish register study: Strongly reduced risk of SUD in ADHD if treated with stimulants **before age 15**

Tomohiro Nakao, M.D., Ph.D.
Joaquim Radua, M.D.
Katya Rubia, Ph.D.
David Mataix-Cols, Ph.D.

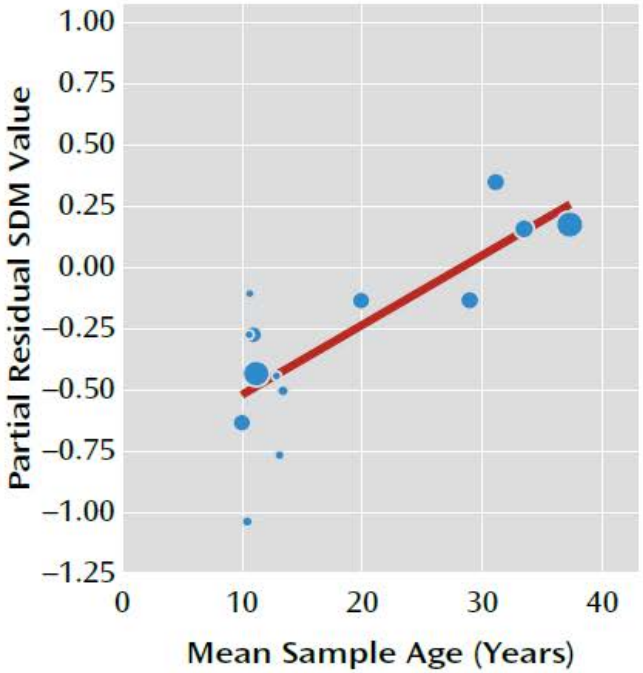
Gray Matter Volume Abnormalities in ADHD: Voxel-Based Meta-Analysis Exploring the Effects of Age and Stimulant Medication

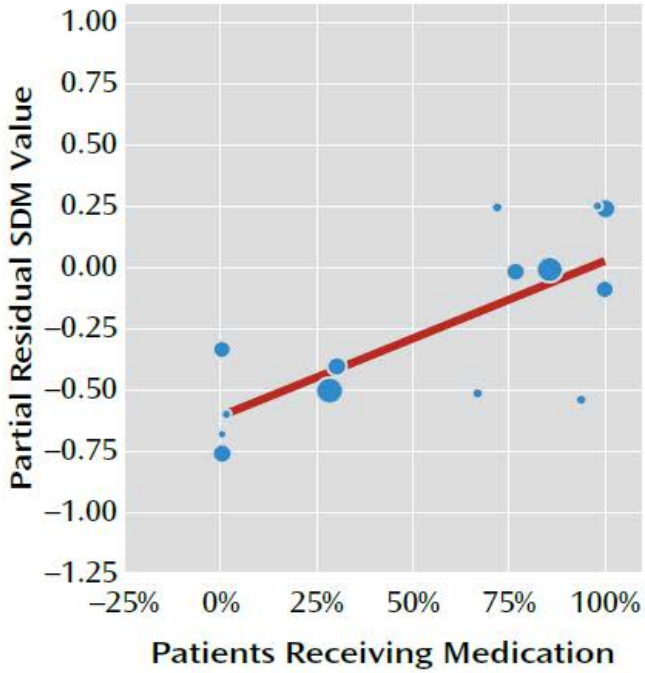
(Am J Psychiatry 2011; 168:1154–1163)

FIGURE 2. Results of the Metaregression Analysis Showing Independent Associations of Mean Age and Percentage of Patients Receiving Stimulant Medication With More Normal Gray Matter Volumes in the Right Basal Ganglia[a]



Right Basal Ganglia





Age and stimulant medication (independently) associated with normalization basal ganglia volume

ADHD and Addiction: Treatment

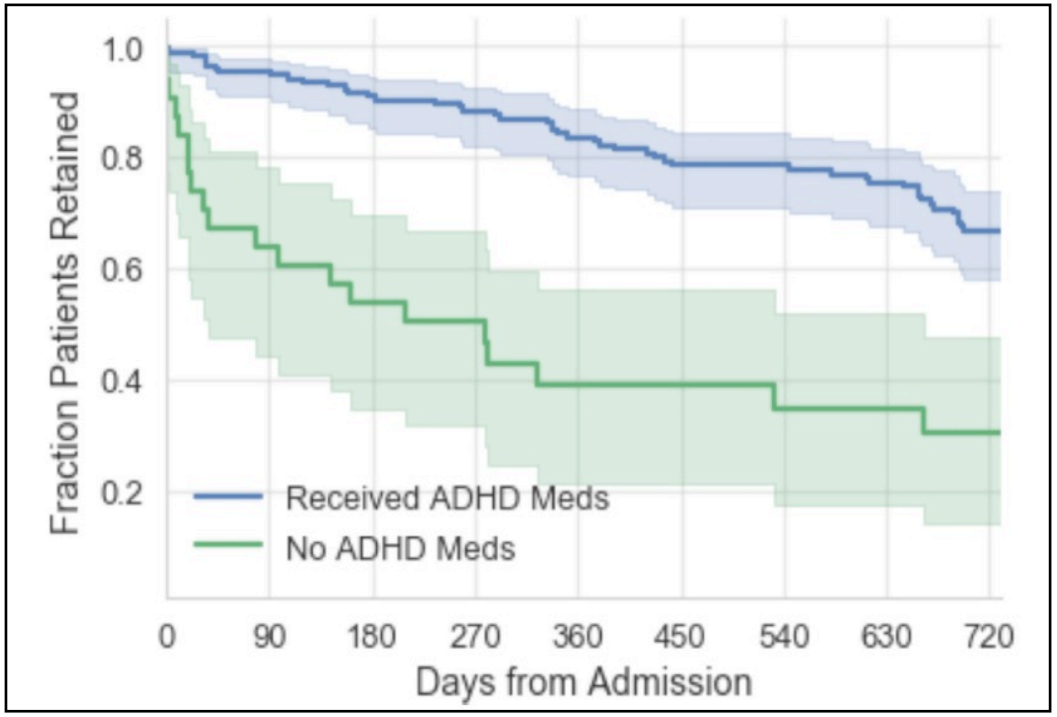
Do we need special interventions to treat ADHD patients with a comorbid addiction?

Pharmacotherapy for attention-deficit hyperactivity disorder (ADHD) and retention in outpatient substance use disorder treatment: a retrospective cohort study.

Kristopher A. Kast, MD,
Department of Psychiatry and Behavioral Sciences, Vanderbilt University Medical Center, Nashville, TN, USA

Vinod Rao, MD PhD,
Department of Psychiatry, Massachusetts General Hospital, Boston, MA, USA

Timothy E. Wilens, MD
Department of Psychiatry, Massachusetts General Hospital, Boston, MA, USA



J Clin Psychiatry. 2021 Feb 23;82(2):20m13598.

Retrospective cohort study:

SUD outpatients with comorbid ADHD on ADHD Meds show much better Tx retention than comorbid patients not on ADHD Meds.

... but do they have better outcomes??

Pharmacological Treatment of ADHD in Addicted Patients: What Does the Literature Tell Us?

Pieter-Jan Carpentier, MD, PhD, and Frances R. Levin, MD, PhD

Learning objectives: After participating in this activity, learners should be better able to:

- Evaluate pharmacologic treatment of attention deficit/hyperactivity disorder (ADHD) in patients with substance use disorder (SUD)
- Assess the causes of the diminished efficacy of ADHD medication in patients with comorbid SUD

Objective: Substance use disorder (SUD) and attention-deficit/hyperactivity disorder (ADHD) frequently co-occur, and the presence of ADHD complicates the treatment of the addiction. Pharmacotherapy is a potent intervention in childhood and adult ADHD, but findings have been mixed in adolescent and adult ADHD patients with SUDs. This review focuses on several contributing factors and possible explanations, with implications both for future research and for clinical practice.

Method: This systematic review examined all randomized, placebo-controlled trials of pharmacotherapy for ADHD in adult and adolescent SUD patients.

Results: The number of studies is limited, and several studies are hampered by qualitative flaws. The results, in general, are inconclusive for most medications studied, but more recent trials using psychostimulants in robust dosing have demonstrated significantly positive results.

Conclusion: In reviewing these trials, possible explanations relating to the particular characteristics and problems of this complex patient group are discussed. Several factors, including ADHD symptom severity, psychiatric comorbidity, persistent drug use, choice of medication, and concomitant psychosocial intervention, influence study results. Taking these factors into account may improve the likelihood of detecting significant effects in future research, as the recent positive trials have indicated, and may help in the appropriate selection of pharmacotherapy in clinical practice.

Keywords: adult, ADHD, atomoxetine, comorbidity, dexamphetamine, methylphenidate, pharmacotherapy, placebo, randomized controlled trial, substance use disorder

Tx with stimulants (e.g. 60-90 mg MPH) and other medications has very little/no effect on ADHD Sxx and no effect on substance use.

Aberrant reward processing network in ADHD + SUD

European Neuropsychopharmacology (2013) 23, 1714–1723

ELSEVIER

www.elsevier.com/locate/euroneuro

Low dopamine transporter occupancy by methylphenidate as a possible reason for reduced treatment effectiveness in ADHD patients with cocaine dependence

Cleo L. Crunelle[a,b,c,*], Wim van den Brink[a], Dick J. Veltman[a,d], Katelijne van Emmerik-van Oortmerssen[e], Geert Dom[c], Robert A. Schoevers[f], Jan Booij[b]

2013

Table 2 Baseline [^{123}I]FP-CIT BP_{ND} (baseline) and post-treatment [^{123}I]FP-CIT BP_{ND} (after MPH) in ADHD patients without cocaine dependence (ADHD) and with cocaine dependence (ADHD+COC). Means±standard deviations (SD) are presented. NS: not significant.

BP_{ND}	ADHD			ADHD+COC			Difference in effect (group × time)
	N=16			N=8			
	Baseline	Post-treatment	Treatment effect	Baseline	Post-treatment	Treatment effect	
CAUDATE nucleus (mean±SD)	3.6±0.6	2.0±0.5	$p<0.001$	2.7±0.4 *	1.7±0.4	$p<0.001$	$p=0.008$
Putamen (mean±SD)	4.2±0.9	2.7±0.5		3.2±0.6 *	2.2±0.5		$p=0.177$, NS
Thalamus (mean±SD)	0.6±0.2	0.5±0.3		0.5±0.2	0.3±0.2		$p=0.941$, NS
Midbrain (mean±SD)	0.4±0.3	0.4±0.2		0.4±0.2	0.3±0.2		$p=0.394$, NS

54 mg MPH-OROS p.o. → lower striatal DAT-occupancy in ADHD patients with compared to without cocaine dependence

Need for higher doses of stimulants in patients with ADHD+SUD/Cocaine?

Higher dosis of Stimulants in SUD + ADHD

Addiction

RESEARCH REPORT

doi:10.1111/add.12369

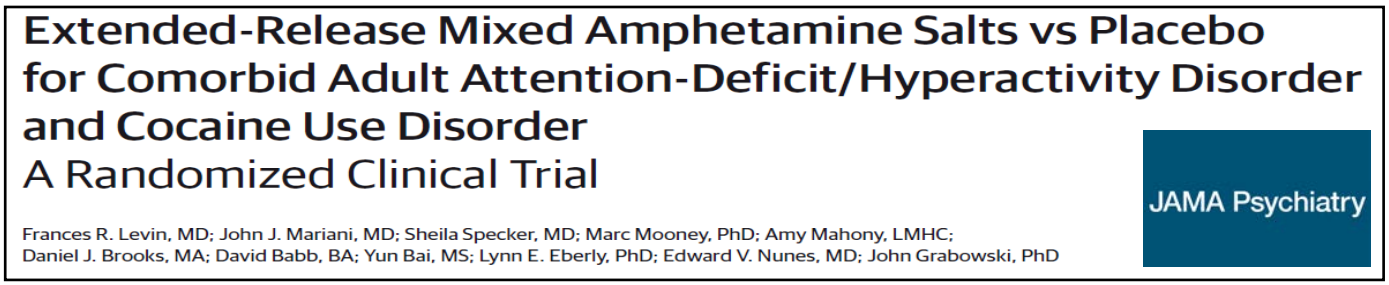
Methylphenidate for attention deficit hyperactivity disorder and drug relapse in criminal offenders with substance dependence: a 24-week randomized placebo-controlled trial

Maija Konstenius[1], Nitya Jayaram-Lindström[1], Joar Guterstam[1], Olof Beck[2], Björn Philips[3] & Johan Franck[1]

Division of Psychiatry, Department of Clinical Neuroscience, Karolinska Institutet, Stockholm, Sweden,[1] Division of Clinical Pharmacology, Department of Medicine, Karolinska Institutet, Stockholm, Sweden[2] and Department of Psychology, Linköping University, Linköping, Sweden[3]

MPH up to 180 mg/day

Addiction. 2014 Mar;109(3):440-9



Extended-Release Mixed Amphetamine Salts vs Placebo for Comorbid Adult Attention-Deficit/Hyperactivity Disorder and Cocaine Use Disorder

A Randomized Clinical Trial

JAMA Psychiatry

Frances R. Levin, MD; John J. Mariani, MD; Sheila Specker, MD; Marc Mooney, PhD; Amy Mahony, LMHC; Daniel J. Brooks, MA; David Babb, BA; Yun Bai, MS; Lynn E. Eberly, PhD; Edward V. Nunes, MD; John Grabowski, PhD

Amph Salts 60-80 mg/day

JAMA Psychiatry. 2015;72(6):593-602. doi:10.1001/jamapsychiatry.2015.41 Published online April 18, 2015.

Safety and tolerability of oral lisdexamfetamine in adults with methamphetamine dependence: a phase-2 dose-escalation study

BMJ Open

Nadine Ezard ,[1,2,3,4] Brendan Clifford,[1,5] Adrian Dunlop,[4,6,7] Raimondo Bruno,[8] Andrew Carr,[9] Zhixin Liu ,[9,10] Krista J Siefried ,[1,2,3] Nicholas Lintzeris[4,11,12]

Lisdexamph. up to 250 mg/day

BMJ Open. 2021 May 18;11(5):e044696.

Higher dosis of MPH in SUD + ADHD

Addiction

RESEARCH REPORT

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MPH vs Placebo: Changes in ADHD Sx

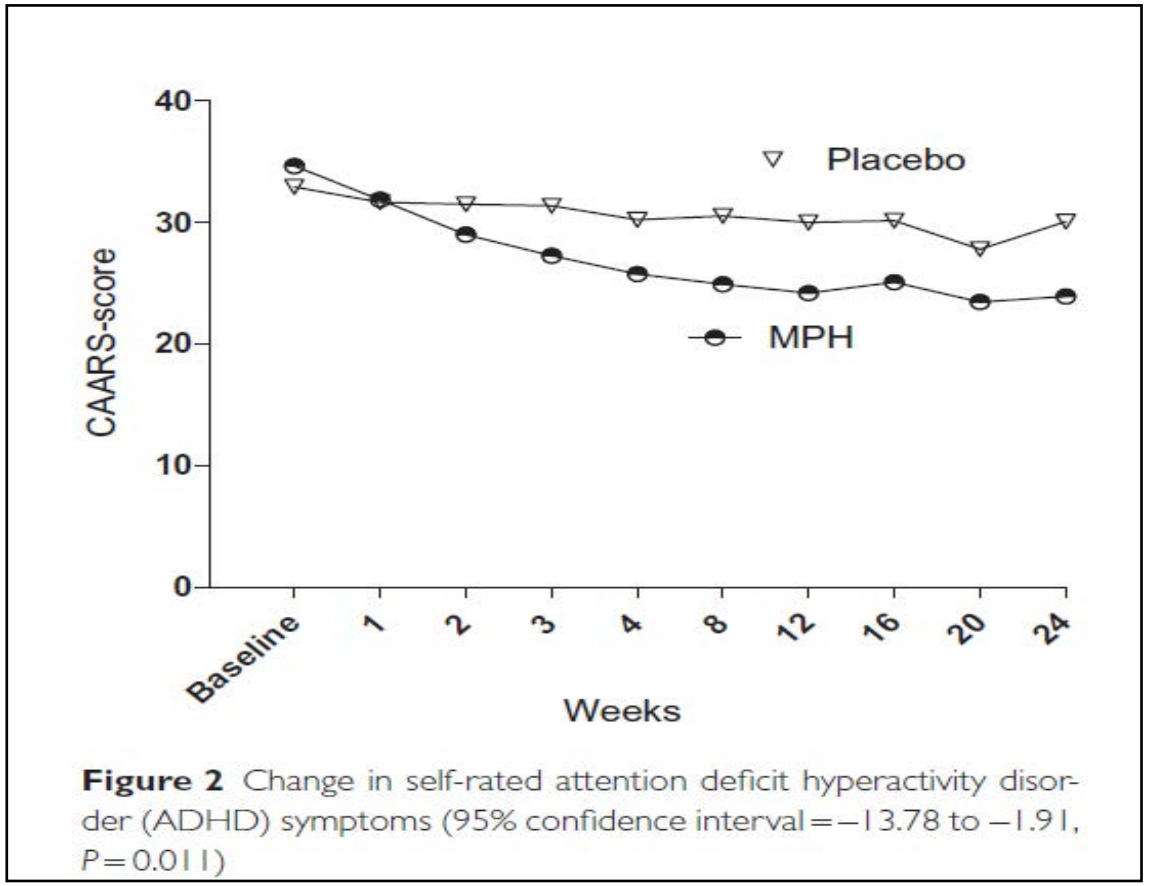


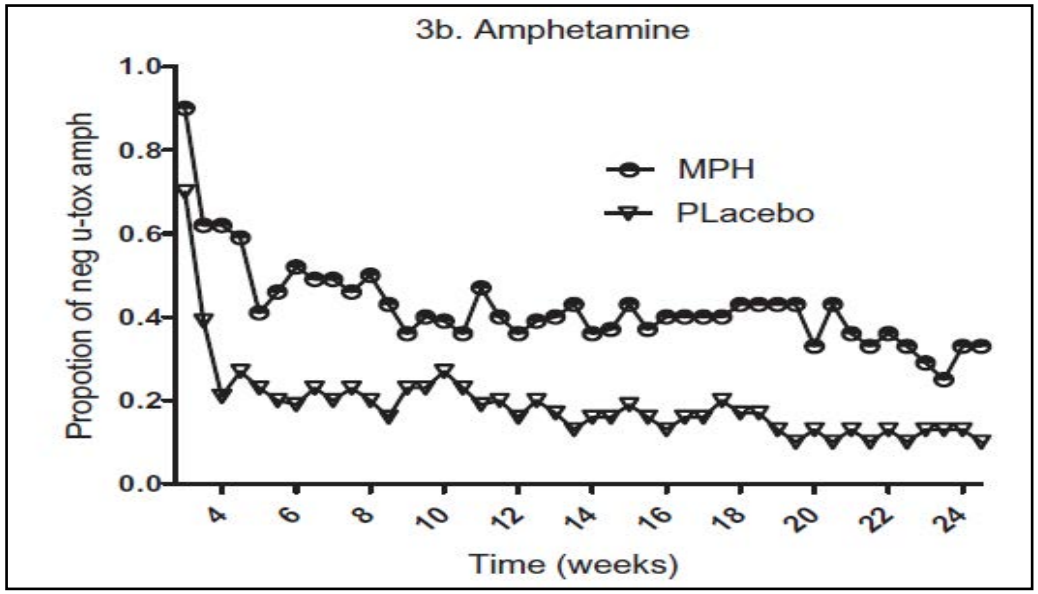
Figure 2 Change in self-rated attention deficit hyperactivity disorder (ADHD) symptoms (95% confidence interval = −13.78 to −1.91, $P = 0.011$)

Titration:
MPH-OROS: 63% 180 mg/day
11% 144 mg/day
7% 96 mg/day
19% no

ADHD responders (>30% Sx↓)
* Placebo + CBT = 27%
* MPH-OROS + CBT = 75%
(**NNT=2.1**; p=0.012)

∆ CGI
Placebo + CBT (p=0.688)
MPH-OROS +CBT (p=0.039)

MPH vs Placebo: Changes in drug use

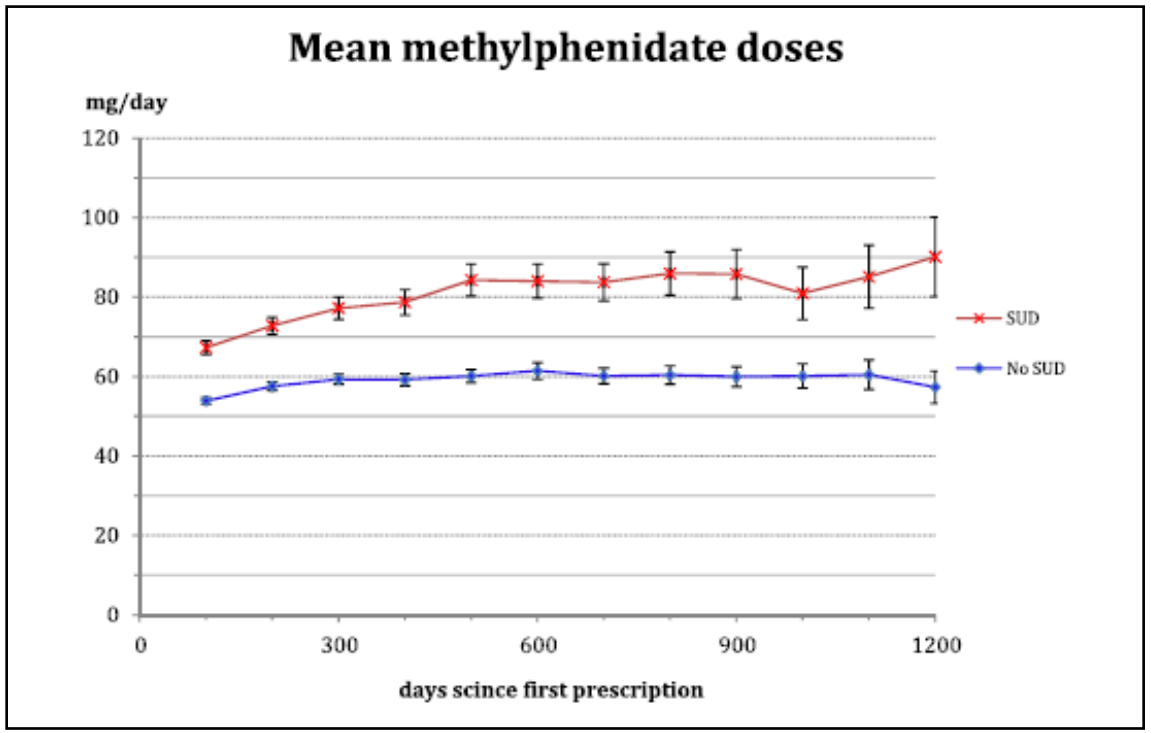


Placebo +CBT: 14% amph free urines
MPH-OROS + CBT: 23% amph free urines
(p=0.019)

Methylphenidate doses in Attention Deficit/ Hyperactivity Disorder and comorbid substance use disorders

Charlotte Skoglund[a,*], Lena Brandt[b], Brian D'Onofrio[d], Henrik Larsson[c], Johan Franck[a]

2017



Swedish Case Register

ADHD without SUD: N=9,444
ADHD with SUD: N=4,870

Follow-up 6.5 years

ADHD+SUD start with somewhat Higher dose, show some dose increase in 1st two years and then stabilize → titration & no tolerance

Robust doses mixed amphetamine salts in cocaine dependence + ADHD

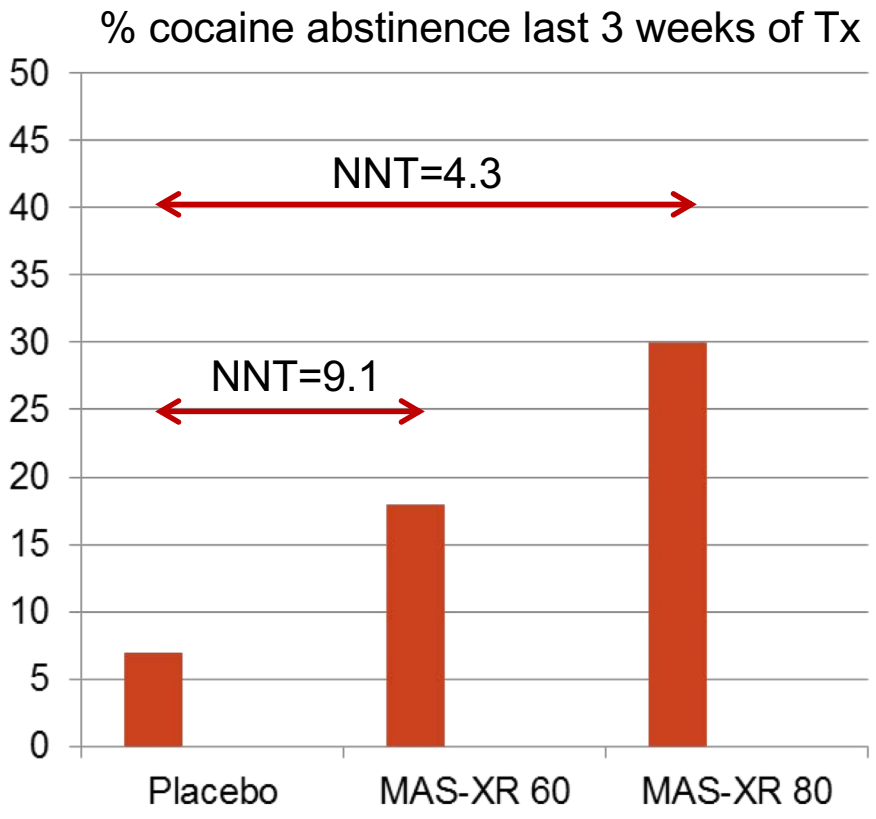
Extended-Release Mixed Amphetamine Salts vs Placebo for Comorbid Adult Attention-Deficit/Hyperactivity Disorder and Cocaine Use Disorder

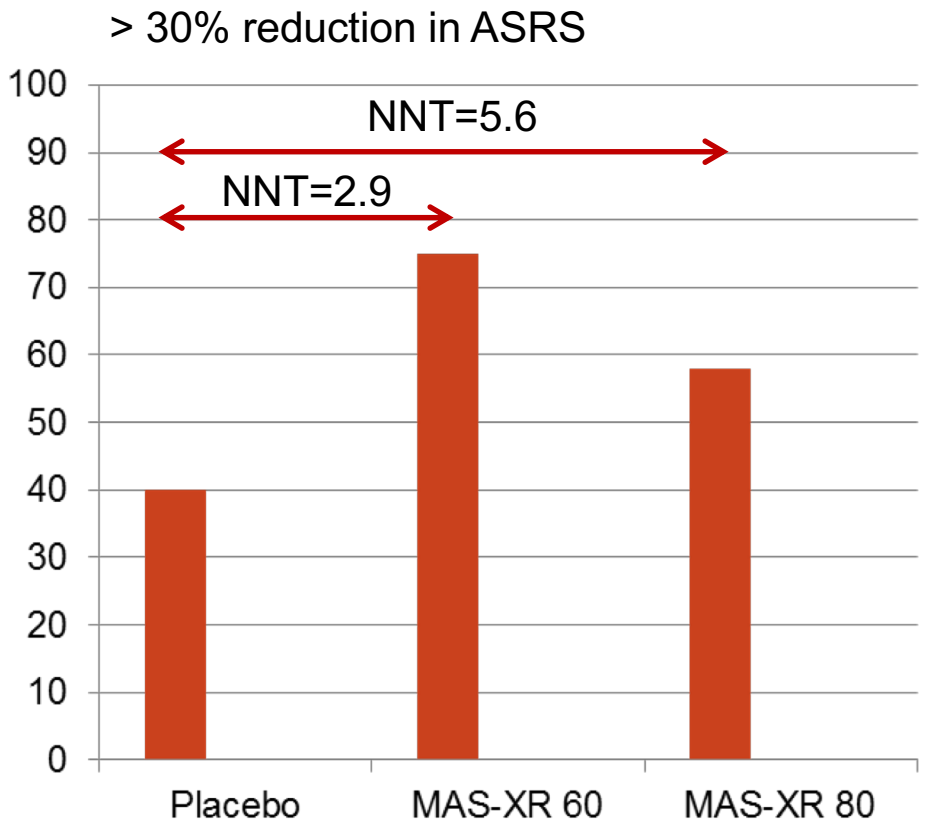
A Randomized Clinical Trial

Frances R. Levin, MD; John J. Mariani, MD; Sheila Specker, MD; Marc Mooney, PhD; Amy Mahony, LMHC; Daniel J. Brooks, MA; David Babb, BA; Yun Bai, MS; Lynn E. Eberly, PhD; Edward V. Nunes, MD; John Grabowski, PhD

JAMA Psychiatry. 2015 Jun;72(6):593-602

Effect of robust doses dexamphetamine XR





with permission of Frances Levin

Extended-Release Mixed Amphetamine Salts vs Placebo for Comorbid Adult Attention-Deficit/Hyperactivity Disorder and Cocaine Use Disorder
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How treatment improvement in ADHD and cocaine dependence are related to one another: A secondary analysis

Frances R. Levin[a,b,*], C. Jean Choi[e], Martina Pavlicova[c], John J. Mariani[a,b], Amy Mahony[a], Daniel J. Brooks[a], Edward V. Nunes[a,b], and John Grabowski[d]

Conclusion: When treating co-occurring ADHD and cocaine dependence with stimulant medication, abstinence is most likely preceded by improvement in ADHD, which tends to occur early with medication treatment. Other observed temporal patterns suggest the potential complexity of the relationship between ADHD and cocaine dependence.

Drug Alcohol Depend. 2018 Jul 1;188:135-140

Impulsiveness as a moderator of amphetamine treatment response for cocaine use disorder among ADHD patients

Derek Blevins, MD[1,2], C. Jean Choi, MS[3], Martina Pavlicova, Ph.D[4], Diana Martinez, MD[1,2], John J. Mariani, MD[1,2], John Grabowski, PhD[5], Frances R. Levin, MD[1,2]

Conclusions: The results show an association between higher within-group trait impulsiveness, as measured by the BIS-11, and response to MAS-ER for CUD in a cohort with comorbid ADHD. This result further demonstrates that impulsiveness is an important factor when considering treatment options for patients with CUD and that higher baseline impulsiveness may predict response to treatment with psychostimulants for CUD.

Drug Alcohol Depend. 2020 May 25;213:108082.

* Cocaine use reduction is more likely to be preceded by ADHD improvement than vice versa

* Higher levels of (BIS) impulsiveness are associated with better cocaine use outcomes

Safety and tolerability of oral lisdexamfetamine in adults with methamphetamine dependence: a phase-2 dose-escalation study

BMJ Open
2021

Nadine Ezard [1,2,3,4] Brendan Clifford,[1,5] Adrian Dunlop,[4,6,7] Raimondo Bruno,[8] Andrew Carr,[9] Zhixin Liu [9,10] Krista J Siefried [1,2,3] Nicholas Lintzeris[4,11,12]

Design:

* N=16 methamphetamine (MA) dependent patients (44% possible adult ADHD)
* *Single-blind ascending-descending dose study*:
  * 12 week study: week 1-4 (110→250mg), week 5-8 (250→110mg), week 9-12 (0mg)

Outcomes:

* Primary: safety, tolerability, (S)AE
* Secondary: change in MA use, craving, withdrawal

Main results:

* 14/16 (87.5%) complete escalation to 250mg/day (no drop-out due to adverse events)
* **Reduction in MA use in first 4 weeks: median 21 days → median 13 days (p=0.013)**

Review

European Addiction Research

Eur Addict Res 2018;24:43–51
DOI: 10.1159/000487767

Received: October 10, 2017
Accepted: February 18, 2018
Published online: March 6, 2018

International Consensus Statement on Screening, Diagnosis and Treatment of Substance Use Disorder Patients with Comorbid Attention Deficit/Hyperactivity Disorder

Cleo L. Crunelle[a, b] Wim van den Brink[c] Franz Moggi[d]
Maija Konstenius[e] Johan Franck[e] Frances R. Levin[f] Geurt van de Glind[g]
Zsolt Demetrovics[h] Corné Coetzee[i] Mathias Luderer[j] Arnt Schellekens[k]
Frieda Matthys[a] ICASA consensus group

Brynte *et al. BMC Psychiatry* (2022) 22:625
https://doi.org/10.1186/s12888-022-04259-6

BMC Psychiatry

RESEARCH Open Access

The clinical course of comorbid substance use disorder and attention deficit/hyperactivity disorder: protocol and clinical characteristics of the INCAS study

Christoffer Brynte[1*], Myriam Aeschlimann[2], Csaba Barta[3], Alex Hendikus Abraham Begeman[4], Amanda Bäcker[1], Cleo Lina Crunelle[5], Constanza Daigre[6,7,8,9], Laura De Fuentes-Merillas[10], Zsolt Demetrovics[11,12], Geert Dom[13,14], Lara Grau López[6,7,8,9], Romain Icick[15], Brian Johnson[16], Peter Joostens[17], Máté Kapitány-Fövény[18,19], Emily Karsinti[20], Falk Kiefer[21,22], Maija Konstenius[1], Frances R. Levin[23,24], Mathias Luderer[25], Wiebren Markus[26], Frieda Matthys[5], Franz Moggi[27], Raul Felipe Palma-Alvarez[6,7,8,9], Maria Paraskevopoulou[28], J. Antoni Ramos-Quiroga[6,7,8,9], Arnt Schellekens[28,29], Leila M. Soravia[27,30], Norman Therribout[20], Anil Thomas[31,32], Geurt van de Glind[33], Michiel Willem van Kernebeek[5], Sabine Vollstädt-Klein[21,22], Florence Vorspan[15], Wim van den Brink[34] and Johan Franck[1]

How much of our consensus statement is already reality?

How much of our consensus statement is already reality?

Table 3 Current and previous treatment for ADHD and SUD

Previously received ADHD treatment	
Yes	56.9%
No	40.8%
Unknown	2.3%
Received pharmacological ADHD treatment before 18 years old	
Yes	21.3%
No	51.0%
Unknown	27.6%
Currently receives ADHD treatment (at inclusion)	
Pharmacological	34.9%
Psychological	20.9%
No	60.7%
Unknown	1.7%
Previously received SUD treatment	
Yes	71.1%
No	24.0%
Unknown	4.9%

US: 70%, mainly atomoxetine
Europe: 20%, mainly stimulants

Addiction and ADHD: Conclusions

Conclusions ADHD-SUD Comorbidity

- ADHD and SUD frequently co-occur (10-25% of treatment seeking SUD patients have adult ADHD)
- Childhood ADHD is a predictor of SUD independent of ODD/CD
- Early stimulant treatment of ADHD can prevent the development of SUD
- Common genetic network underlies comorbidity ADHD with SUD (Acros-Burgos, 2012)
- Neurobiology ADHD and SUD is similar (high reward sensitivity, deficient cognitive control) with differences in self-reported impulsivity and reward processing predicting SUD
- ADHD patients with SUD need different Tx (higher doses long-acting stimulants+abuse prevention)

Thank you for your attention!

ICASA
INTERNATIONAL COLLABORATION ON ADHD AND SUBSTANCE ABUSE

w.vandenbrink@amc.uva.nl