

McDonaldisation of Children's mental health

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Cultural and political drivers



Growing up is dialectically unfolding process full of uncertainty.

It has been corrupted by:

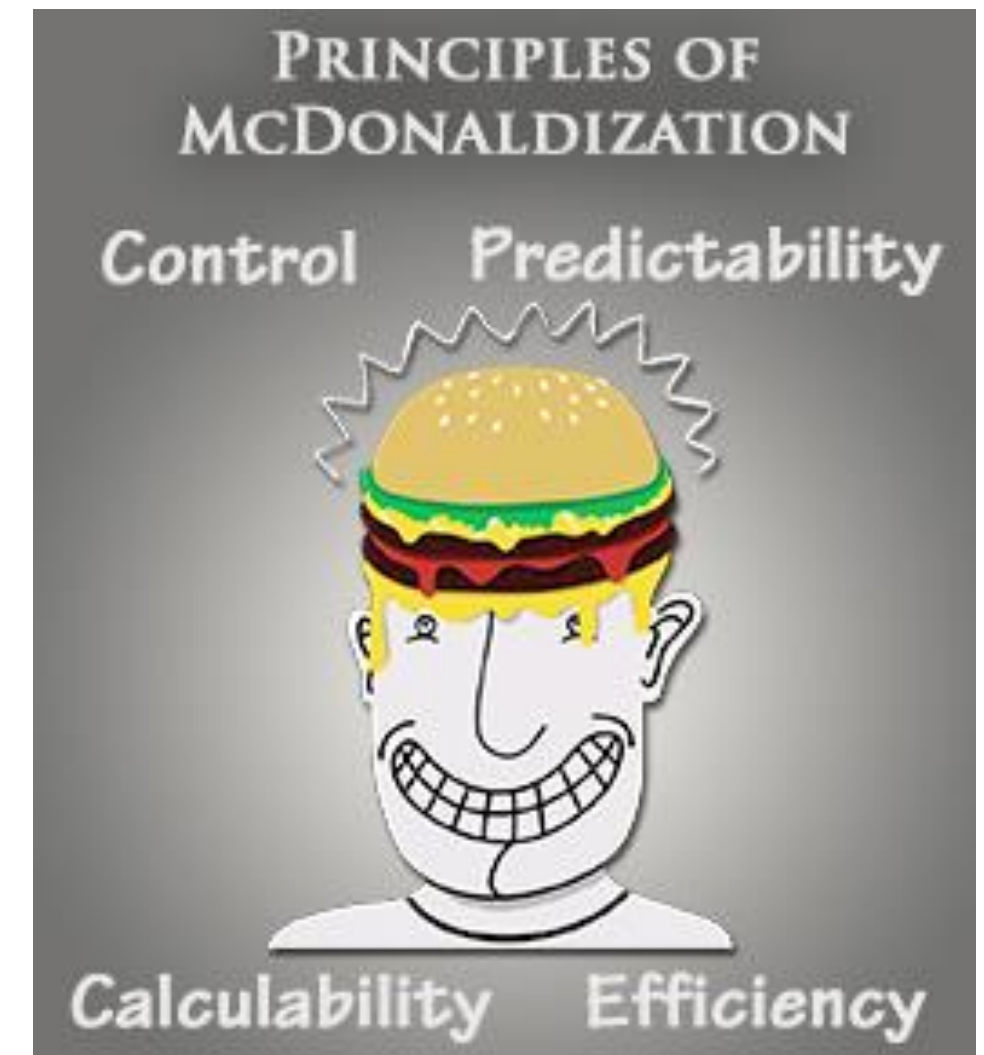
- Scientism
- individualism
- Commodification.

Epidemics of Mental illness in the young?

- Individualisation serves capitalism.
- Performance based culture leads to winners and losers.
- Construct 'losers' as 'vulnerable' to sooth the systemic guilt.
- Epidemics of mental disorders is a social construct that lets the neoliberal capitalists off the hook!
- The problems are individual deviations that need 'expert' knowledge to identify and fix in the individual.
- Intolerance of suffering combines with introspection. Methods to express 'distress/dissatisfaction' e.g. self-harm, results in cultural 'looping'.

McDonaldisation

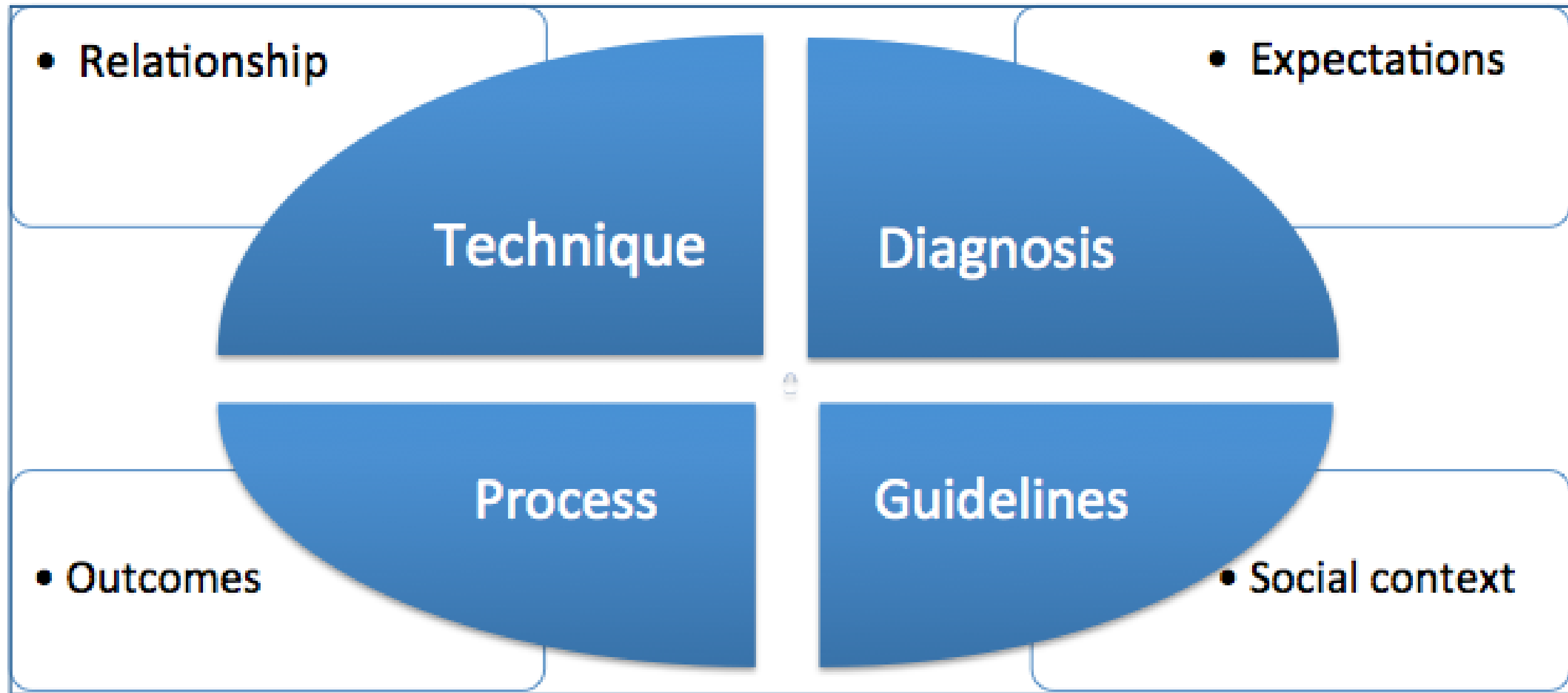
- ‘Branding’ exploits fears and desires.
- Psychiatric diagnoses are brands.
- Technical interventions such as CBT and ‘anti-depressants’ sell quick and efficient remedies.
- Evidence shows little ‘extra value’ for any Mental Health brand.



In psychiatry there are no diagnoses

- Diagnoses in psychiatry cannot explain (except dementias).
- Consider the question ‘What is ADHD?’ and compare with the question ‘What is diabetes?’
- Consider what happens when we argue that ‘ADHD *causes* poor hyperactivity and inattention’.
- In psychiatry we have **classification** (that is descriptive) not diagnosis.
- Ecosystems use multiple classifications – more appropriate for context-rich and dynamic open systems.

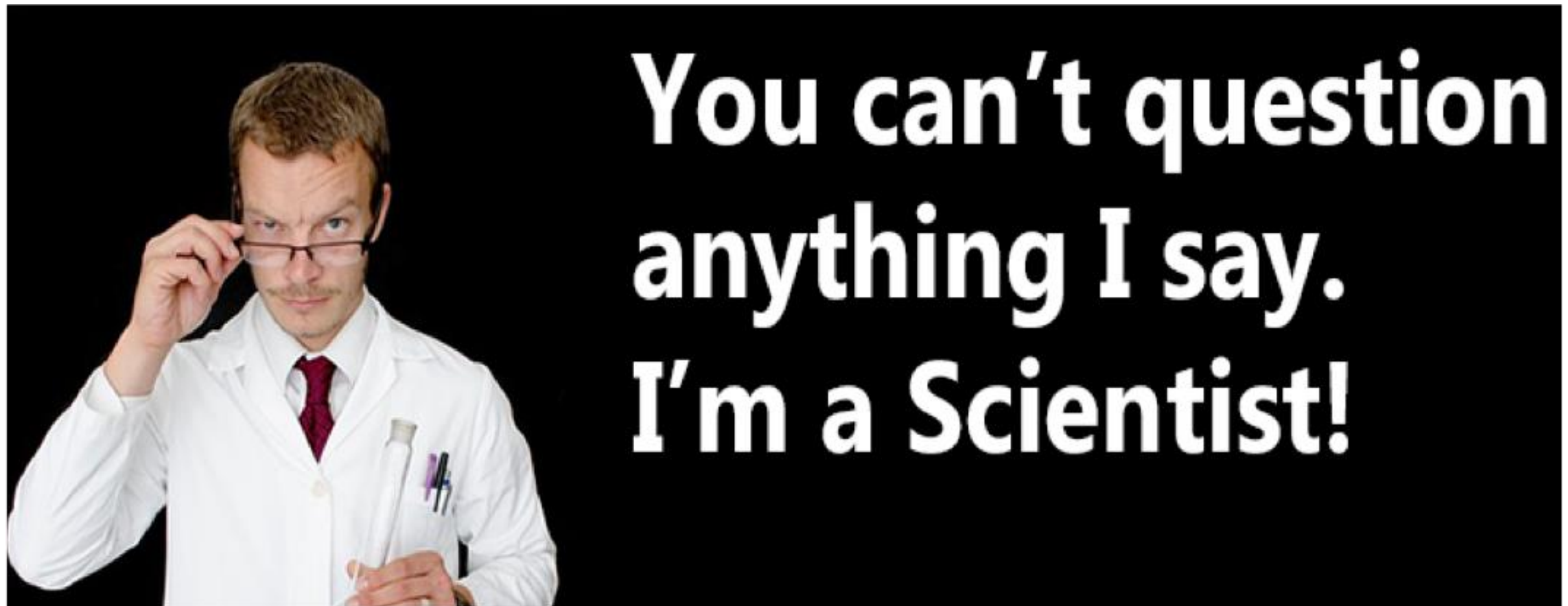
Diagnostic/technical Paradigm



ADHD symptoms

- *Often* fails to give close attention to details or makes careless mistakes in schoolwork, work, or other activities.
- *Often* does not seem to listen when spoken to directly.
- *Often* fidgets with hands or feet or squirms in seat.
- *Often* runs about or climbs excessively in situations in which it is inappropriate.
- *Often* has difficulty awaiting turn.
- *Often* interrupts or intrudes on others.

The spread of 'Scientism'



ADHD is genetic and neurodevelopmental



This is really exciting because it gives us the first direct genetic link to ADHD. Now we can say with confidence that ADHD is a genetic disease and that the brains of children with this condition develop differently to those of other children," she said.

What did they really find?

Found 13.9% with ADHD have deleted or replicated genes (Copy Number Variants - CNVs), as do 7.4% controls (leaves 6.5%).

36% of those with IQ below 70 had these CNVs.

Once those with IQ below 70 excluded 11.4% have CNVs (leaves 4%).

Average IQ in ADHD group 86 (leaves ?0%).

If have CNVs 1 in 25 chance of ADHD.

Conclusion: strong association between low IQ and increased CNVs. Probably none with ADHD per se.

Elevated risk of increased CNVs associated with, autism, schizophrenia, ADHD, bi-polar (not depression/anxiety) mainly accounted for by IQ.

ADHD Brains are ‘abnormal’

“The results from our study confirm that people with ADHD have differences in their brain structure and therefore suggest that ADHD is a disorder of the brain,” lead author Martine Hoogman, Ph.D., said in a news release

The Telegraph

“ADHD is a brain disorder, not a label for poor parenting, say scientists”

February 16, 2017
by Henry Bodkin

Newsweek

“Study Finds Brains of ADHD Sufferers Are Smaller”

February 16, 2017,
by Conor Gaffey

WebMD

“Imaging Study Confirms Brain Differences in People With ADHD”

February 15, 2017, by
Robert Preidt

CNN

“Brains of those with ADHD show smaller structures related to emotion”

February 15, 2017, by
Susan Scutti

What did they really find?

MRI brain-scan data 3,242 participants from 23 different sites.

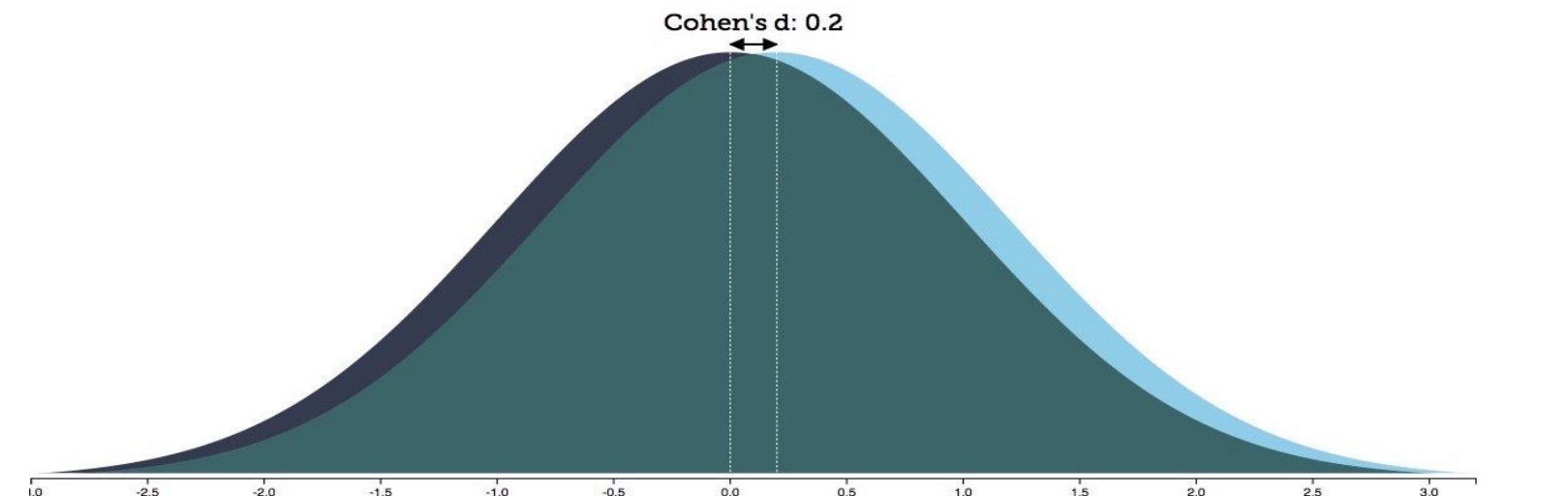
Some brain structure pooled data revealed on smaller average sizes from $d=0.19$ to $d=0.1$ (tiny).

Findings varied more between sites than within sites.

Once IQ controlled for significant differences disappear.

Medication use not properly accounted

Conclusion: No consistent finding.



Site	ADHD Cohort	Controls
Wuzberg, Germany	462.3	448.9
Dublin, Ireland	712.9	717.5
Baltimore, USA	688.1	719.9
New York, USA	721.6	749.0
Peking, China	692.0	687.0
Oregon, USA	703.1	720.6
Aachen, Germany	646.7	700.4
Bergen, Norway(1)	689.7	715.1
Bergen, Norway(2)	757.8	805.1
London, UK	486.4	486.2
Nijmegen, Netherlands	585.4	584.0
New York, USA	704.8	718.1
Utrecht, USA	600.9	608.9
New York, USA	704.8	718.1
Barcelona, Spain	652.9	692.9
Mannheim, Germany	721.0	653.5
London, UK	755.4	746.0
Amsterdam, Netherlands	686.1	683.0
Nijmegen, Netherlands	640.6	640.3
NIH	647.6	643.6
MTA	612.8	629.3

■ Larger ADHD accumbens volume
■ Smaller ADHD accumbens volume
■ Equal volumes (difference of less than 5.0 mm3)

At 10 of 21 sites, the ADHD cohort had equal or larger accumbens than the controls. Yet, the investigators reported, in their conclusion, that the MRI scans showed that *individuals* with ADHD had a smaller accumbens than "healthy" controls.

Heterogeneity in Autism

- **Poor social reading:** Language disorders, ADHD, anxiety, depression, personality disorder, attachment disorders, bipolar affective disorder, psychosis, learning difficulties, a sense of alienation, cultural difference, normal variation, deliberate provocation, boredom, a strong desire for attention, love etc.
- **Restricted interests:** OCD, obsessional personality disorder, schizoid personality disorder, ADHD (such as with computer games), depression (morbid preoccupation with negative aspects), eating disorders (fixation with food and/or weight), most men (e.g. with football), sportsmen and women, and humanity: If someone had not fixated long enough on the tendency of logs to roll down slopes, the wheel may never have been invented!
- “Each person with autism has a different combination of symptoms, and the combination may change over that person’s lifetime” Hughes (2012)

Creating the spectrum



Lorna Wing: in 1981, describes 6 cases with little in common with Asperger's classic four cases beyond sharing a lack of social reciprocity.



Michael Rutter Defined the 'triad' and argued that autism and its spectrum is beyond doubt genetic in origin

Disintegrating concept



Happé (2006): suggests that it is time to give up on the search for a monolithic cause or explanation for the three core aspects of autism, at the genetic, neural and cognitive levels

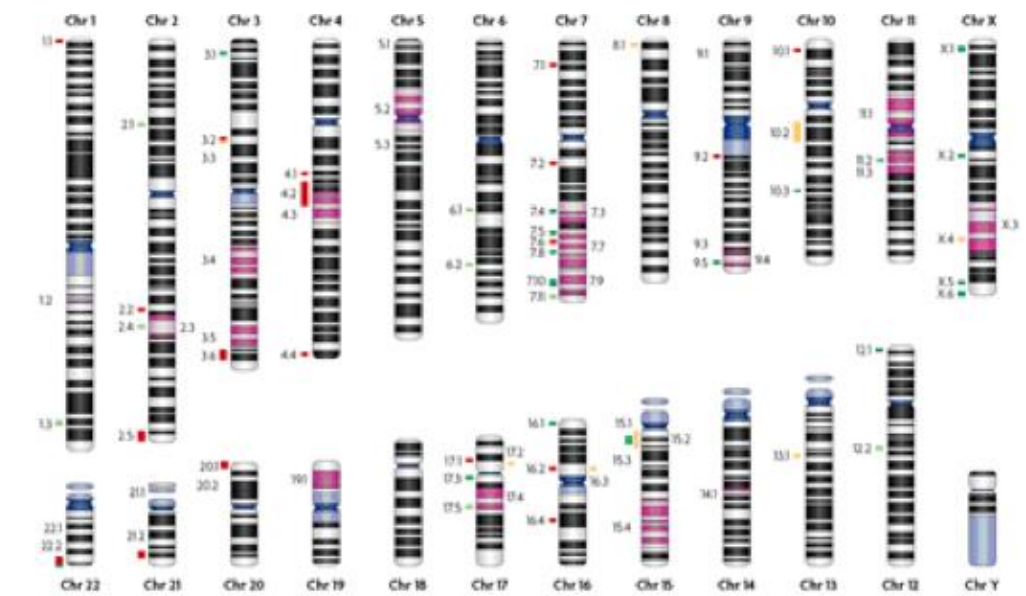


Baron-Cohen: (1997):
develops the empathising-systematising theory: Autism is extreme of systematising 'drive'.

Disintegrated concept

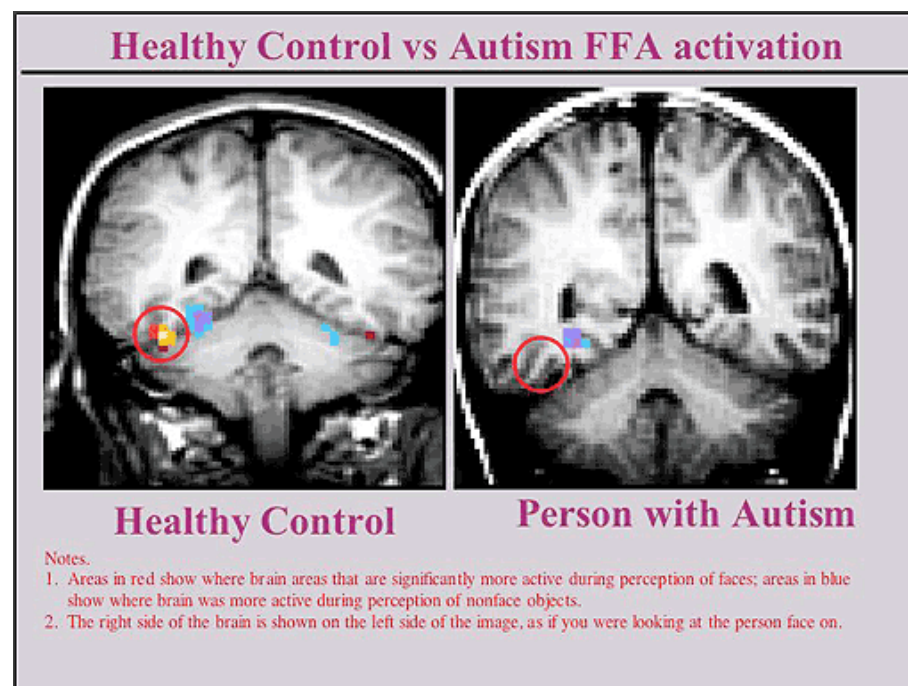
- **Waterhouse, London, Gillberg (2016):** “ASD should be disbanded in research because it lacks validity”.
- **Muller and Amaral (2017)** “Replication rates in ASD neuroimaging research have been unacceptably low ... the field has been outstandingly productive in generating thousands and thousands of findings reaching statistical significance in one or the other cohort, but disappointingly incapable of creating a coherent picture of neurobiological features underlying ASD”.
- **Al-jawahiri and Milne (2017)** “This heterogeneity is potentially a major factor impacting on the rate of replication of ASD studies, and is leading some researchers to give up on a single explanation for autism and others to propose the possibility that ASD should not be considered as a single disorder .. Instead, they suggest that within ASD, there could be groups of distinct disorders with many aetiologies”.

Genetics and autism

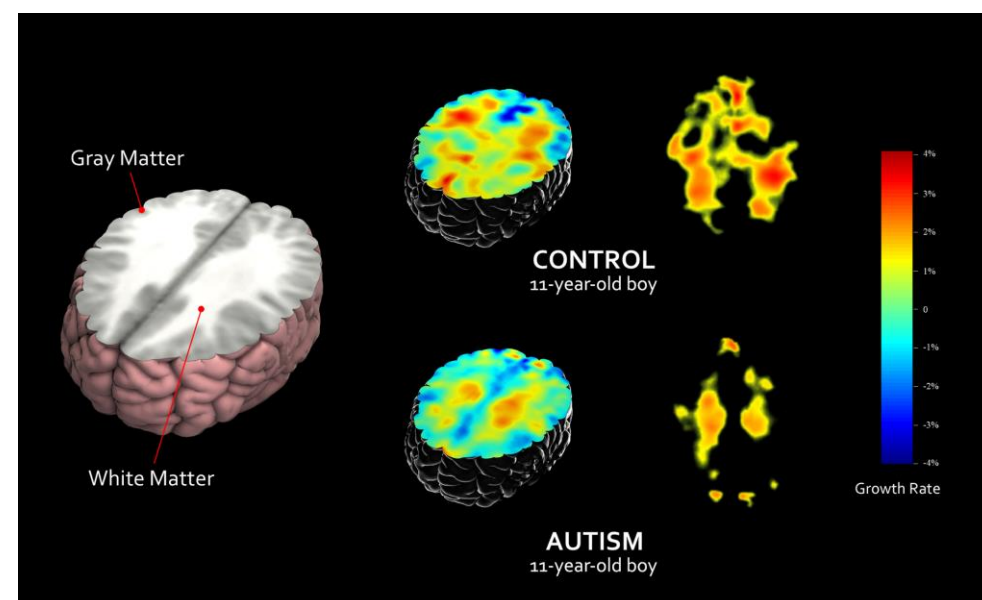


- Assumed high heritability based on twin studies.
- “Many research teams have searched for genes that may be involved. They haven’t turned up any prime candidates yet, only dozens, maybe hundreds of bit players” (Hughes, 2012)
- “First Genome Wide Association Studies (GWAS) on autism implicated two regions with mild effects.. subsequent GWAS failed to turn up any parts of the genome with statistical significance” (Williams, 2012)
- “With the advent of next generation sequencing techniques, the number of genes found that are associated with ASD is increasing to over 800 genes; consequently, it is becoming even more challenging to find unified explanations and functional associations between the genes involved.” (Al-jawahiri and Milne, 2017)

Neuroimaging in autism



- Lack of consistently replicated findings. e.g. studies documented an increase in cerebellar volume, smaller than average, and no significant differences. sample heterogeneity (such differences reflecting IQ differences) regular problem.
- Technical challenges: Brain connectivity theory replicated by head movement.
- “Until its biological basis is found, any attempts to use brain imaging to diagnose autism will be futile” Lange (2012)
- “A Bug in fMRI Software Could Invalidate 15 Years of Brain Research” (2016).



Debate in March 2017

- Person debating with me put forward 3 papers, summarising the evidence on the 'biological basis' for ASD.
- Chen et al (2015) Limbic and Cerebellum.
- Ecker et al (2015) Frontal, Temporal lobes and Cerebral Cortex.
- Fakhoury (2015) Balance of excitatory and inhibitory synapses.
- No attempt to control for or even mention of ID as a confound.

Clinical utility?

“I just knew, that the minute I said – “He is autistic” – this would be the only thing in people’s minds. They wouldn’t be interested in anything else about him, and their heads would be full of the paperwork they would need to complete and applications for funding etc., as well as lots of negative associations with the term ‘autism’. My son, as a person, would just disappear... I think the autism label offers what the social theorist Lauren Berland calls cruel optimism.”

Katherine Runswick-Cole (2017)

“I was suddenly pounced upon by the Head and deputy Head, who shooed me into their office to discuss my son’s SEN, while the rest of the parents continued the tour. I could tell how anxious they were that a problem child might be landing in their midst. Again, it was horrible. Most of these negative reactions were from people who had never met my son or read anything about him... I think what I object to was being made to feel that my son’s autism was some sort of a separate entity both to him and from us, his parents” Rebecca Wood (2017)

Prognosis



- No improvement in long term outcomes.
- Big increase in numbers on long term disability due to 'mental illness'.
- Many diagnoses conceived as life-long chronic disorders.
- Stigma increased by 'illness like any other illness' model.
- 'Internal stigma' associated with worse outcomes.

More long term patients

- US: Supplemental Security Income (SSI) or Social Security Disability Insurance (SSDI) increased and – from 1 in 184 in 1987 to 1 in 67 in 2007.
- For US children – a 35 fold increase in two decades.
- US: Mental illness is now the leading cause of disability in children, well ahead of physical disabilities like cerebral palsy or Down syndrome, for which the federal programs were created.
- UK: Mental illness became the leading reason for sickness and disability benefits in 2011. Increase 103% from 1995 – 2014. Other conditions fall by 35%.

Real life service outcomes

- 50-70% or more can recover or significantly improve according to research.
- 75% entering community MH treatments in US no improvement (Lambert, 2010).
- No improvement in population MH in Australia last 2 decades— poor ‘Mental Health knowledge’ a protective factor! (Goldney et al, 2010).
- 15% entering services achieve recovery (UK Centre for Social Justice, 2012).
- Attending CAMHS no improvement (Weiss et al, 1999; 2000), Non attenders do better than CAMHS long-term attenders (Jorg et al, 2012), CAMHS UK recovery rate of 18% (Wolpert, 2015).
- Psycho-education for depression in schools increased likelihood of depressive thoughts (stallard et al, 2012).
- 24% entering community CAMHS get worse (Warren et al, 2009).

The Social construction of ADHD

- Differences between mothers, fathers, teachers, peers.
- Highest stimulant prescription **middle class and white in US**, **working class in UK**. UK children associate with **anger**, US children with **performance**. Outcomes in US studies more likely to focus on academic outcomes, UK/Europe more likely to focus on behavioural outcomes (e.g. conduct symptoms).
- ADD in a school in Mexico.
- **Caucasian v African American** “He was just born with a chemical imbalance or something like similar to the components of a car” v “Yes, he’s a little wild at times. But the average boy is. That’s just a boy. It even says it in the bible: ‘Foolishness is tied up in the heart of a boy’.”



Surveillance and discipline

- Systems for ‘discipline’ and regulating behaviour.
- Move from external to internal, surface, visual, ‘adultified’.
- Increasing levels of surveillance, ‘performance’ anxiety, focus on consumerism spurring sense of inadequacy, ‘self monitoring’: growth of ‘identity’ politics.
- Boys emotional well-being becomes obscured.



"The blood tests confirm what we thought: he has ALBD - Annoying Little Bastard Disorder."

Key findings from outcome research:

Context and relationships

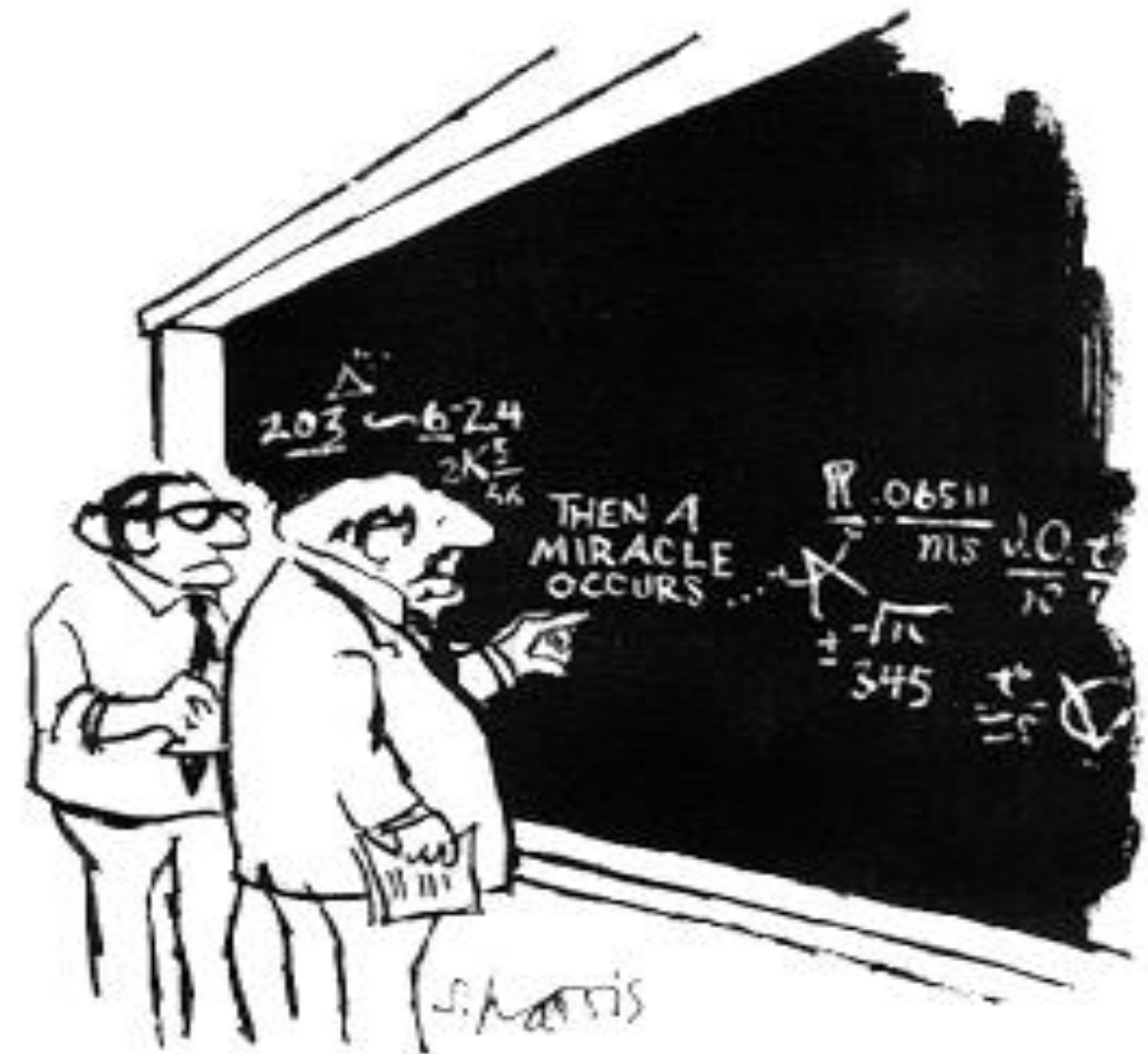
- Research finds therapy is effective for mental health problems
- Model or technique has a minimal impact on outcomes. BUT some models (that lead to expert/technical dependency) are worse than others.
- **Extra-therapeutic factors** such as social circumstances and motivation and expectation have biggest impact on outcomes.
- **Quality of therapeutic alliance** important.
- Regular monitoring of progress and alliance may improve outcomes.

Treatment with Stimulants

Systematic Reviews: inadequate reporting of study methodology, possible publication bias, limited reliability of results, inadequate data regarding adverse events, and lack of RCT evidence of a long term benefit from taking stimulants.

Adverse events include: loss of appetite, insomnia, low mood, violence and aggression, psychosis, perseveration.

Long term: largely naturalistic. US, UK, Australia, Canada. No significant difference in outcomes between medicated and not medicated. Some worse outcomes on medication in some studies (school achievement, ADHD symptoms, blood pressure). Some better outcomes educationally in some studies but claims distorted by conflict of interest problems. Stimulants should be used with caution with children until we have better evidence.



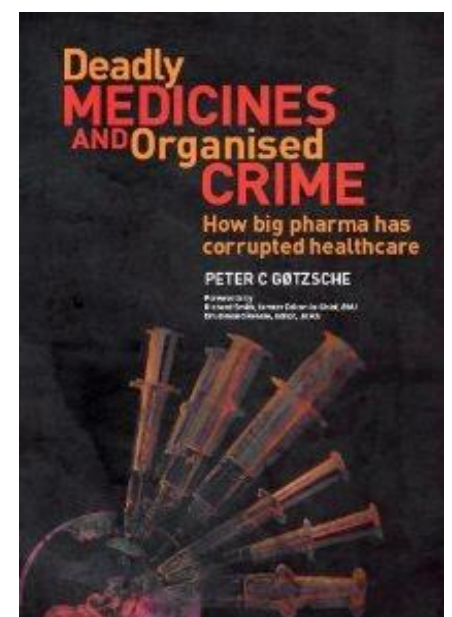
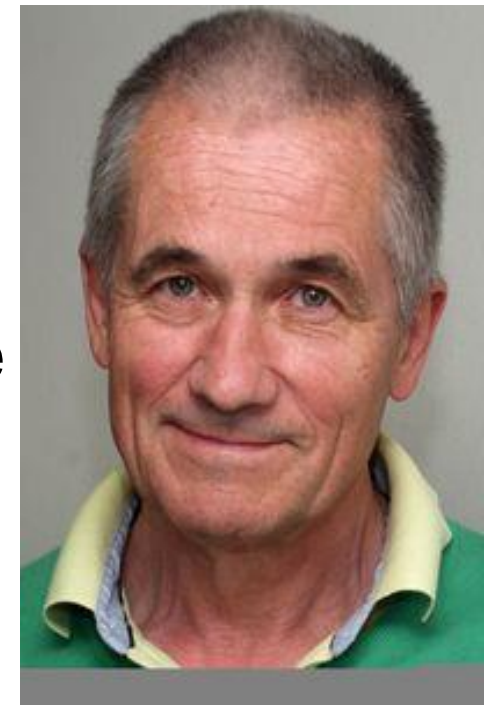
"I think you should be more explicit here in step two."

Mainstream medicine questions



Marcia Angell, former chief editor at New England Journal of Medicine and a Harvard professor, in book review series entitled “*The illusions of Psychiatry*” launches a serious attack on the orientation and practice of modern psychiatry in a series of book reviews in 2011

Peter Gotzsche cofounder of The Cochrane Collaboration head of The Nordic Cochrane Centre,
“Our citizens would be far better off if we removed all the psychotropic drugs from the market, as doctors are unable to handle them. It is inescapable that their availability creates more harm than good.” (2013)



Psychiatrists re-appraise

- **Francis Allen (2013)**, lead editor of DSM IV: DSM IV had many errors leading to “false epidemics” of ‘bipolar, ADHD and Autism’ diagnoses.
- **Thomas Insel (2013)** “The weakness is its lack of validity” (DSM)
- **David Kupfer (2013)** “We've been telling patients for several decades that we are waiting for biomarkers. We're still waiting.”
- **Sir Robin Murray (2016)** “it has become obvious that child abuse, urbanization, migration, and adverse life events contribute to the aetiology of schizophrenia and other psychoses... in treating schizophrenia with antipsychotics, we sometimes compound ...by causing secondary dopamine supersensitivity.”
- **Leon Eisenberg (2012)** ADHD is a “fake disorder” and child psychiatrists need to become much more adept at evaluating patient’s psychosocial circumstances.
- **Chris Gillberg (2016)** “Autism is not a valid diagnosis and should not be used in research”

Beyond diagnosis

- Don't pay too much attention to it.
- Deconstruct - 'descriptions not explanations'.
- Privilege relational/contextual (model determined by this not diagnosis)
- Monitor outcomes.
- Use alternative concepts: 'intense child', 'there are thousands of ADHDs out there – tell me about yours', 'they are developmental because people grow out of them', framework of 'we all have different skills', 'if we found the perfect diagnosis, what would be different?'.
- Prescribe with enabling frameworks (e.g. medicine cannot make decisions).

Team	Open Effect size	Open Improved/ Recovered	Open No change	Open Deteriorated	Closed Effect size	Closed Improved/ Recovered	Closed No change	Closed Deteriorated
T2	0.7	59%	30%	10%	1.1	76%	17%	7%
N=	463	174	90	32	690	408	91	37
Boston	0.6	60%	22%	18%	0.9	75%	16%	8%
N=	167	57	21	17	343	210	45	23
SW	0.7	65%	23%	12%	1.1	76%	16%	8%
N=	446	171	62	31	1047	666	144	69
North	0.7	67%	23%	9%	1.3	73%	20%	6%
N=	915	403	139	54	1104	579	164	47

OO-CAMHS

Table 2. Proportions of CYP showing reliable change with n in parentheses.

RCADS	Social phobia		Panic		Depression		GAD		Separation anxiety		OCD	
	Child REPORT	Parent REPORT	Child REPORT	Parent REPORT	Child REPORT	Parent REPORT	Child REPORT	Parent REPORT	Child REPORT	Parent REPORT	Child REPORT	Parent REPORT
Improvement	26% (94)	20% (26)	25% (90)	13% (16)	31% (123)	8% (10)	28% (100)	13% (17)	10% (36)	6% (8)	16% (57)	8% (10)
No change	70% (249)	70% (90)	68% (243)	81% (99)	64% (249)	92% (120)	67% (237)	78% (104)	87% (304)	89% (116)	82% (292)	89% (114)
Deterioration	4% (13)	9% (12)	7% (25)	7% (8)	5% (20)	1% (1)	5% (16)	9% (12)	2% (8)	5% (6)	2% (6)	3% (4)
N	356	128	358	123	392	131	353	133	348	130	355	128

CYP-IAPT

Thanks

<http://www.outcomeorientated.com>

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