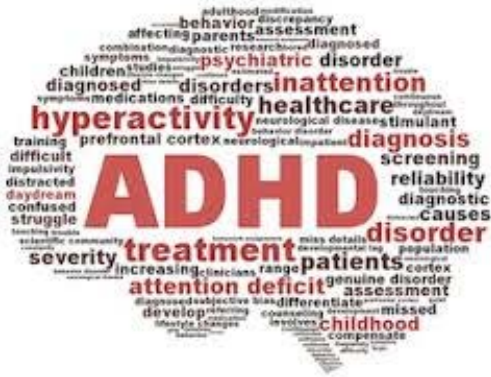




ADHD

Today....



What is ADHD?

Characteristics/Symptoms

Classification/Diagnosis

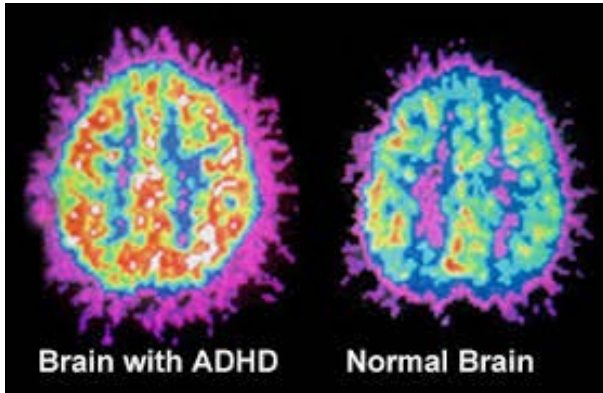
Prevalence

Aetiology – Genetics and Environmental risks

Executive functions and ADHD

Impact on Life and Schooling

What is ADHD?



It is a
neurological
problem!

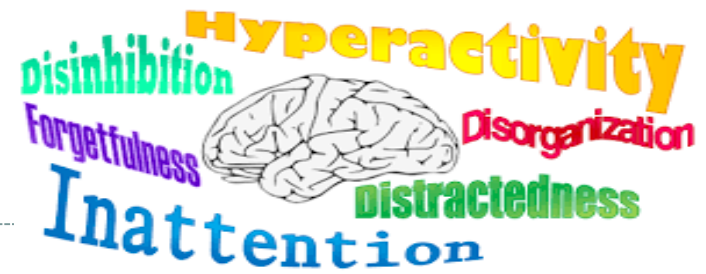
It's a problem
in the area of
the brain that
allows us to
tune out of what
we shouldn't be
focussing on.

It's also the part
of the brain that
controls our
impulses and
social behaviour

In many
children with
ADHD this part
of the brain has
not fully
developed.



Attention-deficit/hyperactivity disorder involves...



ADHD is characterized by a pattern of behaviour, present in multiple settings (e.g., school and home), that can result in performance issues in social, home, or educational settings.



Two categories ...

- **inattention** and **hyperactivity and impulsivity** that include behaviours like failure to pay close attention to details, difficulty organizing tasks and activities, excessive talking, fidgeting, or an inability to remain seated in appropriate situations.

ADHD Classification



International
Classification
of Mental and
Behavioural
Disorders
10th revision
(ICD-10)

Attention-deficit
hyperactivity
disorder,
unspecified type

...originating in childhood in which the essential features are signs of developmentally inappropriate inattention, impulsivity, and hyperactivity. At least some of the symptoms must be present before the age of 7 years.

Diagnostic
and
Statistical
Manual of
Mental
Disorders –
5th Edition
(DSM-5)

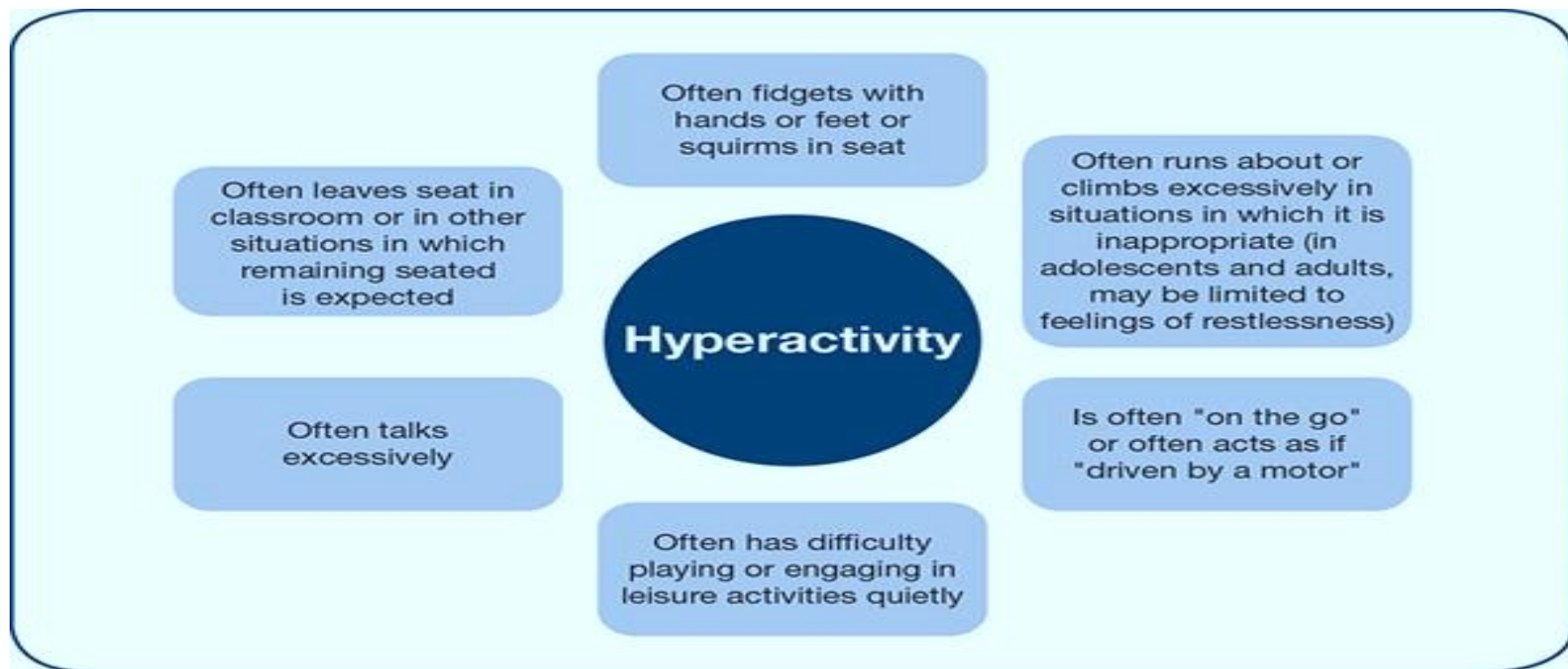
attention-deficit
hyperactivity
disorder
(ADHD)

"a persistent pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development"

<http://www.adhd-institute.com/assessment-diagnosis/diagnosis/#sthash.tCbZmDnY.dpuf>



Figure created from information in the DSM-5TM and ICD-10 classifications



Impulsivity

Often blurts out answers before questions have been completed

Often has difficulty awaiting turn

Often interrupts or intrudes on others



PresenterMedia

Presentation of ADHD

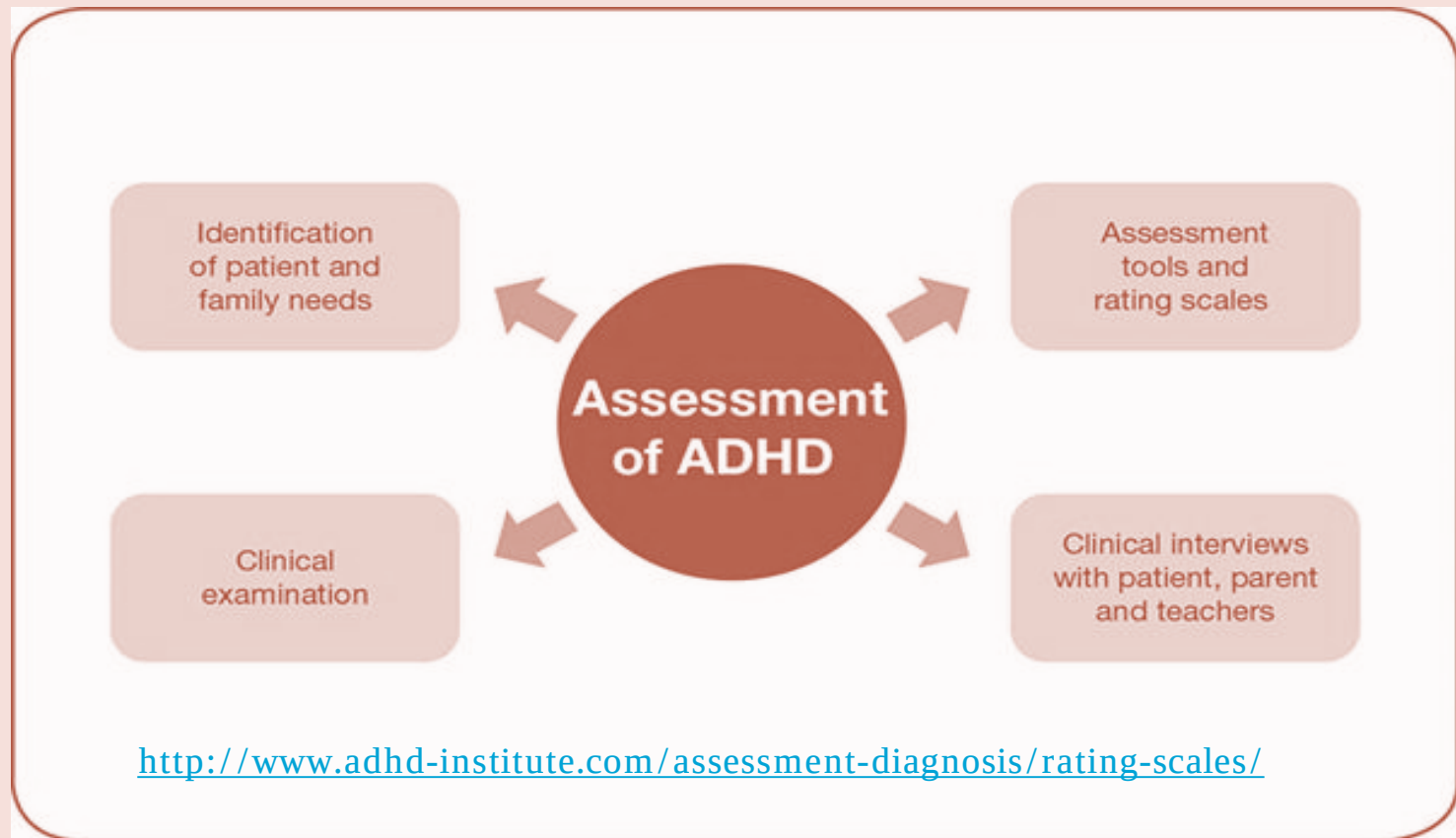
<i>Combined</i>	<i>Inattentive</i>	<i>Hyperactive/impulsive</i>
All three core features are present and ADHD is diagnosed when ≥ 6 symptoms of hyperactivity/impulsivity and ≥ 6 symptoms of inattention have been observed for ≥ 6 months	Diagnosed if ≥ 6 symptoms of inattention (but < 6 symptoms of hyperactivity/impulsivity) have persisted for ≥ 6 months	Diagnosed if ≥ 6 symptoms of hyperactivity/impulsivity (but < 6 symptoms of inattention) have been present for ≥ 6 months

Current severity of ADHD

<i>Mild</i>	<i>Moderate</i>	<i>Severe</i>
Few, if any, symptoms in excess of those required to make the diagnosis are present, and symptoms result in no more than minor impairments in social or educational functioning	Symptoms or functional impairment between “mild” and “severe” are present	Many symptoms in excess of those required to make the diagnosis, or several symptoms that are particularly severe, are present; or the symptoms result in marked impairment in social or educational functioning



There is currently no single available tool
for the diagnosis



Formal diagnosis is made using evidence accumulated from the initial clinical assessment and medical classification systems

ADHD: Prevalence and Demographics - UK



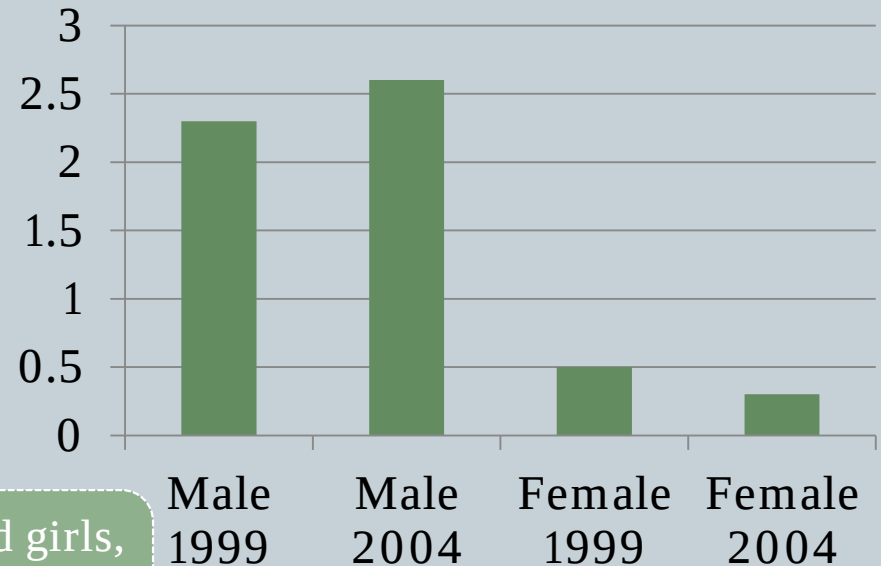
Between the ages of 5 and 15 years - 3.62% of boys and 0.85% of girls had ADHD.

The wide variation - the lack of a strict definition, lack of precise and objective methods for assessment, geographic differences.



- same definitions of ADHD for both boys and girls, and usually find more boys than girls with ADHD (a ratio of about 3 to 1).

Sex ratios vary widely - referral bias. Males are more likely to have behaviour and conduct problems and thus more likely to be referred for psychiatric follow-up (Barkley, 1988)



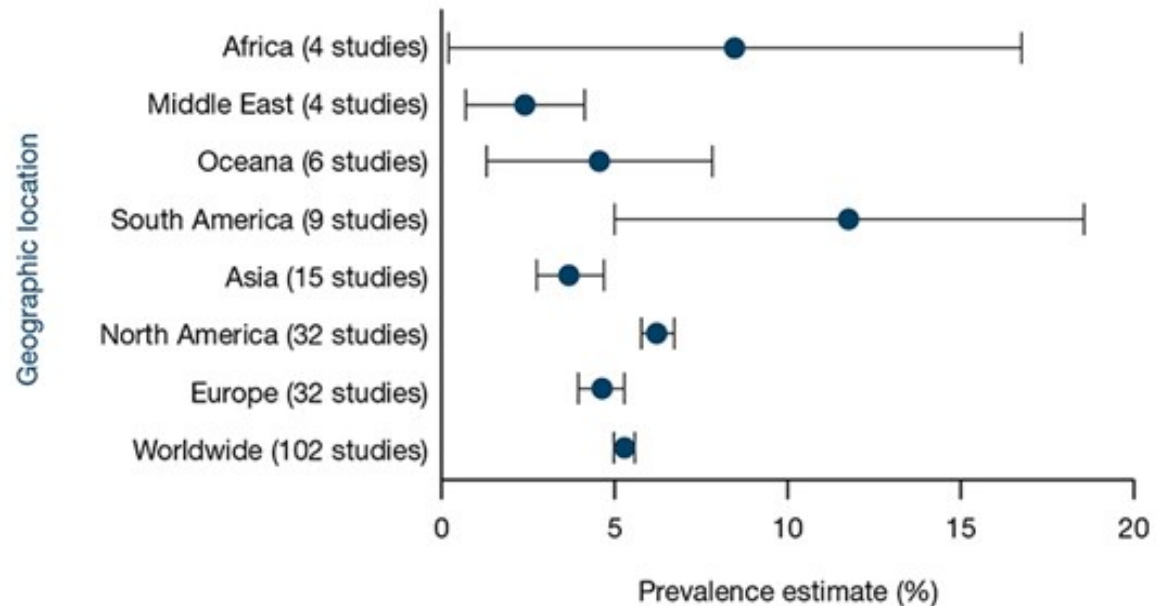
Children and Families Bill, 2013

Geographical location

DSM-V suggests that cultural attitudes towards the interpretation of behaviour may contribute to differences in prevalence estimates across studies

Meta-analysis of studies (n=102) - aged ≤ 18 years varied.

Prevalence for Europe was just under 5%



Children and adolescents with ADHD, n=171,756

Reproduced with kind permission Polanczyk G, de Lima MS, Horta BL, et al. Am J Psychiatry 2007; 164: 942-948.

More recently - Worldwide prevalence of ADHD is estimated at between 5.29% and 7.1% (Willcutt, 2012)

Comorbidity

A large percentage will have a comorbid learning disorder, another mental health disorder, or a neuro-developmental disorder.

This creates the chicken or egg conundrum.

Which came first: the ADHD or the supposed comorbidity?

The presence of comorbidity conditions makes the diagnosis of ADHD more difficult.

Larson (2011)

60,000, children, 6-17 years including over 5,000 with ADHD.

33% had one comorbidity disorder, 16% had two, and 18% had three or more.

Overall, 67% of ADHD children had at least one other mental health or neuro-developmental disorder compared to 11% of children without ADHD.

Learning disorders (46% vs 5%)

Conduct disorder (27% vs 2%)

Anxiety (18% vs 2%)

Depression (14% vs 1%)

Speech problems 12% vs 3%)

Autism spectrum Disorder (6% vs 0.6%)

Epilepsy/Seizures (2.6% vs 0.6%)

Aetiology

- Causes still largely unknown
- What we do know...
 - the occurrence of ADHD is closely tied with biological factors
 - ✦ Size and density of various brain structures
 - ✦ The way chemical reactions take place within the brain

Interplay between multiple genetic (Gizer, Ficks and Waldman, 2009; Larsson, et al. 2013) and environmental risk factors (Grizenko, et al. 2012)

Risk factors that affect the neurobiological basis of the disorder (Cortese, et al. 2012)

Brain structure differences



- Smaller cortex in ADHD
 - thoughts and actions
 - Diminished size usually occurs in the Frontal lobe
 - impulse control
 - socialisation
 - reason
 - judgement

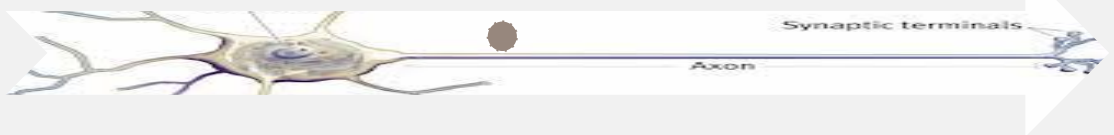
- Not fully developed – neurons in this part of the brain are needed for focussing, paying attention.



Chemical differences

Neurons communicate with one another using neurotransmitters – Dopamine, Serotonin, Norepinephrine

Neurotransmitter – chemical messengers that carry information to and from nerve cells



ADHD - Dopamine and Serotonin deficiency – Why?

To produce D and S– we need vitamin B6 and B9 (Folate)



When we eat/ supplement – they are in an inactivated form

We need to activate them



MTHFR

GENETIC DEFECT
(Methylenetetrahydrofolate)



So – defect does not allow activation

Genetics

DRD4 Dopamine receptor gene

DAT1 Dopamine transporter gene

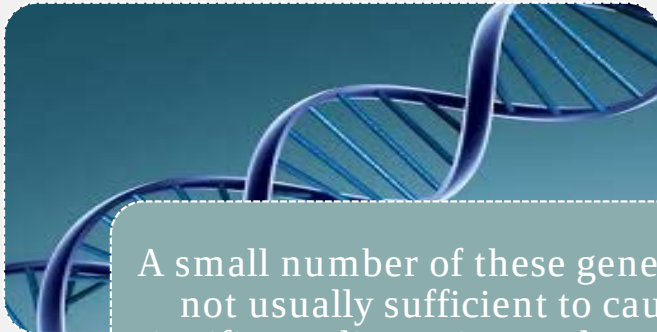
All chemical process in the body are under the control of genes.

Dopamine – no exception

There are 100s of dopamine genes

A number of defective variants have been found

So – ADHD is not caused by one faulty gene – but is ‘polygenetic’ disorder



A small number of these genes are not usually sufficient to cause significant disruption to dopamine transmission – **other processes will compensate for small defects.**



It's only when there are multiple defects that overwhelm the compensatory mechanisms

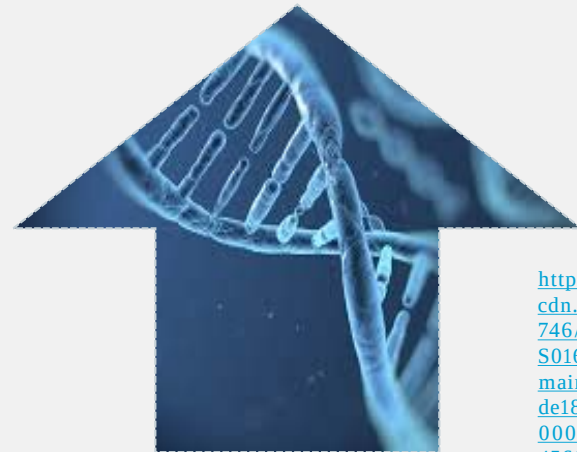
Genetics



Twin studies -
heritability estimated
to be 76% in the
population

(Farone , Perlis, Doyle, et al. 2005).

Swedish twin registry (n=21,000
children twin pairs, and n=37,714
adult twin pairs) - the genetic
contribution to ADHD was 88%
across child/adolescent twins; and
72% in adult twins (Larsson H, Chang Z,
D'Onofrio BM, et al. 2013)

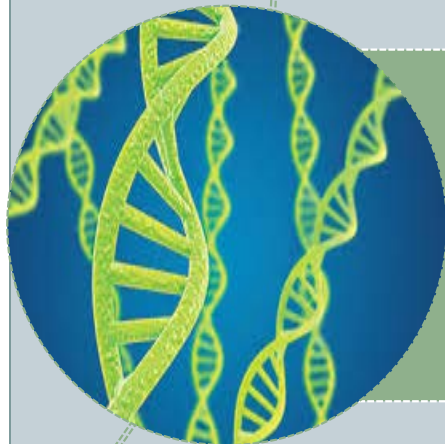


http://ac.els-cdn.com/S0165178114003746/1-s2.0-S0165178114003746-main.pdf?_tid=27823106-de18-11e5-9190-00000aabb0f02&acdnt=1456663282_c5d6a0399db243ede83a88b7d1b85617

Genetics



Adoption studies in children and adolescents have also indicated that **rates of ADHD are significantly higher between biological relatives** (parent-child or sibling) **compared with adoptive relatives** (Sprich, Biederman, Crawford, et al. 2000)



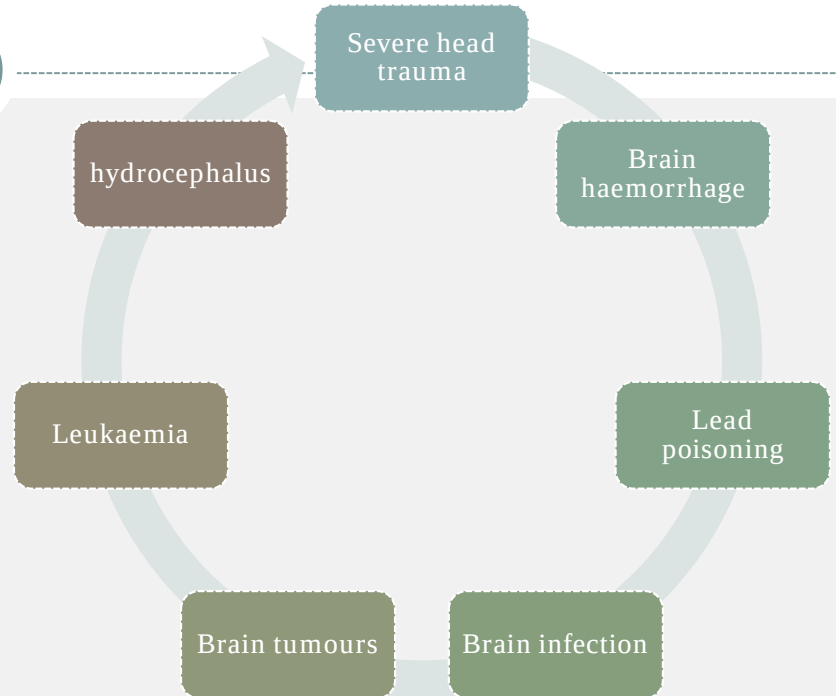
In addition, results from a genome-wide association study of pooled DNA from adults with ADHD suggested that a **genetic component was likely to contribute** to the persistence of ADHD from childhood into adulthood (Lesch , Timmesfeld, Renner, et al. 2008)

Non-genetic causes

Any condition that causes sufficient damage to the frontal lobes can cause ADHD



Some environmental conditions interact with a genetic susceptibility to ADHD



Low birth weight

Smoking during pregnancy

Alcohol abuse during pregnancy

Impact of ADHD on the life of a child



European Lifetime Impairment Survey

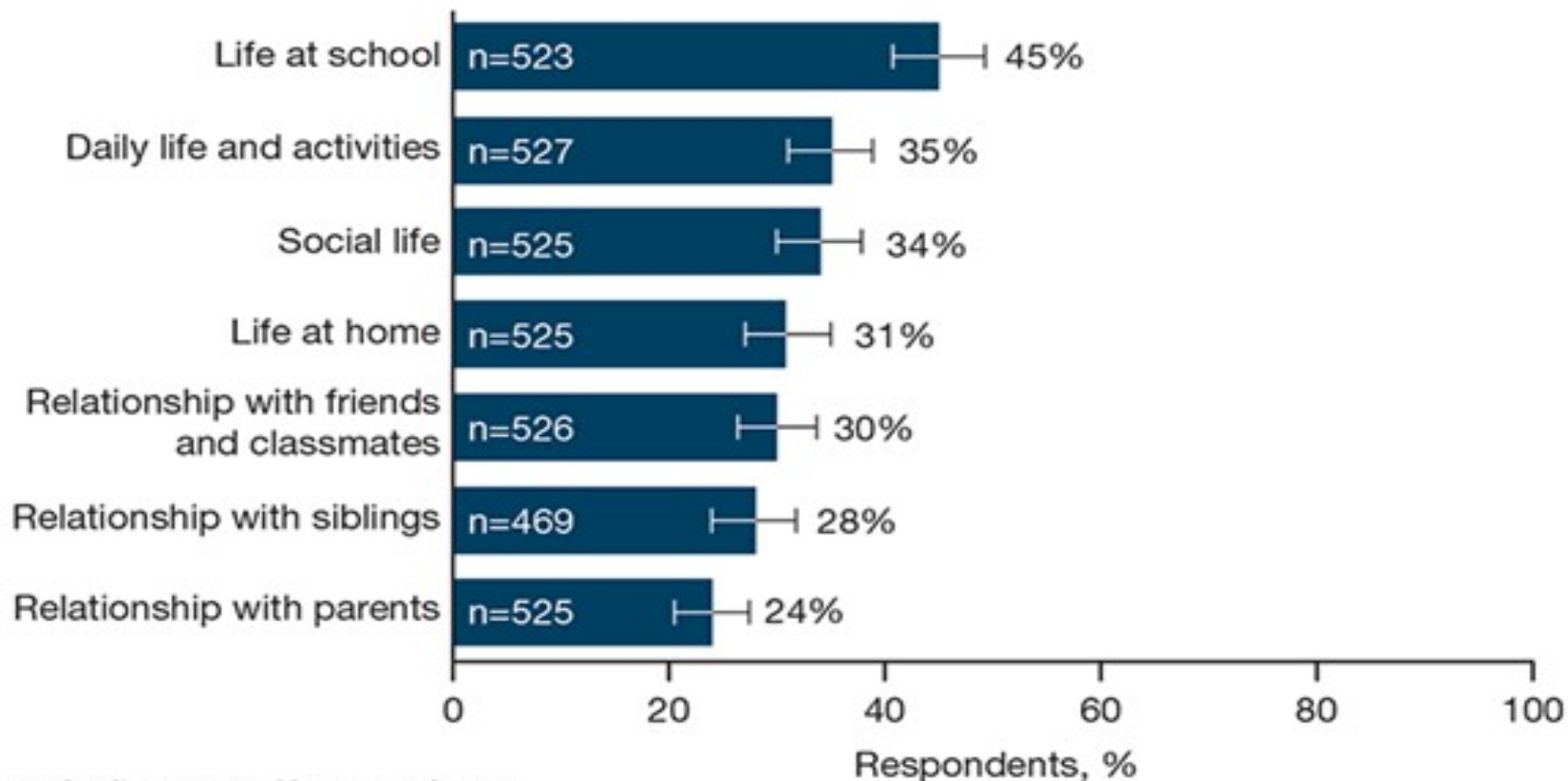


Assessed the **impairment and symptoms** of ADHD in children and adolescents from **six European countries** (France, Germany, Italy, Spain, the Netherlands and the UK).



Parents/caregivers of children and adolescents with ADHD (n=535) and without ADHD (n=424) participated in the online survey

Caci H, Doepfner M, Asherson P, et al (2013)



Life at school* as reported by parents/carers of children and adolescents with ADHD

*limited to children aged >6 years only

Reproduced with kind permission. Caci H, Doepfner M, Asherson P, et al. Eur Psychiatry 2013; 1-8.

Parents/caregivers reported a **perceived negative impact** on various aspects of their child's life, including life at school (45%), daily life and activities (35%) and social life (34%)

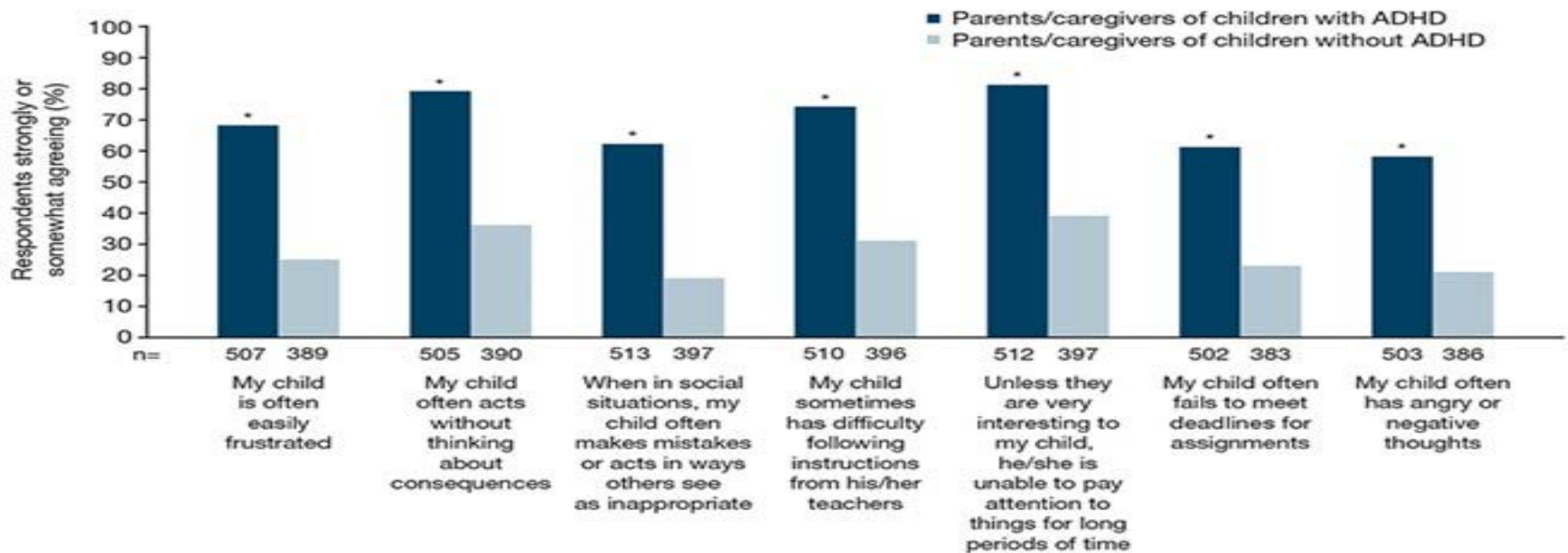
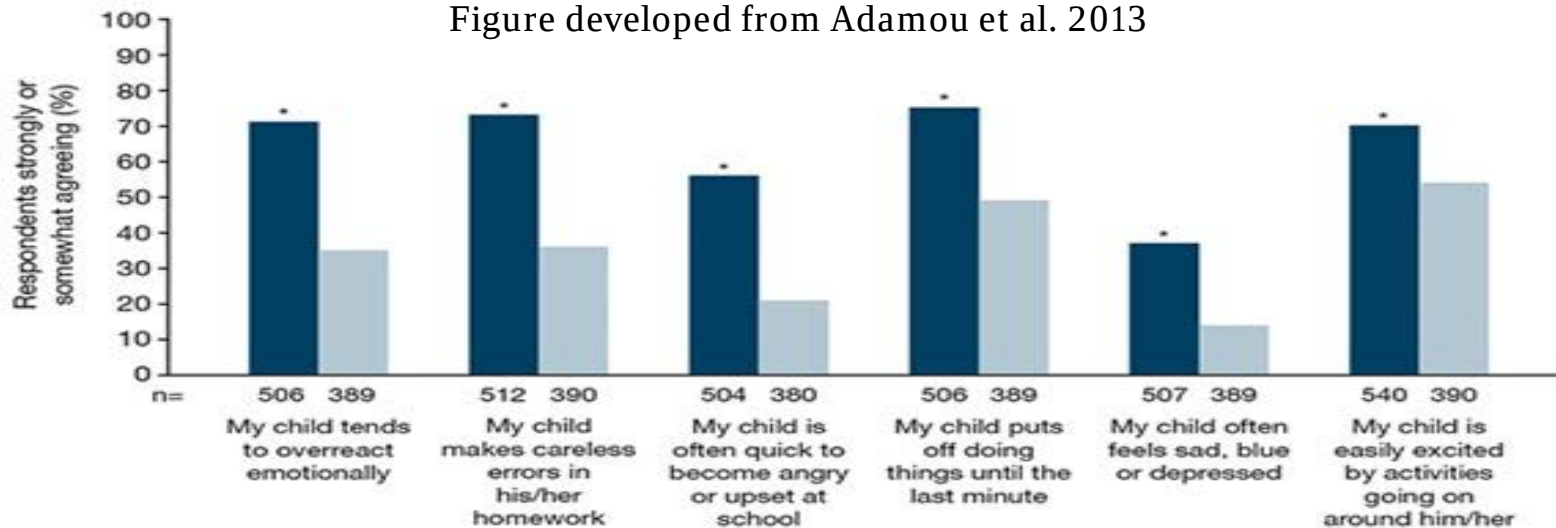


Figure developed from Adamou et al. 2013



Impairment at school as reported by parents/caregivers
Statements that more parents/caregivers of children with ADHD agreed with compared with parents/caregivers of children without ADHD when thinking about their child's experiences at school.
* $p < 0.001$ between ADHD and control groups; chi-squared test

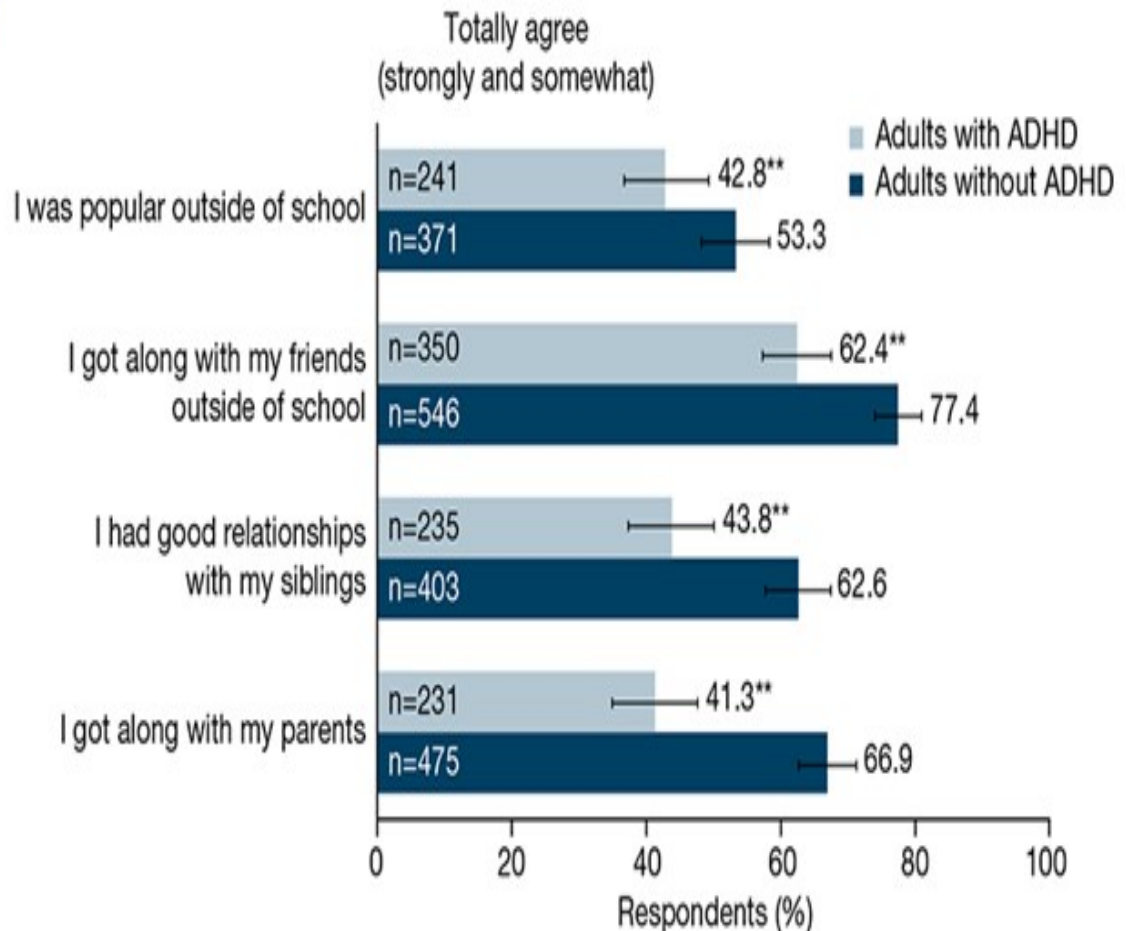
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Caci H, Doepfner M, Asherson P, et al. Eur Psychiatry 2013; 1-8.

European Lifetime Impairment Survey

(Caci, Asherson,
Donfrancesco et al. 2015)

Impact as recalled by adults
with ADHD (n=588)
compared with adults
without ADHD (n=736).

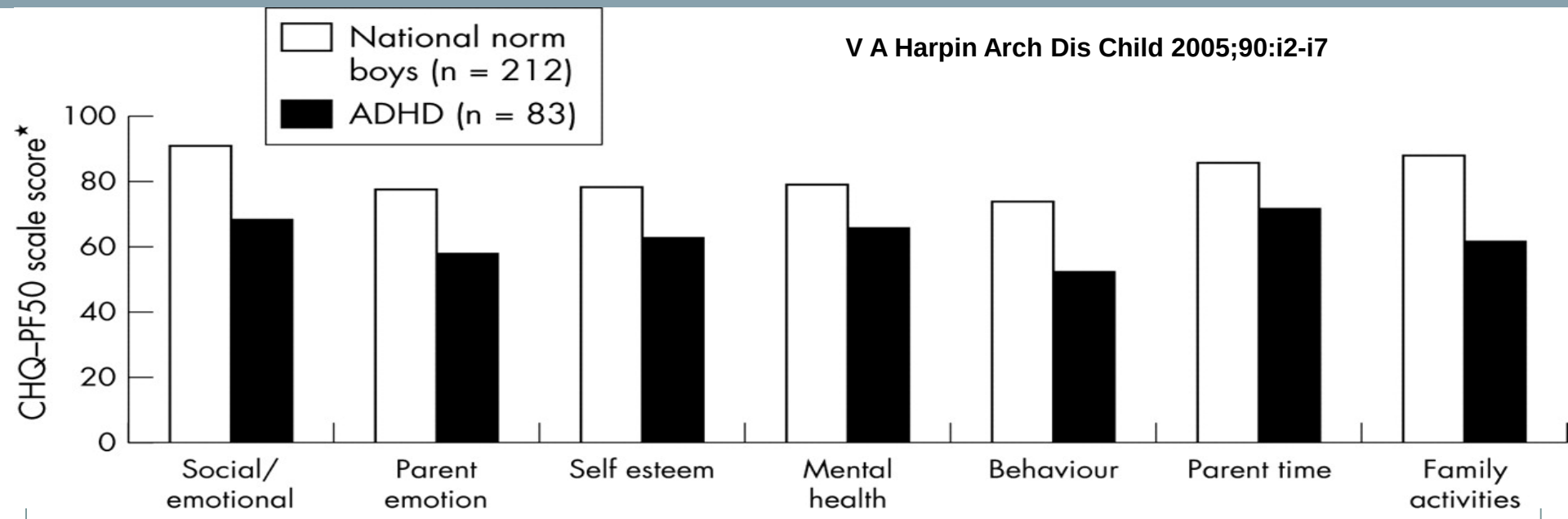
Adults with ADHD
recalled **similar**
impact from their
childhood, as
indicated by a lower
degree of agreement
with **positive**
statements related
to daily life and
relationships
compared with adults
without ADHD



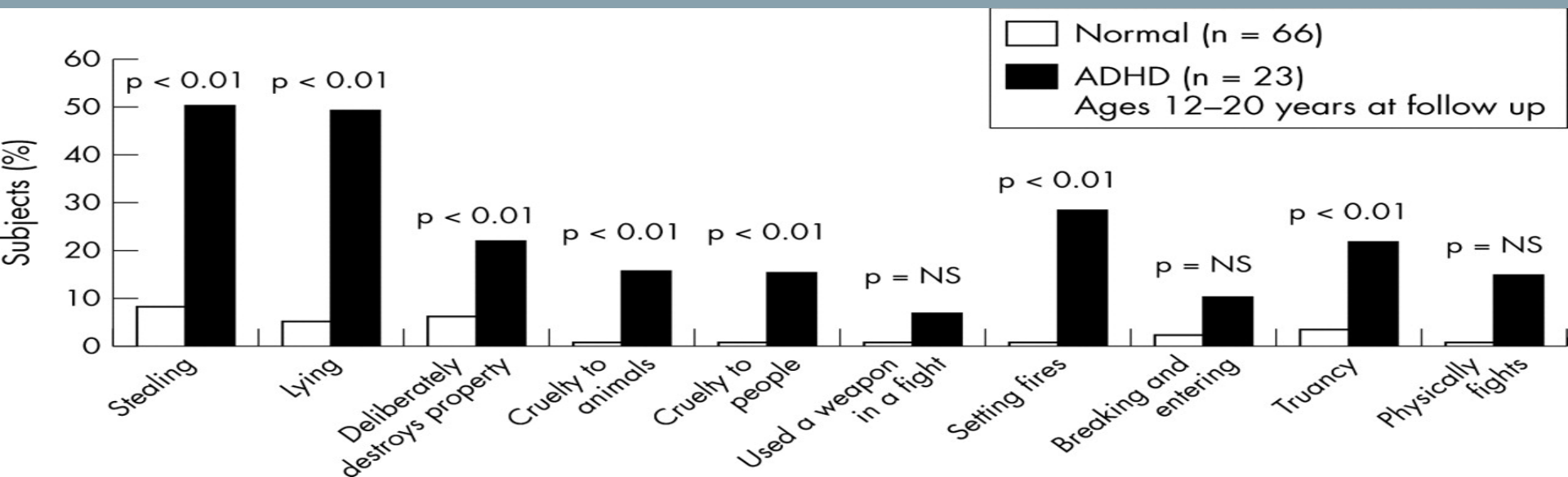
Impairment at home as recalled in adulthood
Statements with the greatest difference in terms of reported
agreement between adults with ADHD and adults without ADHD.
**p<0.001, chi-squared test
Error bars, 95% confidence intervals

Reproduced with kind permission.
Caci H, Asherson P, Donfrancesco R, et al.
CNS Spectr 2015; 20: 112-121.

Emotional and family functioning in children with ADHD compared with controls. *Higher scores indicative of greater functioning.



Antisocial behaviour in adolescents with ADHD. Data primarily represents outcomes in those with conduct disorder as teenagers.



Impact of ADHD on School Performance



Poor classroom behaviour



Poor academic achievement



Special education requirements
(tutoring and special educational
programmes)

School exclusion
Repetition of grades
Failure to gain
external
qualifications

Low self-esteem
Social clumsiness
Inflexibility
Defiant behaviour

Association between symptoms of **inattentiveness** (as measured by the Conners 10-item scale) in children aged 7 and 10 years and academic **underachievement** at age 16 years (APA, 2013)

Adolescents and adults with ADHD have been found to be **less likely to have achieved a further education degree** (Davis, Claudius, Palinkas, et al. 2012)

Executive dysfunction

This stems from an inability to inhibit present behaviour so that demands for the future can be met (Barkley, 2000)

Executive functions

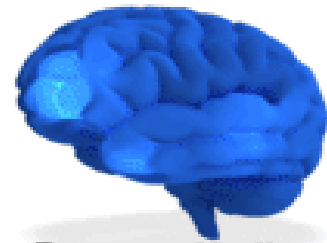
- Snake bite – no plan...
- Stepped on = bite back



Humans
fortunately
have the
option to
modulate
behaviour

Frontal and pre-frontal
lobes play a large role in
this **self control and
planning**.

Where we came from, where
we want to go, and how to
flexibly control ourselves
in order to actually execute
the plan = EF



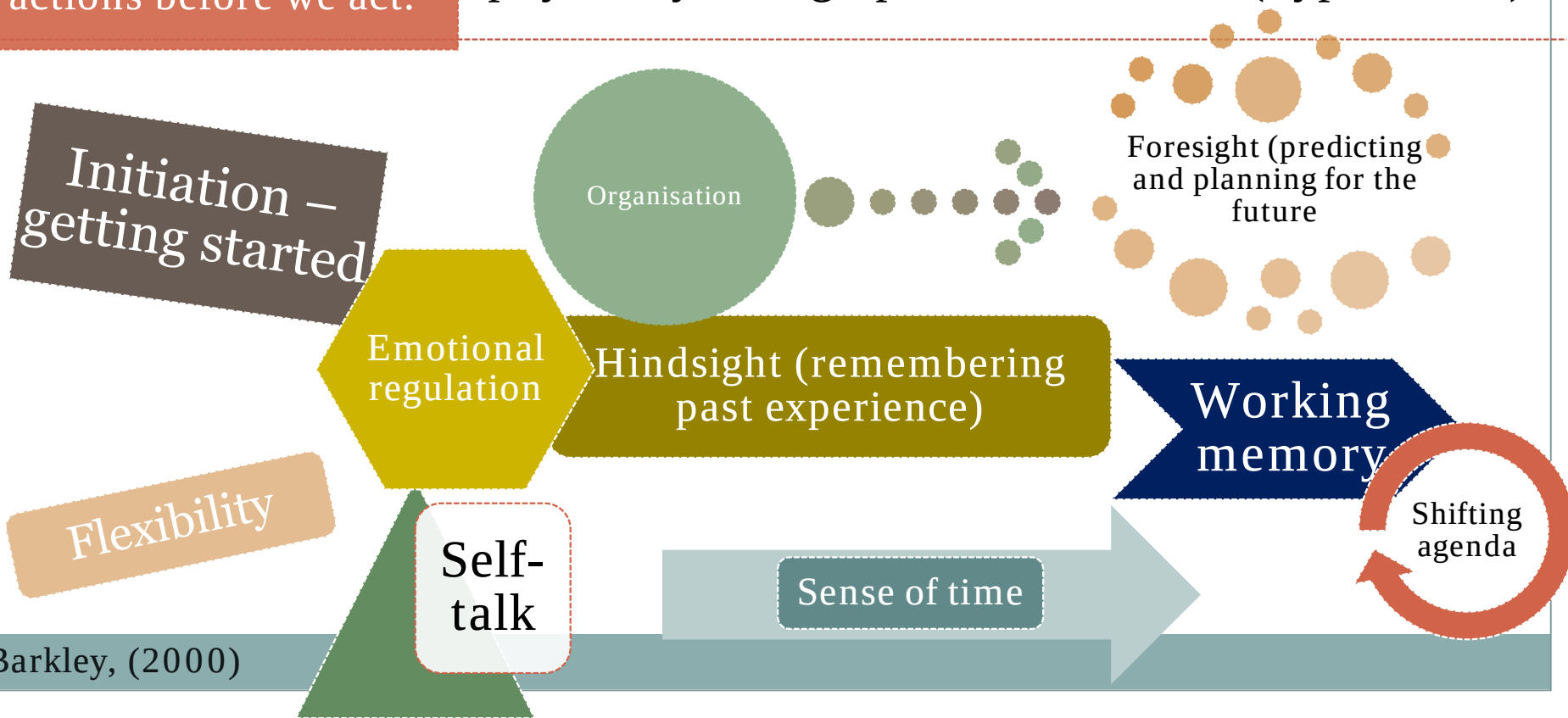
PresenterMedia

Executive dysfunction



Inhibition - putting brakes on your behaviour – to plan involves putting on the brakes on distracting activities – allows us to stop and think to consider our actions before we act.

Without these brakes – unable to inhibit distractions (inattentive), unable to inhibit instant reactions (impulsive), or unable to inhibit physically acting upon these stimuli (hyperactive).



Barkley, (2000)

EF- Attention deficit

- *Poor concentration*

- Inefficient attentional mechanisms
- Difficulty concentrating on tedious tasks
- Careless errors, lack of precision and attention to detail
- More difficulty with listening than looking – learning style
- Attentional mechanisms tire more quickly

- *Task Impersisitence*

- Failure to complete tasks

- *Disorganisation*

- Difficulty following sequences with supervision

- *Forgetfulness*

- LTM better than STM
- Difficulty following instructions

EF- Impulsivity

- *Stop and think*

- Difficulty with this before they act
- Often say the first thing that comes to mind
- Blurt out answers
- Tactless
- Risk taking
- Can't wait their turn

- *Do not learn from mistakes*

- *The problem isn't that they don't know the correct thing to do or that they don't want to do the right thing!*
- *It's their lack of SELF CONTROL*

Why? Impulsivity deficit

- The mechanisms that control behaviour in the brain seem unreliable
- *ADHD is a problem of performance, not of knowledge.*
- Russell Barkley PhD. – it's not a matter of not knowing what to do, but of not being able to do what you know.
- *Difficult delaying gratification*
 - LT goals difficult
 - Reward driven – immediate

- *So teaching a child to consider the consequences of their actions does not solve impulsivity.*
 - They lack the reflective and behavioural inhibition mechanisms needed to apply this to their everyday lives.
- *Act without thinking*
 - Risk taking
 - Accident prone
- *Difficulty sharing and cooperating - groups*

EF- Hyperactivity

- *On the go*

- Restless
- Overactive
- Fidgeting

- *Difficulty remaining seated*

- Walk about the classroom

- *Running and climbing*

- Slips and trips
- Injuries

- *Boisterous and loud*

- Difficulty engaging in quiet activities

- *Talk excessively*

- Disruptive

- *Repetitive sounds*

- Disruptive

Associated Problems



Peer problems

- Inattentive symptoms → ignored
- Hyperactive/impulsive symptoms → actively rejected
- Not deficient in social reasoning/understanding, but rather the execution of appropriate social behaviour

Family dysfunction/parental issues

- No clear causal relationship between family problems and ADHD
- Family problems can impact the severity and developmental course/outcomes of ADHD

Self-esteem

- Inflated: Positive illusory bias (Hoza)
- Low self esteem associated with comorbid depression

Summary



- ADHD is a neurological disorder
- Causes
 - Genetic and environmental
- 5% of school aged children in the UK
- Complex – co-morbidity
- Impact
 - Executive functions
 - Classroom behaviour
 - Academic achievement

Therapy Options



- Behavioural treatment
- Medication management
- Combining medication/behavioural treatment
- Educating parents/patient about ADHD
- Educational support services

Well-Established ADHD Treatments

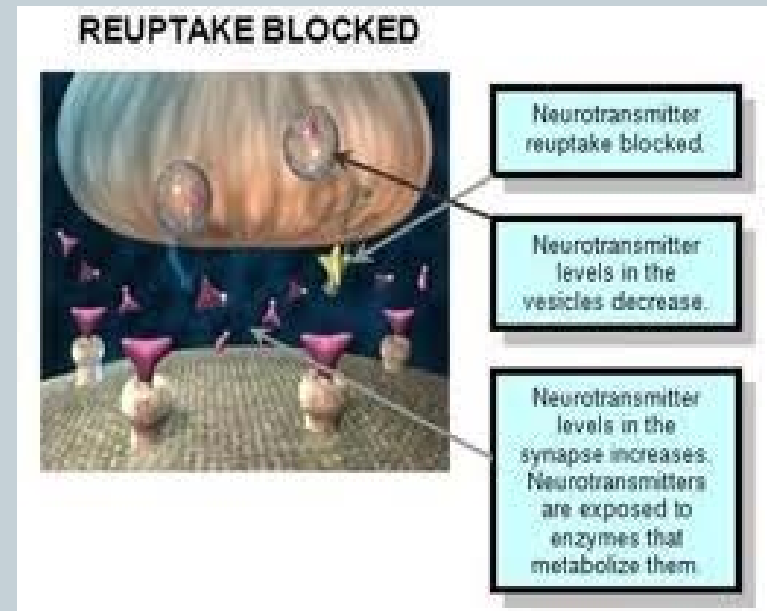


- Stimulant Medications
- Behavioural Interventions
 - Behavioural parent training
 - Behavioural classroom management
 - Intensive summer treatment programs



Medication: Stimulants

- Most well-researched, effective, and commonly used medication treatment for ADHD.
 - Methylphenidate (**Ritalin**, **Concerta**, and **Metadate**)
 - Dextroamphetamine (**Adderall**)
 - These medications reduce ADHD symptoms by:
 - Blocking the reuptake of **norepinephrine (NOR)** and **dopamine (DOP)** and facilitating their release
- Enhances NOR and DOP availability in in certain brain regions: **PFC** and **basal ganglia**



Stimulant Medications



- Research has shown that stimulants:
 - Are highly effective in reducing ADHD symptoms in the short term
 - Decrease disruption in the classroom
 - Increase academic productivity and on-task behaviour
 - Improve teacher ratings of behaviour
- Different formulations work best for different children
- Common side effects: insomnia, decreased appetite
- Strattera (atomoxetine)
 - A non-stimulant alternative that works well for some children
 - Has not been studied as long or as intensively as the stimulants
 - Smaller effect size relative to the stimulants

Limitations of Stimulant Treatment



- Individual differences in response
 - Not all children respond (approximately 80%)
- Limited impact on domains of functional impairment
 - Primary reason for treatment seeking
- Does not normalize behaviour
- Family problems beyond the scope of medication
- No long-term effects established
- Long-term use rare
- Limited parent/teacher satisfaction
- Some families are not willing to try medication

Behavioral Treatment Components



- Psychoeducation about ADHD
- Structure/routines
- Clear rules/expectations
- Attending/rewards
- Planned ignoring
- Effective commands
- Time out/loss of privileges
- Point/token systems
- Daily school-home report card
- Intensive summer treatment programs

Behavioral Treatment Considerations



- Need to address cross-situational impairments
 - Poor generalization from treatment setting to real-world
 - Implement treatments in all settings in which child shows impairment
- School behaviour
 - 504 Plan/Individualized Education Plan (IEP)
 - Academic interventions needed in addition to behavioural interventions (Raggi & Chronis, 2006)
- Environmental contingencies must be delivered **consistently**, which is difficult to maintain
 - Parental psychopathology can interfere with implementation

References



- American Psychiatric Association (2013) Diagnostic and Statistical Manual of Mental Disorders. Fifth edition. Washington, DC: American Psychiatric Association.
- Cortese S, Kelly C, Chabernaud C, et al. (2012) Toward systems neuroscience of ADHD: a meta-analysis of 55 fMRI studies. *Am J Psychiatry*; 169: 1038-1055.
- World Health Organization. The ICD-10 Classification of Mental and Behavioural Disorders. Available at: www.who.int/entity/classifications/icd/en/bluebook.pdf. - See more at: <http://www.adhd-institute.com/assessment-diagnosis/#sthash.iophPmFE.dpuf>
- Farone SV, Perlis RH, Doyle AE, et al. (2005) Molecular genetics of attention-deficit/hyperactivity disorder. *Biol Psychiatry*; 57: 1313-1323.
- Larsson H, Asherson P, Chang Z, et al (2013) Genetic and environmental influences on adult attention deficit hyperactivity disorder symptoms: a large Swedish population-based study of twins. *Psychol Med*; 43: 197-207.
- Gizer IR, Ficks C, Waldman ID (2009) Candidate gene studies of ADHD: a meta-analytic review. *Hum Genet*; 126: 51-90.
- Grizenko N, Fortier ME, Zadorozny C, et al. (2012) Maternal Stress during Pregnancy, ADHD Symptomatology in Children and Genotype: Gene-Environment Interaction. *J Can Acad Child Adolesc Psychiatry*; 21: 9-15.
- National Institute for Clinical Excellence. Full Guidance - Attention deficit hyperactivity disorder: Diagnosis and management of ADHD in children, young people and adults, March 2009.
- <http://www.adhd-institute.com/burden-of-adhd/impact-of-adhd/#relatedmedia>