School of Psychology
Ysgol Seicoleg

A Systematic Review of the Association of Early Life Maternal Depression and Offspring ADHD, and an Empirical Study of Cool and Hot Executive Function, in Relation to Conduct and Inattention/Hyperactivity Problems in Young Children.

Thesis submitted in partial fulfilment of the requirement for the degree of:

Doctorate of Clinical Psychology (DClinPsy)

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Cardiff University

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Thesis Preface

This thesis was submitted as partial fulfilment for the degree of Doctorate of Clinical Psychology (DClinPsy) and comprises an empirical paper and a systematic review.

The systematic review sought to understand the relationship of maternal depression and ADHD in the children of mothers who have experienced a period of depression, including during pregnancy. There are a variety of theories about how depression in mothers impacts upon rates of ADHD in children, some of which include shared genetic factors, changes in parenting practices, and an increase in other risk factors which may be related to an increased risk of ADHD, such as smoking during pregnancy.

In order to assess the relationship between maternal depression and ADHD in children, the systematic review focussed upon longitudinal studies only. Three databases were used to collect the studies (Medline, Web of Science and PsychInfo), resulting in 1,223 studies being reviewed and fourteen articles being included in the review.

Results indicated that a significant relationship between maternal depression and ADHD symptoms in children were reported in the majority of studies. This relationship was also significant when researchers took into account children's early life temperament, or past ADHD symptoms.

There were however several limitations of the studies reviewed including a bias towards wealthy westernised countries, limited consideration of the effect of fathers' mental health on children and using measures of ADHD which were mainly completed by parents and may be less accurate.

The empirical study looked at the relationship between executive function (a collection of brain processes which help us to think, plan, and problem solve) and symptoms of attention problems and hyperactivity, and conduct problems in young children. Although inattention/hyperactivity symptoms (e.g., fidgeting, not paying attention) and conduct problem symptoms (e.g., lying, aggression) commonly co-occur, it is conduct problems which better predict the development of

serious behaviour problems and antisocial/criminal behaviour later in life. Therefore, it is particularly important to understand which processes underlie each set of symptoms.

One aspect of executive function is the ability to make decisions (e.g., by inhibiting incorrect responses and being flexible in how one responds) when having to manage our emotions under conditions of reward or loss, known as "hot" executive function. "Cool" executive function refers to similar skills but when there are no clear rewards/losses (and hence emotions are less activated). Previous research has found that symptoms of those with conduct problem disorders (such as Oppositional Defiant Disorder and Conduct Disorder), but not inattention/hyperactivity symptoms (including those diagnosed in Attention-Deficit Hyperactivity Disorder; ADHD) are associated with difficulties on hot executive function tasks. Likewise, past research has found that attention/hyperactivity problems, but not conduct problem symptoms are more strongly related to cool executive function. However, research on this topic in samples of younger children when problems are just beginning to develop is less common.

The present study assessed cool and hot executive function in relation to inattention-hyperactivity and conduct problems, in a sample of children referred for emotional and behavioural problems (average age = 6.09 years). The study is important as understanding the underlying processes of particular symptom profiles will help inform the development of tailored preventative interventions in young children.

Overall 132 children took part in the study. Each child was given a variety of tasks to complete, some of which assessed cool executive function,(working memory and cognitive flexibility), whilst other tasks related to hot executive function (reward-related decision-making tasks). Against our hypotheses, cool executive function was not significantly related to either inattention-hyperactivity or conduct problems. Also, against predictions, conduct problem symptoms were not uniquely related to scores on hot executive function tasks. However, further analysis on the hot executive function tasks found that children who had concurrent high inattention-hyperactivity and conduct problem symptoms had the poorest performance on hot executive function tasks, including increased risk taking, slower learning, and they were less sensitive to punishment. It was

therefore concluded that in young children, hot executive function difficulties appear to be related to a combination of inattention/hyperactivity and conduct problem symptoms.

A systematic review of longitudinal studies investigating the association between early life maternal depression and offspring ADHD.

James R.D. Tucker

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A systematic review of longitudinal studies investigating the association between early life maternal depression and offspring ADHD.

Abstract

The systematic review sought to understand the relationship between maternal depression and later ADHD in children. Three databases were used to identify the studies (Medline, Web of Science and PsychInfo) resulting in 1,223 studies being reviewed and fourteen articles being included in the review. The majority of studies (N =8) were rated as high quality as assessed by the Critical Appraisal Skills Programme (CASP) guidelines. Results indicated that the majority of studies (N = 11) reported a significant relationship between maternal depression (across both prenatal and postnatal periods) and ADHD symptoms in children. This relationship was also significant when temperament, or past ADHD symptoms were controlled for. Several methodological issues were identified through the course of the review including mothers themselves being the predominant informants of ADHD symptomatology, limited consideration of paternal psychopathology, and limited consideration of the confounding effects of parental ADHD. The review nevertheless adds support to the literature regarding the relationship between maternal depression and possible negative impacts on the development of psychopathology in children.

Keywords (4) Maternal depression, ADHD, longitudinal, systematic review

Maternal depression has been consistently associated with the development of psychopathology in children, related to rates of both internalising and externalising disorders [1]. Prevalence rates of prenatal depression are estimated to be between 17% and 25%, with some evidence that rates of prenatal depression are increasing over time [2]. Postnatal depression is estimated to affect around 17% of mothers [3], with women twice as likely as men to experience depression during their lifetime [4]. Maternal depression has been frequently reported to be associated with child physical and mental health, and wellbeing [1,5-7]. This review seeks to focus on the longitudinal association between early life maternal depression and Attention Deficit Hyperactivity Disorder (ADHD), which is one of the most commonly diagnosed childhood psychiatric disorders.

ADHD refers to a collection of symptoms of inattention (including difficulties with sustained attention, following instructions and organisational skills) and hyperactivity and impulsivity (including fidgeting, restlessness, and difficulties in turn taking) [8]. It is estimated that around 5% of children meet criteria for ADHD, with a further 5% displaying sub-diagnostic threshold levels of attention/hyperactivity problems that nevertheless cause impairment in function. ADHD is two to three times more commonly reported in males than females [9].

ADHD has a significant genetic component, with a recent meta-analysis by Faraone and Larsson [10] reporting a 74% heritability estimate, based upon family, twin and adoption studies. It has been found that some environmental factors, such as maternal tobacco and alcohol use, interact with genetic risk associated with increased rates of ADHD [11]. Similarly, both prenatal alcohol and tobacco use have been associated with higher rates of ADHD in offspring [12,13]. Maternal stress in pregnancy has also been shown to be more prevalent during the gestation periods of children with ADHD, compared to their healthy siblings, with some evidence that this effect is mediated by genetic factors [14].

There is an increasing body of evidence which suggests that diagnosis of ADHD in early childhood is related to later life outcomes. ADHD difficulties in early childhood are associated with increased rates of substance use and poorer academic performance in adolescence [15,16], and

increased prevalence of mood, substance use and antisocial behaviour disorders in adulthood [17]. It is therefore important to better understand what specific factors contribute towards the development of ADHD, so that rates of ADHD symptomatology can be reduced to mitigate against longer term negative outcomes. One such avenue is considering the impact of familial mental health problems upon children's development of ADHD, such as the impact of maternal depression on ADHD symptomatology in their children.

Socioeconomic status (SES) is a broadly relevant environmental factor in that low SES is associated with a 1.85 – 2.21 increase in rates of ADHD, although this increase is likely to be confounded by multiple other risk factors associated with low SES [18]. Indeed, Adverse Childhood Experiences (ACEs) have been found to be significantly associated with ADHD, with children with ADHD having a higher number of ACEs than children without ADHD [19]. Jimenez et al. (2017), reported a dose-response relationship, with greater number of ACEs associated with more severe ADHD symptomatology, controlling for early life ADHD symptoms [20]. Although the ACEs research is informative for public health interventions, it lacks sophistication in terms of understanding specific pathways to development of ADHD and is limited in informing on how to intervene at a clinical service level, or even more so, at an individual case level. Maternal depression, for example, comes under the broad ACE of "household mental illness"; understanding its specific role in the development of ADHD would have implications for both service development and intervention with individual families.

There are multiple posited mechanisms through which maternal depression may be considered to confer risk for offspring ADHD, including shared genetic risk for ADHD, intrauterine mechanisms, and exposure to risk factors for ADHD which are associated with higher rates of maternal depression, such as smoking or alcohol use during pregnancy [21]. Maternal depression has also been reported to alter attachment styles and patterns [22,23], with attachment insecurity associated with externalising behaviour difficulties in children [24], including Oppositional Defiant Disorder (ODD) [25] , and Attention Deficit Hyperactivity Disorder (ADHD) [26]. Disorganised attachment style has been specifically reported to be associated with ADHD symptoms, with

disorganised attachment style in infancy associated with teacher rated ADHD symptoms in school age children [27], with evidence suggesting that the relationship between disorganised attachment and ADHD symptoms remains when controlling for executive function and conduct disorder measures [28] and also persists into adolescence [29]. Alongside this attachment deprivation in early life has been associated with ADHD symptoms in adolescence, with greater exposure to attachment deprivation associated with greater increases in ADHD symptoms [30].

The task of understanding the role of the different risk factors is complicated due to the paternal psychopathology which confers additional genetic and environmental risk [31] , the chronicity of maternal depression [32], and individual child factors such as temperament [33].

A recent meta-analysis and systematic review by Cheung et al. (2016) reported a moderate positive relationship between maternal depression symptoms and ADHD symptoms in child-mother dyads [34]. However, as the review mainly included cross-sectional studies (i.e., examining the co-occurrence of maternal depression in children with ADHD), it could not conclude whether maternal depression was a causal mechanism in the development of ADHD. The current review aims to expand on this review by assessing the longitudinal relationship between maternal depression and ADHD. Longitudinal designs which attempt to control for likely confounding variables are pivotal in understanding the association between maternal depression and offspring ADHD over time, and also allow for the controlling of multiple factors over time.

A previous review by Sfelinioti and Livaditis (2017) examined the relationship between maternal depression and ADHD [35], however this review was of very questionable quality. For example, it could not be considered to be a comprehensive review as the PRISMA guidance was not adhered to, only a singular database was searched, and the article did not utilise a quality assessment tool to evaluate the quality of the papers included. Importantly, there was not a clear inclusion and exclusion criteria. Therefore, the current review was deemed necessary.

The aims of the current review were threefold: (i) To assess the relationship between maternal depression and ADHD, and whether the presence of early life maternal depression is significantly associated with later diagnosis of ADHD or symptoms of ADHD; (ii) To assess whether there are

differential effects for prenatal and postnatal depression; and (iii) To consider potential mediating or moderating factors; or confounding variables which may impact upon any reported relationship between maternal depression and ADHD.

Methods

The systematic review was conducted in reference to the guidance set out in the Preferred Reporting Items for Systematic Review and Meta-analysis (PRISMA) guidelines [36] .

Search Procedures

Articles published from 1809 through to December 2019 were identified through systematic searching of three electronic databases: Medline; PsychInfo; and Web of Science. Search terms were utilised in a database-specific manner utilising key terms which related to the three core concepts (maternal depression, ADHD, childhood). The search utilised maternal depression terms (prenatal OR pre-natal OR maternal or mother* OR post-partum OR postpartum OR post-natal OR postnatal AND depression OR depressive* OR postpartum dep*). These search terms were combined with the child specific terms (offspring OR child* OR bab* OR infant* OR adolescen* OR teen* OR young people OR young person*) and terms related to ADHD (adhd or attention deficit* or attention-deficit*). Key terms were exploded to include related terms. Upon completion of the systematic literature search, the introduction sections of each included study were reviewed in order to identify any additional publications which met the inclusion/exclusion criteria.

Inclusion/Exclusion Criteria

All studies assessing maternal depression and childhood ADHD or Attention Deficit Disorder (ADD) were considered for inclusion. Only studies written in English and published in peer-reviewed journals were included in the review process. Study designs were defined as longitudinal if they had a follow up period of six months or more. Both prospective and retrospective designs were suitable for inclusion in the review. Studies which did not include a validated measure of maternal depression or childhood ADHD were excluded from the review. Many studies included a measure of "externalising" constructs, such as those contained within the Childhood Behaviour Checklist (CBCL) [37]. These studies were only included if they reported ADHD-related sub-scales (e.g.

"hyperactivity"). Study designs were required to report the association between maternal depression and childhood ADHD, or ADHD symptomatology.

Maternal depression was defined as any period of depression experienced by mothers during the first ten years of a child's life, assessed by validated depression measures including structured clinical assessment tools and validated questionnaire measures. ADHD was similarly defined when it was assessed by validated questionnaires and/or structured clinical assessment tools. Studies which identified cohorts solely based upon ICD or DSM codes within medical records were excluded, as they did not fulfil this criterion. In line with the focus of the review upon early childhood, studies were only included in the current review if participants' first follow up time point was ≤10 years of age.

Quality Assessment

Studies were assessed utilising the Critical Appraisal Skills Programme (CASP) guidelines for assessing the quality of Cohort Studies [38]. This framework does not provide a specified scoring criteria or system. Therefore, a unique scoring system was devised based on the CASP criteria and is reported in Table 1 (maximum score = 24). Studies were graded into high (19 - 24), medium (13 - 18) and low (≤ 12) quality categories. Quality rating reliability was assured by a postgraduate researcher as an independent reviewer. A random sample of papers (25%) were selected for review by the independent reviewer, who was blinded to the original quality assessment ratings. Ratings were then compared to the category assessments of the first author: high, medium or low quality. Agreement on category definition was very high (100%), the mean discrepancy in numerical scores was 1 (SD =1).

The main methodological problems relating to lower ratings were: insufficient blinding to exposure for outcome measures, due to the majority of studies utilising parental rated measures of ADHD; limited identification of confounds, particularly paternal psychopathology and maternal ADHD symptoms; significant follow up drop out; and bias within samples towards those of higher SES, income or educational backgrounds.

Overview of Studies

There were 1,223 studies that were identified during the systematic review, following the searches and deduplication of articles. Out of these studies, 112 were screened and 98 articles excluded due to not meeting the inclusion criteria of the review. Fourteen studies were therefore selected for inclusion in the systematic review. Primary reasons for exclusion included: no measure of maternal depression or ADHD; utilising externalising measures only; no validated measures utilised (e.g. studies reliant on ICD or DSM coding in clinical records); design that was not longitudinal; and no reported analysis of the relationship between maternal depression and ADHD. The PRISMA flow diagram below in Figure 1 describes the process. Study characteristics are reported in Table 2, with study results reported in Table 3.

Figure 1.

PRISMA Flow Diagram

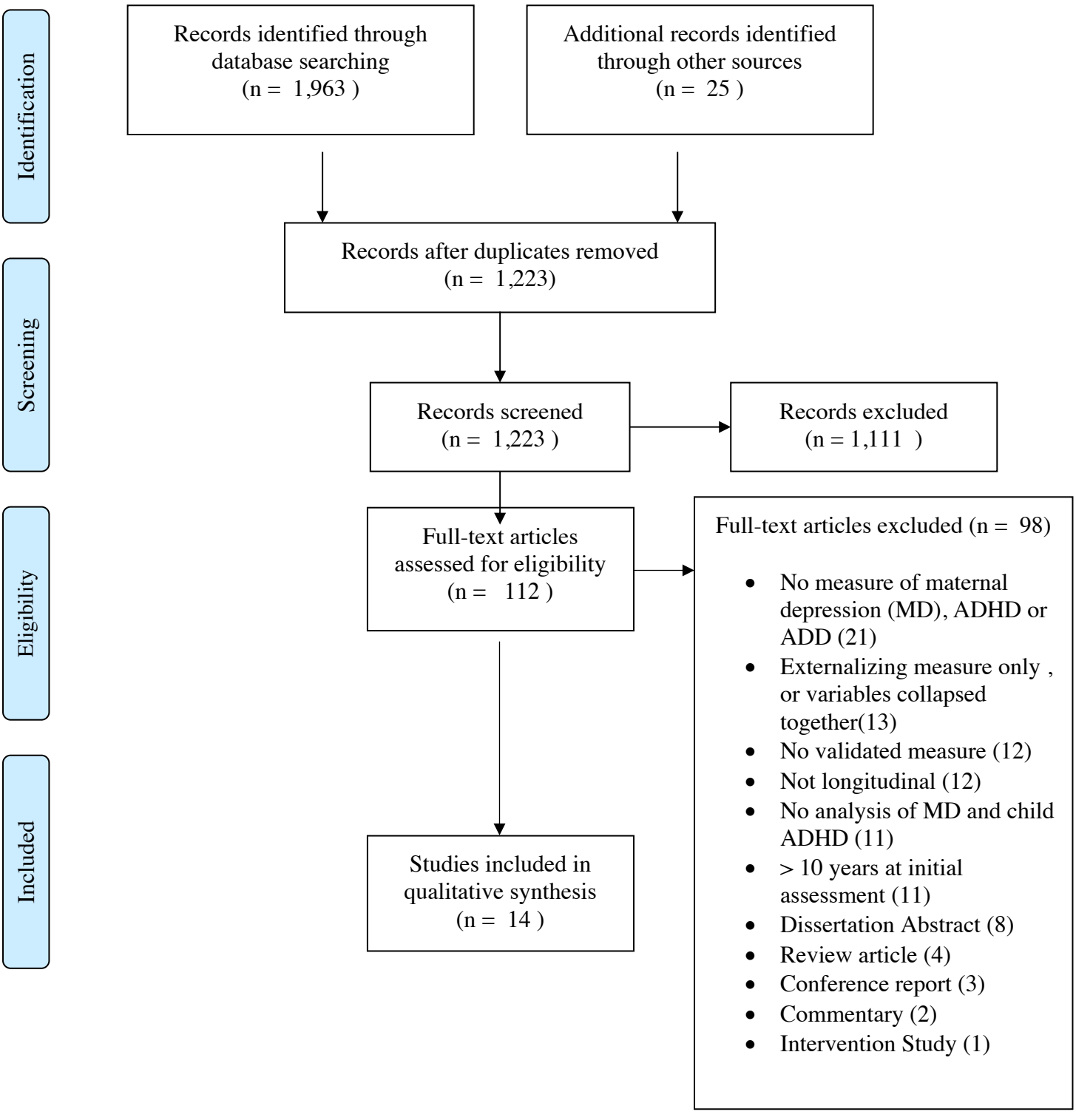

Table 1. *CASP Quality Review Table*

	Apter-Levy et al. (2013) [39]	Ashman et al. (2008) [40]	Barker et al. (2012) [41]	Breaux et al. (2019) [42]	Choenni et al. (2019) [43]	Galera et al. (2011) [44]	Jusiene et al. (2015) [45]	Koutra et al. (2017) [46]	Leis et al. (2013) [47]	Park et al. (2018) [32]	Romano et al. (2006) [48]	Van Batenburg-Eddes et al. (2013) [49]	Vergunst et al. (2019) [50]	Wolford et al. (2017) [51]
1. Does the study address a clearly focussed issue?	1	2	2	2	2	2	2	2	2	2	2	2	2	2
Is the population studied clear?	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Are the factors studied clear?	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Are the outcomes clear?	P	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
2. Was the cohort recruited in an acceptable way?	0	1	2	1	2	2	1	2	2	1	2	2	2	2
Was the cohort respresentative of a defined population?	N	P	Y	P	Y	Y	P	Y	Y	P	Y	Y	Y	Y
Was everybody included who should have been included?	N	P	Y	Y	Y	Y	Y	Y	Y	N	Y	Y	Y	Y
3. Was the exposure accurately measured to minimise bias?	2	2	2	2	2	2	2	2	2	2	2	2	1	2
Did they use objective measurements?	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Are they validated measures?	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Were all subjects classified into exposure groups using the same procedure?	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	N	Y	Y
4. Was the outcome accurately measured to minimise bias?	2	1	1	1	1	1	1	1	2	1	1	1	1	1
Did they use objective measurements?	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Were the measures validated?	Y	Y	Y	Y	Y	P	Y	Y	Y	Y	P	Y	P	Y
Were the measurement methods similar in different groups?	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
Were the subjects and/or the outcome assessor blinded to exposure?	Y	P	N	N	N	P	N	N	Y	N	N	N	P	N

Table 1. (continued).

	Apter-Levy et al. (2013) [39]	Ashman et al. (2008) [40]	Barker et al. (2012) [41]	Breaux et al. (2019) [42]	Choenni et al. (2019) [43]	Galera et al. (2011) [44]	Jusiene et al. (2015) [45]	Koutra et al. (2017) [46]	Leis et al. (2013) [47]	Park et al. (2018) [32]	Romano et al. (2006) [48]	Van Batenburg-Eddes et al. (2013) [49]	Vergunst et al. (2019) [50]	Wolford et al. (2017) [51]
5. Have the authors identified all important confounding factors?	0	1	1	1	1	1	1	1	1	1	1	2	2	1
Have they taken account of the confounding factors in the design/analysis?	N	P	P	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
6. Was the follow up of subjects complete enough?	1	1	1	1	1	2	1	2	1	2	1	1	2	1
Was there anything special about the outcome of the people leaving, or the exposure of the people entering the cohort? (Y = Yes, but is a negative response in this context)	P	Y	Y	P	P	N	Y	N	P	N	P	Y	N	P
Was the follow up of subjects long enough?	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y
7. What are the results of the study?	1	1	2	1	1	2	2	2	2	1	2	2	2	2
Have they reported the rate of proportion between the exposed/unexposed, the rate/ratio difference?	P	N	Y	N	N	Y	Y	Y	P	Y	Y	Y	Y	y
8. How precise are the results?	1	1	2	1	1	2	2	2	2	2	2	2	2	2
Are confidence intervals reported?	N	N	Y	N	N	Y	N	Y	N	Y	Y	Y	Y	Y
9. Do you believe the results?	0	1	2	1	1	2	2	2	2	1	2	2	2	2
Are the design and methods of this study sufficiently flawed to make the results unreliable ?	P	N	N	N	N	N	N	N	N	P	N	N	N	N

Table 1 (continued).

	Apter-Levy et al. (2013) [39]	Ashman et al. (2008) [40]	Barker et al. (2012) [41]	Breaux et al. (2019) [42]	Choenni et al. (2019) [43]	Galera et al. (2011) [44]	Jusiene et al. (2015) [45]	Koutra et al. (2017) [46]	Leis et al. (2013) [47]	Park et al. (2018) [32]	Romano et al. (2006) [48]	Van Batenburg-Eddes et al. (2013) [49]	Vergunst et al. (2019) [50]	Wolford et al. (2017) [51]
10. Can the results be applied to the local population?	1	1	2	2	1	2	1	1	2	0	2	2	2	2
Are the subjects covered in the study sufficiently different from your population to cause concern?	P	P	Y	N	P	N	P	P	N	Y	N	N	N	N
11. Do the result of the study fit with other available evidence?	1	2	2	2	1	2	2	2	1	1	2	2	2	2
12. What are the implications of the study for practice?	1	1	2	2	1	2	1	1	2	1	2	2	2	2
Total score (maximum = 24)	11	15	21	17	15	22	18	20	21	15	21	22	22	21
Quality rating	Low	Medium	High	Medium	Medium	High	Medium	High	High	Medium	High	High	High	High

NB: Main questions are highlighted in grey and scored from a scale of 0 - 2 (0= standard not met, 1 = standard partially achieved, 2 = standard fully met). Sub questions are reported in white rows, and were utilised to aid scoring (Y = Yes, N = No, P = Partial)

High Quality = 24 - 19
Medium Quality 18 - 13
Low Quality ≤12

Table 2. *Study Characteristics (N = 14)*

Study	Location & Study Name	Participants and Study (e.g. ALSPAC)	Mean Maternal Age in Years (SD)	Measures (Maternal Depression and Child ADD/ADHD)	Informants on child outcome	CASP Quality Rating
Apter-Levey et al. (2013) [39]	Israel (study name not specified).	156 mother-child dyads	38.66 (4.40)	**Depression**: Beck Depression Inventory (BDI) administered at childbirth, 6 months, 9 months and 6 years. **ADHD**: Development and Well-Being Assessment (DAWBA) at 6 year follow up.	Parents	11
Ashman et al. (2008) [40]	USA, Adjustment to Parenthood Study.	159 mother-child dyads	31.1 (4.54)	**Depression**: Structured Clinical Interview for DSM-III (SCID); Center for Epidemiological Studies Depression Questionnaire; and a modified version of the Longitudinal Interval Follow Up Questionnaire. Measures were administered when their children were 14 months, 24 months, 3.5 years, 4.5 years, and 6.5 years of age. **ADHD**: Diagnostic Interview Schedule for Children IV - Parent version; Child Behaviour Checklist (CBCL) and the Child Adaptive Behaviour Inventory (CABI). Teachers completed the Achenbach Teacher Report Form, CABI, ADHD rating Scale and the Child Behaviour Scale peer subscales at age 6.5.	Parents and teachers	15

Table 2. (continued).

Study	Location & Study Name	Participants and Study (e.g. ALSPAC)	Mean Maternal Age in Years (SD)	Measures (Maternal Depression and Child ADD/ADHD)	Informants on child outcome	CASP Quality Rating
Barker et al. (2012) [41]	UK, ALSPAC.	7429 mother-child dyads.	Not reported	**Depression**: Edinburgh Postnatal Depression Scale (EDPS) at 21 months. **ADHD:** DAWBA assessment at 7-8 years of age.	Parents	21
Breaux et al. (2019) [42]	USA, (study name not specified).	258, mother-child dyads.	Not reported	**Depression:** The Millon Clinical Multiaxial Inventory–III at each time annually (4 time points from the child age of 3 - 6) **ADHD**: National Institute of Mental Health Diagnostic Interview Schedule for Children–Fourth Edition at age, 3, 4, 5 and 6.	Parents	17
Choenni et al. (2019) [43]	Netherlands, Generation R.	584 mother-child dyads	31.9 (SD = 3.7)	**Depression:** Brief Symptom Inventory at age 3. **ADHD:** Conners Parent Rating Scale-Revised: Short Form (CPRS- R:S) administered at age 8.	Parents	15
Galera et al. (2011) [44]	Canada, Quebec Longitudinal Study of Child Development.	2120 mother-child dyads (2057 used to model hyperactivity-impulsivity and inattention trajectories)	Not reported	**Depression:** Center for Epidemiological Studies Depression Scales (CES-D) (abbreviated version) at 5 months. **ADHD:** Interviewer computerised questionnaire (comprising questions from the CBCL, Ontario Child Health Study Scales and Preschool Behaviour Questionnaire) completed when children were 1.5, 2.5, 3.5, 4.5, 5, 6 and 8 years of age.	Parents	22

Table 2. (continued).

Study	Location & Study Name	Participants and Study (e.g. ALSPAC)	Mean Maternal Age in Years (SD)	Measures (Maternal Depression and Child ADD/ADHD)	Informants on child outcome	CASP Quality Rating
Jusiene et al. (2015) [45]	Lithuania (study name not specified).	281 mother-child dyads	Not reported	**Depression:** EDPS scale at 3 and 6 months, and 3 years after childbirth. **ADHD:** CBCL - at age 18 months, 2 years and 4 years.	Parents	18
Koutra et al. (2017) [46]	Greece, Rhea study.	642 mother-child dyads.	Not reported	**Depression:** EDPS at 28-32 weeks of gestation and postnatally at 8 weeks postpartum. **ADHD**: Attention Deficit Hyperactivity Disorder Test and the Strength and Difficulties Questionnaire (SDQ) at 4 years of age.	Parents	20
Leis et al. (2013) [47]	UK, ALSPAC.	2,891 mother-child dyads	Not reported	**Depression:** EPSD at 18 and 32 week gestation, 8 weeks, and 8 months postpartum and childhood (21, 33, 61 and 73 months, and 11 years of age.) **ADHD:** SDQ - collected at child age 10 by teacher and age 11 by mother report.	Teacher and Parent (Parent report at 11 years of age)	21
Park et al. (2018) [32]	Canada (study name not specified).	191 mother-child dyads.	Not reported	**Depression:** Hamilton Depression Rating Scale (HAMD) and the EDPS during 2nd and 3rd trimesters, 6 weeks, 3 months, 6 months and 10 months. BDI was used at 3 year follow up and the EDPS at 6 year follow up. **ADHD:** The MacArthur Health and Behavior Questionnaire at age 6.	Parents	15

Table 2. (continued).

Study	Location & Study Name	Participants and Study (e.g. ALSPAC)	Mean Maternal Age in Years (SD)	Measures (Maternal Depression and Child ADD/ADHD)	Informants on child outcome	CASP Quality Rating
Romano et al. (2006) [48]	Canada, The National Longitudinal Survey of Children and Youth (NLSCY).	2946 mother-child dyads	Not reported	**Depression:** CES-D at cycle 1. Child aged (0 - 23 months) **ADHD:** Hyperactivity symptoms measured by CBCL at cycle 2, 3 and 4. (age 2 - 7)	Parents	21
Van Batenburg-Eddes et al. (2013) [49]	Netherlands / UK, Generation R/ALSPAC	Generation R, n = 2,280 and ALSPAC, n = 3,442. (mother-child dyads)	Generation R: 31.7(SD= 3.9) ALSPAC: 29.3(SD= 4.4)	Generation R: **Depression** Brief Symptom Inventory at 20 weeks gestation and repeated when the child was aged 3. **ADHD:** CBCL at child age 3, attention subscale. ALSPAC: **Depression:** EDPS at 18 weeks of pregnancy and at 33 months. **ADHD:** SDQ at child aged 4 years. Hyperactivity/inattention subscale	Parents	22

Table 2. (continued).

Study	Location & Study Name	Participants and Study (e.g. ALSPAC)	Mean Maternal Age in Years (SD)	Measures (Maternal Depression and Child ADD/ADHD)	Informants on child outcome	CASP Quality Rating
Vergunst et al. (2019) [50]	Canada, Quebec Longitudinal Study of Child Development.	1374 mother-child dyads	25.9 (SD = 4.9)	**Depression:** CES-D completed at 5 months. **ADHD:** Combined early childhood behavior scale including items from the CBCL, Ontario Child Health Study Scales and the Preschool Behavior Questionnaire. Assessment at age 15 and 17 were made using the Mental Health and Social Adaptation Assessment for Adolescents.	Parents, Teachers and Self-report.	22
Wolford et al. (2017) [51]	Finland, Prediction and Prevention of Pre-eclampsia and Intrauterine Growth Restriction (PREDO)	1,779 mother-child dyads.	31.9 (SD not reported)	**Depression:** CES-D biweekly up to 14 times during pregnancy. Depressive symptoms were also reported using the BDI at 3 and 6 year follow up. **ADHD:** Conners Hyperactivity Index at age between 3 to 6 (mean = 3.8)	Parents	21

Table 3: *Study Results (N = 14)*

Study	Analysis	Covariates	Results	Limitations
Apter-Levey et al. (2013) [39]	MANCOVA and Chi-square test comparison between chronically depressed and never-depressed mothers and their children	Salivary Oxytocin OXTR Genotype Mother and Child Behaviours Paternal emotional distress Child social outcomes	Significant between-group difference between children of chronically depressed mothers compared to never-depressed mothers and diagnosis of ADHD.	Sample: Limited solely to married/cohabiting parents. Exclusion of common co-morbidities (e.g. anxiety). Sample highly educated (80% - college level) Measures/Design: Parents sole informants. Limited co-variates accounted for. Effects are for two divergent cohorts - limited evidence about depression trajectories. No measures for broader parental psychopathology/ADHD.

Table 3. (continued).

Study	Analysis	Covariates	Results	Limitations
Ashman et al. (2008) [40]	Latent growth mixture models with MANOVA analysis	Stressful life events Social support Parenting stress Relationship adjustment ECG Heart rate	Trajectories of decreasing and stable mild depression was related to increased hyperactivity and attention problems in children compared to non-depressed mothers. The effect of maternal depression on externalising-ADHD behaviours was significantly mediated by contextual risk factors. Maternal depression had a significant impact upon child frontal brain activity, but this did not significantly mediate the association between maternal depression and child ADHD.	Sample: Primarily European-American ethnicity. Excluded serious mental health problems, and/or alcohol/substance use difficulties. No measure of paternal mental health or parental ADHD.
Barker et al. (2012) [41]	Logistic regression	Socioeconomic status (SES) Living conditions Family risk factors Early parenthood Educational attainment Substance Use Forensic history	There was a significant effect of maternal depression (when child =1.5 years) on child diagnosis of ADHD at age 7.5 without controlling for the cumulative risk index. When the risk index was added and assessed this resulted in a 41% decrease in externalising disorders although ADHD was not analysed separately.	Sample: 95% Caucasian. Measures/Design: Parents sole informants. ADHD not assessed separately when controlling for risk index. Paternal psychopathology not assessed.
Breaux et al. (2019) [42]	Regression analysis	Covariances between concurrent maternal and paternal functioning variables. Past ADHD symptoms (child), Co-morbid ODD, parental ADHD.	There was initial evidence for a bidirectional relationship between maternal depression and child ADHD, as assessed by yearly follow up between the ages of 3 -6 year. For paternal depression the effect was unidirectional, with child ADHD predicting increased rates of paternal depression. When parental ADHD symptoms were entered into the depression model neither child ADHD, nor parental depression reported significant effects.	Measures/Design: Parents sole informants. Multiple models utilised but not clearly integrated.

Table 3. (continued).

Study	Analysis	Covariates	Results	Limitations
Choenni et al. (2019) [43]	Linear regression analysis	Maternal depression Maternal harsh parenting Child attention Executive function difficulties	Bivariate correlation between maternal depressive symptoms at age 3 and childhood ADHD symptoms at age 8, was statistically significant. This did not control for covariates as maternal depression was not a primary risk factor within the study.	Sample: Highly educated cohort. Ethnicity not reported. Measures/Design: Parents sole informants. No measure of general maternal psychopathology and/or ADHD. Maternal depression only utilised as covariate. Analysis: maternal depression not analysed as unique predictor.
Galera et al. (2011) [44]	Group-based trajectory modelling and logistic regression.	Infant Temperament Methylphenidate exposure Premature Birth Low Birth Weight Alcohol/Substance Use/Smoking during pregnancy Family structure Low maternal education Maternal age at birth of child Household income Family dysfunction Parenting Paternal psychopathology	Maternal depression when child aged 5 months was significantly related with trajectories of high levels of hyperactivity-impulsivity and/or attention, controlling for covariates.	Sample: Ethnicity not reported. Measures/Design: Parents sole informants. Hybrid measure utilised combining items from different instruments to assess ADHD. No measure of parental ADHD.
Jusiene et al. (2015) [45]	Latent class modelling	New-born Health Infant Problem Behaviours Maternal attitudes towards infant care. Maternal self efficacy Maternal responses to children's negative emotions	Maternal postnatal depression was found to be an additional risk factor for stable high attention and behaviour regulation problems trajectories vs decreasing attention and behaviour regulation problem trajectory, accounting for covariates.	Sample: Highly educated. Majority married. Relatively small sample. Measures: Parents sole informants. CBCL utilised as measure of attention and behaviour regulation. No measure of paternal mental health. Maternal ADHD not assessed.

Table 3. (continued).

Study	Analysis	Covariates	Results	Limitations
Koutra et al. (2017) [46]	Multivariate analysis	Maternal age at delivery Maternal education Smoking status Working status Child sex Prematurity Breastfeeding duration Pre-school attendance TV watching Birth order Number of children in family Quality of assessment	Maternal prenatal depressive symptoms were related to higher scores in Hyperactivity subscale. Maternal postnatal depressive symptoms were associated with higher scores in almost all subscales of ADHD (except Inattention). High levels of maternal postnatal depression (EPDS≥13) were associated with increased scores in Hyperactivity, Inattention and Impulsivity subscales and the Total ADHD Index, accounting for covariates within the analysis. The effects of postnatal depression on ADHD symptoms were more pronounced in children whose mothers smoked during pregnancy.	Sample: Excluded mothers with history of previous psychiatric disorder. Measures: Parents sole informants. No measures of paternal psychopathology or maternal ADHD.
Leis et al. (2013) [47]	Bivariable linear regression.	Marital status , Maternal age at birth, Child birthweight, Child gender, Maternal educational attainment Cigarette smoking during pregnancy Alcohol use during pregnancy	Prenatal depression was associated with increased total child emotional and behavioural difficulties. Prenatal depression was also significantly associated with teacher and maternal reported hyperactivity controlling for other periods of depression or anxiety and sociodemographic variables. Elevated symptoms of comorbid prenatal depression and prenatal anxiety did not predict greater increases in offspring emotional/behavioural problems, than prenatal depression alone.	Sample - Homogenous ethnicity (98.9% = Caucasian) Measures: Parents sole informants. No measure of paternal psychopathology. No measure of maternal or paternal ADHD.

Table 3. (continued).

Study	Analysis	Covariates	Results	Limitations
Park et al. (2018) [32]	Multivariable linear regression analyses	Child sex Age Gestational age at birth Birth weight Prenatal SSRI exposure Maternal history of depression Maternal education Maternal minority status	Maternal depressive symptom trajectories were unrelated to children's internalizing and externalizing symptomatology at age 6. Children whose mothers reported a decreasing trajectory of depressive symptoms over time had lower reported levels of ADHD symptoms, than those whose mothers had consistently few depressive symptoms. ADHD symptomatology did not differ between children of mothers in the increasing and low trajectory groups.	Sample: High risk sample. High rate of drop out. Small sample size. Measures: Parents sole informants. No measure of maternal ADHD or paternal psychopathology.
Romano et al. (2006) [48]	Semiparametric group mixture model to estimate trajectories, followed by logistic regressions.	Maternal age Parenting practices Family dysfunction Child Temperament Smoking and substance use Birth Weight	Postnatal depression, assessed between the ages of 0 -23 months, increased the risk of high and persistent hyperactivity significantly after accounting for sociodemographic and psychological covariates.	Sample: Homogenous ethnicity (88.5% - Caucasian) Measures: Parents sole informants. No measure of maternal ADHD. No paternal psychopathology measures. No measure of other ADHD related constructs - e.g. inattention.

Table 3. (continued).

Study	Analysis	Covariates	Results	Limitations
Van Batenburg-Eddes et al. (2013) [49]	Logistic regression	Generation R: Maternal education Parental anxiety Maternal smoking and alcohol use during pregnancy Family Income Child ethnicity Child gender Child birth weight ALSPAC: Parental anxiety Maternal education Family income Ethnicity Maternal smoking and alcohol use during pregnancy Child gender Child birth weight	Prenatal depression was associated with child attention problems at age 3 in both ALSPAC and Generation R Cohorts. Controlling for maternal anxiety and depression after birth, prenatal depression was no longer associated with later child attention problems in the Generation R cohort. ALSPAC reported statistically significant associations for prenatal anxiety and depression when controlling for the same variables. However, there was no significant difference between paternal and maternal symptoms, in their relationship to later child attention problems.	Measures: Parents sole informants. No measure of maternal or paternal ADHD.

Table 3. (continued).

Study	Analysis	Covariates	Results	Limitations
Vergunst et al. (2019) [50]	Group-based trajectory modelling.	Methylphenidate use Child IQ Child Temperament Birth weight Parental tobacco, alcohol and drug use during prenatal and postnatal periods SES Family income Parental education Family structure Family dysfunction Mother-child interaction Parenting Parental Psychopathology	Postnatal depression assessed at 5 months, was a significant risk factor which predicted high-symptom trajectories of hyperactivity–impulsivity and inattention, after controlling for covariates.	Measures: Use of a composite measure utilising items from a number of instruments. No measures of parental ADHD.
Wolford et al. (2017) [51]	Latent profile analysis and logistic regression analysis	Maternal history of physician diagnosed depression Maternal ADHD problems Maternal age at delivery Antidepressant use Psychotropic medication use Smoking during pregnancy Parity (primiparous vs. multiparous) Chronic Hypertension Type I Diabetes Gestational length Birthweight Child sex Family structure Maternal alcohol use Maternal education	Prenatal depressive symptoms displayed a significant association with symptoms of ADHD at age 3, controlling for covariates including further symptoms of depression and maternal ADHD. There was an additive effect of postnatal depression to later child symptoms of ADHD.	Sample: Ethnicity not reported. Measures: Parents sole informants. No measure of paternal psychopathology

Results

Study characteristics of the selected papers are reported in Table 2. The study publication dates were between 2006 – 2019. All included studies utilised prospective designs. Fifty percent of the studies were published within the past five years, further strengthening the case for this review in light of the other two similar reviews undertaken relatively recently. All fourteen papers came from high-income countries as defined by the World Bank [52]. The papers originated from eight different countries: Canada; United Kingdom; Netherlands; United States of America; Israel; Lithuania; Greece; and Finland. The studies examined eleven different cohorts; three studies utilised the UK based Avon Longitudinal Study of Parents and Children (ALSPAC) Cohort, two utilised the Generation R Cohort from the Netherlands, and two utilised the Canadian Quebec Longitudinal Study of Child Development cohort.

Design

All studies were prospective longitudinal designs in accordance with the inclusion criteria. The majority of studies (N = 11) utilised maternal depression as a primary exposure measure, though for three studies [39,42,43] maternal depression was not the primary focus of the study. Thirteen studies focussed exclusively on early childhood (<10 years of age), whilst one study, included due to measurement of ADHD at earlier time points, followed up children until 17 years of age [50]. The earliest initial follow up assessment of ADHD, or ADHD symptoms such as hyperactivity, was 18 months [44,45].

Participants

Samples size ranged from 156 to 5,722 for a study which utilised both ALSPAC and Generation R cohorts [49]. A significant number of studies did not report ethnicity data. For those that did, White European/American was consistently the most frequent demographic recruited. Samples skewed towards parents with higher levels of reported educational attainment. Six studies reported mean maternal age. These ranged from 25.9 [50] to 38.6 years [39] . For large scale cohort studies (ALSPAC, Generation R and Quebec Longitudinal Study of Child Development), samples were representative of the local population.

Measures

The majority of studies (N = 13) utilised self-reported questionnaires for assessing maternal depression, with the exception of one study which utilised the Structured Clinical Interview for DSM-III [40]. The most commonly utilised measure was the Edinburgh Postnatal Depression Scale (EDPS) which was used in six studies, followed by the Centre for Epidemiological Studies Depression Scale (CES-D) which was used in four studies, and the Beck Depression Inventory which was utilised in three studies. More general measures of psychopathology from which depression scores could be extracted were also included, including studies utilising the Brief Symptom Inventory and Millon Clinical Multiaxial Inventory–III.

Five studies measured maternal depression across both prenatal and postnatal time points [32,46,47,49,51]. Prenatal depression was measured across gestation points ranging from 12 weeks [45] to 32 weeks [46,47]. Maternal depression after birth was assessed at a significant range of time points, from 6 weeks post birth [32], to 11 years [47]. Five studies assessed maternal depression at a single time point [41,43,44,48,50]. Three studies assessed prenatal depression at multiple time points [32,47,51], with a further seven studies assessing depression in the postnatal period across multiple time points [32,39,40,42,45,47,51].

All studies utilised some element of parental assessment of ADHD. Three studies also incorporated teacher rated measures [40,47,50] . One study utilised self-report measures at later time points [50]. There was considerable diversity in the measures utilised to assess ADHD with 11 unique measures utilised across studies. The Child Behaviour Checklist (CBCL) and the Strength and Difficulties Questionnaire (SDQ) were the most frequently used measures (N = 4 and N = 3). Two studies utilised a structured clinical assessment tool, the Diagnostic Interview Schedule for Children (Parent Version). Two studies drawing from the Quebec Longitudinal Study of Child Development cohort utilised a hybrid measure comprised of items from validated measures including, the Child Behaviour Checklist, Ontario Child Health Study Scales, and the Preschool Behaviour Questionnaire

[44,50]. There was a significant range in the age at which ADHD or ADHD symptomatology was assessed; from 17 months of age [44] to 17 years of age [50].

Main findings and Synthesis

Association between Maternal Depression and ADHD

The majority of studies (N = 11; mean CASP rating = 19.4 – "High quality") reported a significant positive association between maternal depression and ADHD, assessed via prospective longitudinal designs. To better understand the pattern of results, the studies will be discussed in terms of those where no relationship between maternal depression and ADHD was found, the impact of timing of maternal depression, and the impact of covariates.

Studies Where no Association between Maternal Depression and ADHD was Reported

Three studies did not report a significant association between maternal depression and ADHD (N=3; mean CASP rating = 15.6 "Medium Quality") [32,40,42]. Two of these studies looked exclusively at postnatal depression [40,42], whilst one study reported maternal depression measures across the prenatal and postnatal periods [32]. Two of the studies were trajectory studies [32,40]. Ashman et al. [40] reported significantly higher rates of attention and hyperactivity problems in children of mothers with decreasing levels of depression over time or with stable depression, compared to children of never depressed mothers. However, the effect of maternal depression was found to be significantly mediated by a contextual risk index comprised of measures of stressful life events, social support, parenting stress, and relationship adjustment. Park et al. [32] reported that children of mothers who reported decreasing depressive symptoms over time, had lower ADHD symptom scores, compared to children of mothers who had consistently low depressive symptoms. ADHD symptoms also did not differ between increasing and low trajectory groups. This paradoxical result should however be interpreted with caution due to the small sample sizes described in the increasing and decreasing trajectory groups (N = 27 and N = 15 respectively), which are likely to be underpowered. Breaux and Harvey [42] reported a relationship between maternal depression and later child ADHD, but this relationship was significantly moderated by parental ADHD. Compared to many of the studies that did find a significant relationship between maternal depression and later child

ADHD when controlling for parental ADHD, this study had a relatively smaller sample size (N = 258) and thus might have been underpowered to detect smaller effects.

Timing of Maternal Depression

Prenatal Depression

The timing of when maternal depression occurs is important in understanding any longitudinal relationships it has with ADHD. For example, relationships between maternal depression and ADHD in middle childhood might be due to the impact of having a child with ADHD on maternal depression, whereas any relationship between prenatal ADHD and later ADHD is less likely to be due to confounding factors such as child temperament. Five studies examined maternal depression across the pre and postnatal periods [46,47,32,49,51]. No studies examined solely prenatal depression. Two of these studies reported evidence of specific associations between ADHD symptoms and both prenatal and postnatal depression [47,51]. Wolford et al. [51], reported that prenatal depression was associated with ADHD symptoms at 3 and 6 years, controlling for both maternal ADHD and postnatal depression, with a separate unique effect for postnatal depression alone. Depression across the prenatal period was highly correlated, and the study reported no differential effects between depression recorded at different points of gestation. Both prenatal and postnatal depression had unique associations with ADHD symptoms. Similarly Leis et al. [47], reported that prenatal depression was significantly associated with hyperactivity symptoms at 10 and 11 years of age, for both maternal and teacher reports. These effects remained when controlling for maternal psychopathology other than depression (and therefore the potential confounding variable of shared genetic risk) and contextual risk factors including maternal tobacco and alcohol use in pregnancy, low birthweight, gender, maternal educational attainment, and marital status. Postnatal depression assessed at 8 weeks and 8 months, was correlated with hyperactivity at age 10 and 11 in bivariate correlations, no further analyses controlling for covariates were reported of postnatal depression. Koutra et al. [46], reported that prenatal depression was associated with increased hyperactivity at 4 years, with postnatal depression also associated with increased scores across all subscales of ADHD symptoms (hyperactivity, impulsivity and inattention) at 4 years of age. The study did not control for child

temperament in the postnatal analysis, and there was no comparison of whether prenatal or postnatal depression was a stronger predictor of later ADHD symptoms.

Van Batenburg-Eddes et al. [49], who utilised both the Generation R and ALSPAC cohorts; with a combined sample size of 5,722 mother-child dyads (CASP Rating = 22, "High Quality"), assessed the link between prenatal depression and attention/hyperactivity symptoms, encompassing postnatal depression as a key covariate. In the Generation R Cohort, although associations between prenatal depression and later attention problems (at age 3) were found, when controlling for various covariates (gender; ethnicity; age of child; maternal education; alcohol use and smoking during pregnancy; paternal depression and anxiety symptoms during pregnancy; and postnatal depression symptoms at age 3) there was no effect of prenatal depression or anxiety on child attention problems. The ALSPAC cohort however presented significant effects of both prenatal depression and anxiety symptoms upon child attention problems assessed at age 4, after controlling for parental anxiety and depression symptoms, maternal education, family income, ethnicity, maternal smoking, alcohol use during pregnancy, child gender and child birth weight. Importantly, the size of the effect did not differ significantly between prenatal maternal and paternal depression. It is possible that the differences across cohorts are attributable to how depression and ADHD were measured, as both designs utilised different measures of depression and ADHD, and also measured both at different time points.

In the final study to assess depression during pre and post-natal timepoints, Park et al. [32] reported that maternal depressive symptom trajectories from pregnancy to 3 years postpartum were unrelated to ADHD symptomatology at age 6.

Maternal Depression Following Pregnancy

Alongside studies assessing maternal depression across prenatal and postnatal periods, several studies examined maternal depression solely after birth and did not report any prenatal measures [39-45,48,50]. Pivotal to understanding the relationship between postnatal maternal depression and offspring ADHD, are adequate controls for early life factors which may confound any apparently causal association between maternal depression and later ADHD including early life ADHD symptoms or child temperament.

Five studies utilised trajectory modelling designs to assess the impact of maternal depression over time, following birth and not including the prenatal period [40,44,45,48,50]. Four studies reported clear associations with maternal depression and ADHD; each controlling for child temperament within regression analyses [44,45,48,50]. It is notable that three out of these four studies had CASP ratings in the "high" range (CASP ratings of 21-22), whereas the study that did not find an effect was of lower quality (CASP rating of 15).

Galera et al. [44] reported that maternal depression assessed at 5 months postpartum was significantly associated with trajectories of high-levels of hyperactivity-impulsivity and inattention from 17 months to 8 years of age (controlling for methylphenidate exposure, early child temperament, birth factors, smoking and substance use, socioeconomic factors, family dysfunction, parenting, and paternal psychopathology). Vergunst et al. [50] replicated the findings within the same cohort, and expanded upon them utilising multiple informants (mothers, teachers and self-report) from the ages of 1.5 to 17 years of age. Maternal depression was a consistent risk factor for hyperactivity-impulsivity and inattention trajectories. Romano et al. [48] focussed solely upon hyperactive symptoms. Maternal depression significantly increased the risk of high and persisting hyperactivity, controlling for sociodemographic and psychological covariates (including early child temperament, parenting; maternal age, family dysfunction, smoking and substance use, and birth weight). Research by Jusiene et al. [45] examining attention regulation problems also identified maternal depression as a significant risk factor for high attention and behaviour regulation problem trajectories, compared with decreasing trajectories; controlling for new born health, temperament, attitudes to infant care and response to child's emotions, and maternal self-efficacy [45].

One trajectory modelling study did not report a significant association between maternal depression and ADHD. Ashman, Dawson and Panagiotides [40] reported that chronic depression was associated with greater levels of externalising-ADHD problems compared to children whose mothers were never depressed, or had stable mild depression. Further analyses however revealed that contextual risk factors (stressful life events, social support, parenting stress, relationship adjustment) significantly mediated the relationship between maternal depression and externalising-ADHD

symptoms, so the relationship between maternal depression and externalising-ADHD symptoms was no longer significant.

A further four studies examined maternal depression following birth and ADHD using alternative longitudinal designs [39,41-43]. Three of these studies reported an association between maternal depression and offspring ADHD [39,41,43], whilst one did not [42]. Only one of the studies which reported an association, controlled for early life ADHD symptoms [41], and the other studies tended to be of lower quality (CASP ratings in the low to medium range) and thus their results are less helpful in terms of drawing strong conclusions.

Overall five studies controlled for child temperament, or child ADHD symptoms at an earlier time point [41,44,45,48,50]. All of these studies reported a significant relationship over time between maternal depression and offspring ADHD, hence providing evidence that associations between maternal depression and later ADHD are not simply as a result of the child's challenging behaviour causing increases in maternal depression.

Informant effects on outcome

The majority of studies (N = 11) relied solely on parent reports to assess ADHD. Three studies utilised other informant reports (teacher and self-report) [40,47,50]. Two of the studies reported a significant relationship between maternal depression and ADHD [47,50], whilst the third did not [40]. The two studies [47,50] which reported a significant association were rated as high quality by the CASP review, whilst the study which did not report an effect [40], was considered to be of medium quality.

Covariates

Parental ADHD

Parental ADHD was measured in only two out of fourteen studies in the current review; Breaux and Harvey, and Wolford et al. [42,51]. Breaux and Harvey [42], reported that when parental ADHD was accounted for within depression models, the effect of maternal depression on child ADHD was no longer significant. Wolford et al. [51], in contrast reported a significant effect, controlling for the presence of maternal ADHD symptoms. There were several differences in the

study design which may account for these differences including, the period of maternal depression focussed on in the study, how maternal symptoms of ADHD were operationalised within the design, and the incorporation of paternal ADHD symptoms in the Breaux and Harvey study.

Maternal and Paternal Psychopathology

Understanding parental psychopathology in this context is pivotal to understanding the relationship between maternal depression and ADHD, as it allows for the controlling of shared genetic risk factors. Three studies included broader measures of maternal psychopathology, other than ADHD or depression [44,49,50]. Van-Battenberg et al. [49] reported measures of maternal and paternal anxiety and depression. Models were adjusted for paternal depression or anxiety during pregnancy, and later depression and anxiety in both Generation R and ALSPAC cohorts. Only in the ALSPAC cohort did a significant association between maternal depression and later ADHD remain. Despite this finding, there were no significant differences between maternal and paternal depression and anxiety on child ADHD, offering little support to hypothesized intrauterine mechanisms.

Galera et al. and Vergunst et al. [44,50], both utilising the Quebec Longitudinal Study of Child Development cohort, reported significant associations between maternal depression and high symptom trajectories for hyperactivity-impulsivity and inattention, controlling for parental self-reported conduct disorder or antisocial personality disorder in childhood. Parental antisocial behaviour in adolescents was found to be a significant related to high trajectories in Galera et al. [44], but not Vergunst et al. [50], who examined trajectories over a longer time period. The difference between the results in these two studies implies that the effect of parental antisocial behaviour on ADHD symptoms may diminish over time.

A further study recorded paternal depression and anxiety, but did not include these factors as a covariate when examining the relationship between maternal depression and child psychopathology [39].

Parenting

Six studies examined parenting, either as a main outcome variable or a covariate [40,43,44,48,45,50]. Ashman et al. [40], incorporated parenting stress as a measure within the

contextual risk index. This index significantly mediated the effect of maternal depression on externalising-ADHD.

Galera et al. and Vergunst et al. [50,44], examined coercive parenting, overprotection, self-efficacy, and parental impact. Both coercive parenting, and overprotection were not significantly related to high hyperactivity-impulsivity, or inattention in multivariate models. In contrast Romano et al. [48], examined parenting practices, focussing on positive interaction and parental hostility. Hostile parenting independently increased the risk of high persistent hyperactivity significantly, alongside prenatal smoking and maternal depression. Similarly, Jusiene et al. [45], examined maternal self-efficacy. High self-reported maternal self-efficacy was associated with high attention behaviour regulation, alongside maternal depressiveness. Lastly, Choenni et al. [43], utilised parenting as a main outcome measure, but did not report any significant correlations between maternal depression and positive or negative discipline, or maternal sensitivity. Maternal sensitivity, but not maternal negative and positive discipline, was reported to be associated with later ADHD symptoms, controlling for concurrent attention, executive function and ODD symptoms.

Discussion

This is the first systematic review to consider longitudinal studies specifically in the area of maternal depression and later ADHD. The review sought to synthesise the literature examining the relationship between maternal depression and later offspring ADHD, to assess whether there are differential effects for prenatal and postnatal depression, and to consider the potential mediating or moderating factors which may impact upon any reported relationships. Largely these aims were achieved by the present review.

The findings in relation to these aims will now be summarised in turn. For the first aim, the majority of studies (N=11) reported a significant association of maternal depression (prenatal and/or postnatal) with offspring ADHD utilising longitudinal designs, and these effects remained when controlling for relevant confounding variables. Study quality was generally of medium to high quality, with only one study rated as low quality based on the CASP criteria [38]. Where a significant effect of maternal depression was not found (N=3), there was no evidence to suggest that these studies

were of a higher quality than those studies which did find effects (for the majority of these studies, the lack of findings could potentially be explained by a lack of power to detect significant effects). Hence, this systematic review is a first and important synthesis of the literature which offers some evidence of a potentially causal effect of maternal depression on child ADHD.

There was considerable diversity in the type of study covered in the review, with several studies focussed on trajectory modelling designs involving a significant number of covariates [40,44,45,48,50], and several others utilising large cohort models such as ALSPAC [41,47,49]. This variety of designs enabled the review to consider the relationship between maternal depression and the development of ADHD symptoms in offspring over time, as well as utilising high powered designs, which were representative of local populations. A notable strength of the papers reviewed studying depression occurring post-birth, was the consideration of child temperament or early life ADHD symptoms as a covariate. This considerable confound did not appear to alter the relationship between maternal depression and ADHD, indicating that the relationship between maternal depression and later ADHD is not simply as a result of the child's challenging behaviour causing increases in maternal depression. Hence, this review builds on the recent meta-analysis and systematic review by Cheung et al. (2016) [30], which reported cross-sectional co-occurrence rates of depression in children with ADHD.

The present review provided evidence for the association of maternal depression across both the prenatal and postnatal periods, with ADHD. Regarding the second aim, the review did not find conclusive evidence to suggest that either prenatal or postnatal depression is a stronger predictor of later ADHD. Few studies controlled for both prenatal and postnatal depression. Some studies found that both prenatal depression, and depression occurring post-birth were independent predictors of later ADHD [46,51]. Another study reported a relationship for prenatal depression controlling for postnatal depression, but did not report whether postnatal depression was an independent predictor, controlling for depression occurring in the prenatal period [47]. The largest study assessed by the review by Van Batenburg-Eddes et al. [49] found that in the Generation R but not ALSPAC cohort, the effect of prenatal depression was significantly mediated by postnatal symptoms of depression and anxiety, but

did not report if postnatal depression was an independent predictor separately. It is important to note that whilst some of the potential causal mechanisms behind maternal depression and ADHD associations may be equally shared across prenatal depression and postnatal depression (e.g. shared genetic risk of depression or psychopathology), other mechanisms may be unique to each period of depression. Evidence from the prenatal stress literature suggests that prenatal stress can have significant effects upon a wide variety of areas including changes in neural pathways between the amygdala and prefrontal cortex, and altered hypothalamo-pituitary-adrenal (HPA) axis functioning [53]. Pathways in postnatal depression in contrast may be derived through other factors, such as increased exposure to contextual risk factors [40]. The complexity of these shared and independent factors in the potential pathways between prenatal and postnatal depression and later child psychopathology, may explain the somewhat mixed findings regarding the timing of maternal depression in contributing to the development of ADHD.

In relation to the third aim of the review, a number of factors were included as potential confounds or within mediation/moderation models. Parental ADHD was examined in two studies [42,51], with Breaux and Harvey [42] reporting a significantly moderated the relationship between maternal depression and ADHD, whilst the relationship remained significant in Wolford et al. [51] controlling for parental ADHD. Parental psychopathology other than ADHD or depression was reported in several studies. Notably parental history of conduct disorder or antisocial personality disorder in childhood did not account for the relationship between maternal depression and high symptom trajectories for hyperactivity-impulsivity and inattention in two studies [44,50]. Parenting practices were a further frequent covariate, but were not found to be significantly related to ADHD trajectories [44,50], though Jusiene et al. [45], reported that maternal self-efficacy was associated with attention behaviour regulation.

Gaps in the Evidence Base Uncovered by this Review

There were several areas of methodology for which several significant limitations were apparent, which weigh against this review offering full support of maternal depression as a causal factor in the development of ADHD. Firstly, the majority of the studies were based solely on

informant measures. Only three studies out of fourteen utilised teacher reports to measure ADHD symptoms, and only one design included self-report, raising the risk of bias. Despite the common limitation of many studies relying solely on mothers as informants, in the three studies that utilised other informants, a significant effect between maternal depression and later ADHD was found in two out of three of these studies [47,50]. Indicating that this limitation does not significantly detract from the conclusions of this review. Some literature has proposed that mothers with depression may be more prone to appraising their children's behaviour more negatively, compared to mothers who are not depressed. The Depression Distortion Hypothesis, proposed by Richters and Pellegrini [54], predicts that parental depression related to cognitive distortions may alter appraisals of child behaviour. Madsen et al. [55], reported that not only was the association between maternal depression and ADHD stronger when reported by mothers rather than teachers, the Strength and Difficulties Questionnaire (SDQ) was almost twice as likely to predict future ADHD difficulties, in the children of mothers who experienced depression. Mechanistic studies have also reported a relationship between maternal psychopathology and higher ratings of inattention. Haack et al. [56] found that this relationship is mediated in part by higher levels of cognitive errors, in parents with depression and ADHD symptoms, resulting in higher ratings of child ADHD by parents.

Secondly, within the studies identified during the review, there was a notable paucity of studies which controlled for maternal, or paternal ADHD symptoms. This limitation is significant as the absence of this factor as a covariate limits the inferences which may be made, as there is substantial potential for reported associations to be confounded by residual shared genetic risk of ADHD between mothers and children, with ADHD heritability estimated to be as high as 74% [10]. Furthermore, there is evidence that adults with ADHD experience a high level of comorbidity with depression, with 35 -50% of adults with ADHD experiencing at least one episode of depression during their lifetime [57]. In the studies reviewed, there was also a lack of assessment of other facets of parental psychopathology, which may also account for externalising disorders, such as history of conduct disorder or current antisocial personality disorder, both of which are highly comorbid with ADHD and therefore also possibly share genetic risk [58,59]. The lack of regular inclusion of these

covariates in the studies reviewed, means that we are unable to draw strong conclusions from the literature about whether maternal depression is an "environmental" causal predictor or ADHD, or whether the significant observed relationships between maternal depression and ADHD are due to unmeasured genetic risk. The reviewed research also does not deal with the likely relevance of gene-environment interactions concerning the impact of a child experiencing a depressed mother on any existent genetic vulnerability, and the extent to which they develop symptoms of ADHD.

Thirdly, samples within the current review were all drawn from high income countries as defined by the World Bank [52], and were all based within Europe or North America, with the exception of one study from Israel [39]. Similarly, within these samples, cohorts tended to be drawn from populations of higher SES, educational background and married or co-habiting couples. There are therefore limitations as to the generalisability of the studies due to both country and demographic factors.

Fourthly, whilst the majority of studies reported a longitudinal association between maternal depression and offspring ADHD, the mechanisms behind this association were not clearly delineated. The most commonly discussed mechanism of risk was the transmission of risk for ADHD through intrauterine mechanisms. Several potential mechanisms were not comprehensively covered. Notably parenting practice was not significantly considered within the literature which is likely to significantly interact with maternal depression in the development of a child's externalising behaviour difficulties, including ADHD [60]. Moreover, no studies in the current review controlled for attachment, or considered attachment as a mediator, despite evidence of an association between attachment styles and externalising behaviour within the existing literature [26]. Similarly recent literature has highlighted ACEs as a significant factor, where a dose-response relationship has been reported between child ACEs and ADHD symptoms [20] . Whilst maternal depression would be encompassed within the definition of an ACE, it is possible that maternal depression may confer greater risk of exposure to other ACEs and in turn increase the risk of offspring ADHD.

Implications for Clinical Practice

Although not fully conclusive due to the gaps in the evidence base discussed, this review certainly raises the possibility that intervening to treat maternal depression across both pre and postnatal periods, could be a relevant factor in preventing the development of ADHD in offspring. As outlined in the introduction, ADHD has considerable costs to both individuals and society. This review therefore offers support to the importance of funding perinatal mental health support services, which are often very under-resourced even in developed nations [61]. Therefore, there is a significant argument to be made that early interventions in maternal depression are important from both a health economic and public health standpoint, with significant evidence for interventions which can effectively treat depression present within the existing literature [62]. Furthermore, the review would also support the integration of parental mental health interventions into neurodevelopmental services, of which there is increasing evidence of efficacy [63-65].

Future Research Directions

Future research should build upon several of the limitations of the current literature identified by the current review. As highlighted by Madsen et al. [55], measurement of child ADHD should draw on measures taken from multiple informants, to minimise potential bias from maternal reporting in this context, which may be influenced by maternal depression [56] . The review also highlights that future research into the link between maternal depression and ADHD should consider maternal ADHD symptoms as a potential confounding variable. Similarly, as highlighted previously, few studies included measures of paternal psychopathology. Adding these covariates, would significantly improve the generalisability of findings, and allow researchers to mitigate confounding genetic factors. Generalisability of findings would be enhanced by the use of non-western samples, in conjunction with cohorts which are more socioeconomically representative. Knowledge would also be expanded by moving away from large scale cohort designs to mechanistic designs, which look at the underlying mechanisms which may explain the relationship between maternal depression and ADHD, such as attachment, or parenting. To assess more accurately how the reported association between maternal depression and ADHD is derived over time, as the current theories in this area are limited.

Strengths and Limitations of the Present Review

The present review has a number of strengths and limitations. A notable strength of the paper was the focus on longitudinal designs which considered important confounding factors such as early life temperament or early ADHD symptoms. The choice of focus provides confidence that the findings are not significantly confounded by individual child risk factors. The criteria of the review to include only studies which utilise validated measures of ADHD and depression, further strengthens the validity of the study, ensuring that there are accurate measures, and reducing the risk of bias from studies based upon unvalidated tools or unstructured clinical judgements. The review can also be considered to be systematic, in contrast to previous reviews in the literature which have displayed flawed methodology as discussed previously [35].

There are nevertheless several limitations of the review. Due to the heterogeneity of studies reviewed, a meta-analysis was not possible. Whilst the current review focussed upon papers utilising validated measures to assess maternal depression or ADHD, this criterion meant that several large scale population based studies using clinical coding designs were ruled out. Secondly, the review focussed narrowly upon maternal depression and did not consider studies which examined solely the relationship between paternal depression and ADHD, limiting the scope of implications of the review.

A large proportion of the literature also focussed solely upon wider concepts than ADHD, namely the concept of externalising disorders, which ADHD is encompassed within, and are highly correlated with ADHD. This meant that a portion of the literature in this area was omitted. Relatedly, depression is highly correlated with other forms of psychopathology, most notably anxiety disorders. [66] but this was not considered in the present review. It is not clear from the review how other forms of maternal psychopathology relate to offspring ADHD.

Conclusion:

This review provides important initial evidence suggesting a partial causal link between maternal depression and ADHD assessed across both prenatal and postnatal periods, which remained when controlling for symptoms of earlier ADHD and child temperament. There were however several

factors which restrict the generalisability of results, most notably the predominance of reliance upon potentially biased maternal reports of ADHD symptoms, and limited consideration in the literature of potential genetic confounding factors. The study however provides sufficient justification for health care commissioners to consider the potential benefit of effective mental health care treatment for mothers, with downstream effects upon child mental health.

Declaration

The present study was prepared as part of the lead authors Doctorate of Clinical Psychology (DClinPsy) thesis. The author reports no conflicts of interest to declare.

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Cool and hot executive function in relation to the domains of conduct problems and inattention/hyperactivity in young children.

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Cool and hot executive function in relation to the domains of conduct problems and inattention/hyperactivity in young children.

Abstract

Executive function has been conceptualised as involving both hot (reward-related, emotionally driven) and cool (non-reward related) components. Research, mainly conducted in mid childhood and adolescence, has linked this dual account to symptoms of Attention Deficit/Hyperactivity Disorder (ADHD) and Oppositional Defiant Disorder/Conduct Disorder (ODD/CD) , with evidence suggesting that ADHD is more consistently associated with cool executive function difficulties, whereas ODD/CD is more consistently associated with hot executive function difficulties. However, little research has been conducted in early childhood when these externalising symptoms are emerging, and understanding the underlying neuropsychological components of emerging behavioural problems is an important area of research, which may inform preventative interventions. The present study assessed cool and hot executive function in 132 children, ranging from 4 – 8 years of age, who were also assessed for symptoms of inattention-hyperactivity and conduct problems. Two cool executive function tasks assessed working memory and cognitive flexibility, whilst two hot executive function tasks assessed reward-related decision-making. Counter to our hypotheses, no differential associations were found between inattention-hyperactivity symptoms and conduct problem symptoms. Specifically, there was no association found between either inattention-hyperactivity symptoms or conduct problems and performance on cool executive function tasks. Furthermore, both inattention-hyperactivity and conduct problems were associated with riskier decision-making on hot executive function tasks. Analyses revealed a significant relationship between inattention-hyperactivity and conduct problem comorbidity and impaired performance on hot executive function; including increased risk taking, poorer learning, and diminished sensitivity to negative reinforcement.

Keywords (4) Conduct Disorder, ADHD, ODD, hot and cool executive function,

Conduct problems in childhood, together with highly comorbid attention hyperactivity/inattention difficulties, have been demonstrated to be predictive of later life emotional and behavioural problems, mental health difficulties, criminal behaviour, and substance misuse [1-3]. Understanding the underlying neuropsychological processes involved in these different types of externalising behaviours at an early age is crucial in order to inform both educational and health-related preventative interventions [4,5].

"Conduct problems" refers to angry or irritable moods, argumentative or defiant behaviour and norm violating behaviours in children and adolescents which subsume the DSM-V diagnostic categories of Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD) [6]. CD often follows earlier ODD behaviours and can be a precursor to Antisocial Personality Disorder in adulthood [7]. Understanding the underlying neuropsychological processes involved in the development of conduct problems is complex given its high rates of comorbidity with Attention Deficit/Hyperactivity Disorder (ADHD). ADHD is characterised by a persistent pattern of difficulties with inattention, hyperactivity and impulsiveness [6]; however ADHD symptoms in themselves are not "antisocial", and many children with ADHD do not go on to develop antisocial/criminal behaviour patterns. There is evidence of a substantial co-morbidity between ADHD and ODD/CD in childhood with estimates ranging between 22% and 52% of children with ADHD also meeting the criteria for ODD [8-10]. Research examining the long term outcomes of externalising disorders in childhood suggest that symptoms of ODD and CD, but not ADHD are independently linked to difficulties with antisocial behaviour in adulthood [11,12]. Therefore, understanding the underlying factors which underlie ODD/CD specifically in childhood is pivotal in seeking to understand how to intervene in the development of antisocial and related criminal behaviour in adulthood.

Of relevance to understanding externalising behaviours marked by impulsive behaviours that deviate from social norms, is the concept of executive function (EF), which refers to a collection of processes that enable higher order processing and encompass skills such as inhibition, working memory and cognitive flexibility [13]. A wide variety of literature has examined the role executive function has in underpinning the difficulties experienced in childhood externalising behaviour, including both

conduct problems (predominantly research concerning ODD/CD samples) and inattention/hyperactivity (predominantly research concerning ADHD samples).

Increasingly, a key research topic regarding the underlying processes involved in the development of conduct problems is the distinction between "cool" and "hot" executive function. Zelazo and Carlson [14] defined "hot" executive function as processes which involve top-down control to facilitate accurate problem solving and decision making in the context of situations involving a high degree of affective and motivational demands, typically involving regulating responses under conditions of reward or loss. They defined "cool" executive function as facilitating cognitive regulation under non-reward conditions, involving slow, deliberative processing and reasoning. Functional neuroimaging and lesion studies have demonstrated differential patterns of neural activation associated with cool and hot executive function tasks, such that cool executive function is associated with frontal-striatal regions, and hot executive function with orbitofrontal-limbic regions [15].

Within the ODD/CD literature, ODD has been conceptualised as being underpinned by two pathways of executive dysfunction, a cold deficit in response inhibition and a hot deficit of emotion regulation and processing [16]. Noordeemer and colleagues [17], reviewed structural and functional MRI studies examining ODD/CD, revealing structural and functional changes in both the the amygdala and anterior cingulate regions associated with hot EF. Furthermore, there was also evidence that abnormalities in the amygdala distinguished ODD/CD from ADHD controls. Impairments in other regions associated with cold EF such as the prefrontal cortex, were also reported, providing support to Blair's (2005) integrated model [16]. A recent meta-analysis of fMRI studies in disruptive behaviour disorders by Algeria et al. [18], described consistent patterns of dysfunction in rostro-dorsomedial, fronto-cingulate, and ventral-striatal regions, all of which are involved in reward-related decision making. Individuals with ODD have also been noted to have altered psychophysiological responses in response to reward-related decision making paradigms, when compared with controls. [19].

Cool Executive Function and ADHD and ODD/CD

There is consistent evidence that children with ADHD have wide ranging deficits in executive function performance compared with healthy controls. Willcutt and colleagues (2005), in their meta-analytic review, reported consistent deficits of response inhibition, planning, working memory and vigilance in those with ADHD compared to healthy control children [20]. Meta-analyses of executive function in adults with ADHD have also indicated a consistent pattern of executive function deficits in inhibition, as well as further deficits in verbal fluency and set-shifting, suggesting deficits persist into adulthood [21]. However, many of these studies have not considered differential executive function performance in terms of whether the child also exhibits conduct problems on top of general inattention or hyperactive behaviours. In studies that have considered the independent relationships of ADHD and conduct problems (including ODD/CD) with cool executive function, some studies have found that executive functioning difficulties in areas such as inhibition, planning and verbal fluency are largely accounted for by ADHD [22,23] and there appears to be no additive effects of ODD/CD in children with ADHD on executive function performance [24]. Some literature has suggested individuals with ODD/CD demonstrate performance equivalent to healthy controls [25]. Other studies examining executive function in ADHD and ODD/CD have reported an additive effect of ODD/CD symptoms compared with individuals with ADHD alone; for example Noordermeer et al. [26] reported that adolescents with ADHD and ODD exhibited impairment in more domains contrasted with those with ADHD only. Poor performance in set shifting has also been associated with ADHD comorbid with CD [27], but not in ADHD samples without comorbid CD [28]. Whilst working memory has been identified as an area of dysfunction within ADHD samples separate from ODD/CD [22,29], recent studies have highlighted impaired working memory as a characteristic independently related to both ADHD and ODD/CD [26,30,31].

Overall, there is a well-documented basis for executive functioning deficits being found within ADHD populations across multiple cool EF processes. However, there is little evidence for cool EF deficits being found in conduct problems (including ODD/CD) independently of ADHD in most cool EF areas, with the exception of there being some evidence that ADHD and ODD/CD are

independently associated with working memory. Furthermore, the combination of conduct problems and ADHD is potentially associated with more cool executive function deficits.

Hot Executive Function and ADHD and ODD/CD

Hot executive function has been relatively less researched than cool executive function in externalising behaviour disorders. Current evidence suggests that hot executive function might play a crucial role in the development of conduct problems independently of inattention-hyperactivity. Some research [32,33] has demonstrated that neuropsychological performance on hot executive function tasks is independently related to symptoms of ODD/CD when controlling for ADHD symptoms, suggesting that hot executive function may play an important role in the aetiology of antisocial behaviour. Hobson and colleagues [32] found in 93 children aged between 10 and 17 (mean age = 13), a differential effect across ADHD and ODD/CD symptoms, whereby ODD/CD symptoms, but not ADHD symptoms, were independently associated with risky decision making as measured by the Iowa Gambling Task. Similarly van Goozen and colleagues [33], reported that in a sample of 77 children aged 7 to 12 years old, a motivational inhibition task was able to distinguish 77% of participants with ODD from normal controls.

In contrast Antonini and colleagues [34], in a study of 130 children aged between seven to twelve years old recruited from a specialist ADHD clinic, found no differences between; ADHD; ADHD and ODD/CD; and control groups across two hot executive function tasks (a gambling task and a delay discounting measure), with the number of ADHD or ODD symptoms also not associated with task performance. In contrast ADHD, but not ODD, symptoms were predictive of scores on cool executive function tasks.

Although the neuropsychological studies of hot executive function in conduct problems independently of ADHD are not fully conclusive, there is some evidence from brain imaging studies of differential patterns of brain activation in ODD/CD and ADHD samples. A recent review of imaging studies in those with ODD/CD and ADHD provided evidence that areas responsible for hot executive functioning (including limbic areas) are less activated in ODD/CD independently of ADHD

[15]. Impairments in other regions associated with cool EF such as the prefrontal cortex, were also implicated in ODD/CD, but to a lesser extent compared to ADHD.

Cool and Hot Executive Function in Younger Samples:

The majority of research discussed above has been conducted on samples of children who meet diagnostic criteria for ADHD and ODD/CD, and as such ages of participants tend to be older children with established difficulties. However, very little research has been conducted on executive function in younger samples at risk of developing ADHD and ODD/CD. Understanding the underlying neuropsychological constructs which relate to different patterns of externalising behaviour manifestations at an early age, may enable the development of effective targeted early interventions to prevent difficulties developing in the future. Of the studies that have exclusively considered cool and hot executive function in conduct problems and inattention-hyperactivity in early childhood, the findings in this area have been mixed. A meta-analysis by Schoemaker et al. [35] concerning cool executive function in children aged between 3 and 6 revealed deficits in working memory, inhibition and cognitive flexibility across both ADHD and conduct problem groups. There was some evidence that this effect was moderated by age, with a larger effect size for older children and referred/selected samples in comparison with community samples. A further study by Schoemaker and colleagues [36] examined a sample of children aged between 3.5 – 5.5 years of age with a diagnosis of ADHD or ODD/CD. Whilst both groups displayed poor cognitive inhibition compared with controls, the effect for ODD/CD children was derived largely through tasks involving tangible rewards, therefore reflecting difficulties in reward-related decision making, not evident in ADHD children. Similarly a longitudinal study by Griffith et al. [37], in a community based sample examined neuropsychological predictors of ADHD and ODD/CD symptoms in young children (assessed at age 3 to 4). A measure of hot executive function (delay aversion) predicted oppositional behaviour at school age, controlling for symptoms of ADHD. However, in a community based sample of 125 preschool children with ADHD (aged between 4 – 5 years of age) recruited from childcare centres, hot executive functioning was found to be equally relevant to ADHD symptoms, when assessed by three delayed gratification paradigms which were age corrected [38].

The above review highlights that further research is required to consider whether the differential profile of cool and hot executive function deficits in conduct problems and inattention-hyperactivity is found in younger samples of children who are at risk for developing diagnostic levels of problems. The present study focused upon a young sample of children (aged 4-8) whose teachers were concerned about their emotional or behavioural development, to investigate the associations of hot and cool executive function with conduct problem and inattention-hyperactivity symptoms.

Two cool executive function tasks (measuring cognitive flexibility and working memory) and two hot executive function measures (measuring reward-related risky decision-making) were chosen which have been adapted for use for younger children. Conduct problems and inattention-hyperactivity symptoms were assessed by the Strength and Difficulties Questionnaire (SDQ), which is a common parent and teacher measure for screening for childhood emotional and behavioural difficulties. Given the balance of the evidence from neuropsychological and imaging studies, in predominantly older children/adolescents, indicating cool executive function is more closely related to ADHD than ODD/CD, and that hot executive function is more closely related to ODD/CD than ADHD, we made the following hypotheses in our sample of younger children: 1) Performance on cool executive function tasks would be more strongly associated with inattention-hyperactivity symptoms than conduct problem symptoms; and 2) Performance on hot executive function tasks would be more strongly associated with conduct problem symptoms than inattention-hyperactivity symptoms.

Method

Participants (N=132, males = 94) were aged between 4 and 8 years of age (mean = 6.09 SD = 1.05) and were referred to the Neurodevelopmental Assessment Unit (NDAU) at Cardiff University by their teachers due to social, emotional or behavioural concerns. The NDAU is primarily a research study, but also provides feedback in the form of a report (overseen by an Educational Psychologist) about the results of some selected normed tasks; the aim of the report is to help inform teachers of potential intervention strategies depending on the child's profile. The sample were largely of British Caucasian ethnicity (78.8%). Participants completed a battery comprising of multiple cognitive, emotional and

neuropsychological assessments of which the measures described here are included, over two separate sessions. The sample analysed here was comprised of the children who had a full dataset of the variables of interest in this study, from the beginning of the NDAU until December 2019. Parental measures of child psychopathology were collected at the same time participants were assessed by other researchers within the study team. The child and parent assessments were conducted by trained postgraduate researchers; the first author completed a proportion of the parent assessments but was not involved in the child assessments. Teacher measures were collected by post as they formed part of the referral pack. Ethical approval was granted for the project entitled: A Feasibility Study of a Neurodevelopmental Disorders Assessment Unit (EC.16.10.11.4592GRA5). A copy of the most recent approval (amendment) is contained in Appendix B.

Executive function measures

Cool Executive Function

Working memory was assessed utilising the backward digit span task taken from the Automated Working Memory Assessment (AMWA) [39], a validated assessment battery administered via a computer program [40]. The AMWA exhibits stability of scores assessed over time and correlates significantly with well-established measures of working memory such as the WISC-IV Working Memory Index [40]. The backward digit recall test from the AMWA battery was utilised for the present study. Participants were required to recall a number of digits which are audibly presented, and then to recall the digits in reverse order. Scores were standardised according to the age of the subject, with higher scores indicating superior working memory skills.

Cognitive flexibility was assessed using the Response Organization Objects (ROO) task taken from the Amsterdam Neuropsychological Tasks battery (ANT) [41]. The ANT battery has moderate-high test-retest reliability and has been demonstrated to report different profiles across disorders, suggesting a degree of discriminant validity [42] The ANT-ROO task is a computer-based task which is designed to assess response inhibition and cognitive flexibility. Participants were presented with randomly generated coloured circles, which appeared bilaterally of a fixation cross. Participants were informed of incorrect choices by an auditory tone and red spot displayed at the centre of the screen.

At stage 1, participants were presented with a red circle and required to click the left and right mouse buttons, to directly correspond to the side of the screen the red circle appeared on. Stage 2 introduced white circles, requiring an inverse response, whereby participants clicked the opposite side of the mouse to the side of the screen the white circle appeared on to receive a correct response. Stage 3 involved a combination of the red and white circles displayed in stages 1 and 2, which were randomly generated. Cognitive flexibility was calculated using a comparison of reaction times between stage 1 fixed compatible and stage 3: random mixed compatible and incompatible, in line with previous literature utilising the ANT-ROO task [43]. Higher reaction time scores indicated poorer cognitive flexibility. An overview of the task, including images of the task is included in Appendix C.

Hot Executive Function Measures

Hot executive function was assessed by two measures. The Hungry Donkey task [44] and the Balloon Emotional Learning Task [45] (BELT). The Hungry Donkey task, an adapted version of the Iowa Gambling Task (IGT) [46], was administered to children via computer. The Hungry Donkey task utilises the same format and a similar schedule of rewards and losses to the IGT [47] and correlates with both physiological (e.g. heart rate and skin conductance) and neurophysiological measures of reward and punishment [48,49]. Participants viewed a donkey standing in front of two doors (Door A and Door B). They were tasked with gaining the most amount of apples for the donkey by clicking on the doors. Clicking on the doors resulted in two outcomes; a gain or loss of apples. Analogous to the advantageous and disadvantageous decks of the IGT, two doors were presented to the children with different weighted probabilities of reward. Door A provided high rewards, but high losses. Providing a gain of four apples on 50% of trials and a loss of 8 – 12 apples on the other 50% of trials. Door A offered high potential gains of apples (up to 12 apples) but was significantly riskier, resulting in an overall net loss of 10 over 10 trials. Door B in contrast produced a reward of 2 apples for 50% of trials and a loss of between 1 – 3 apples in the further 50% of trials, resulting in a net gain of 10 apples, over 10 trials. Participants undertook 50 trials in total. The main outcome measure was net score (advantageous choices – disadvantageous choices) [50]. Higher scores indicated less risky decision making. An example of the task is displayed in Appendix D.

Hot executive function was further assessed using the Balloon Emotional Learning Task (BELT) [45], a modified version of the Balloon Analogue Risk Task [51]. The BELT has been demonstrated to be associated with measures of sensation seeking in a sample of young adults [45]. The task was administered by computer. Participants were required to press a key which inflated a balloon presented on a screen. There were three different types of balloons signified by different colours. Each balloon varied in the number of pumps which were required before the balloon burst. Two balloons were stable, meaning that the same number of pumps were required to burst them, with one stable balloon bursting after 7 pumps (certain-short) and the second after 19 pumps (certain-long). The third balloon (uncertain) exploded at 7, 13 or 19 pumps, equally across the task. There were 27 trials in total and balloon type was equally distributed. Points were awarded for the number of pumps a participant was able to administer without the balloon popping. A points meter was displayed on the screen. Participants are able to choose to stop inflating the balloon and to bank the points, or to risk losing the points if they continue to inflate the balloon to explosion. Participants were not informed that the colour of balloon affected the tendency of the balloon to burst. Three outcome measures were derived from the task: i) pumps as a measure of general risk taking (higher pumps indicate greater risky decision-making); ii) points as a measure of outcome (higher points indicate more successful decision-making); and iii) explosions as a measure of untampered risk taking (higher explosions indicate more decision-making errors). An overview of balloon conditions and screenshots from the task are reported in Appendix E.

Behavioural Measures

The Strength and Difficulties Questionnaire (SDQ) [52] is a parental and teacher based assessment tool which enables assessment of internalising and externalising difficulties within children. The questionnaire is divided into five subscales: emotional problems; hyperactivity; conduct problems; peer problems; and prosocial scales. Due to the specific hypotheses and focus of this study, only the hyperactivity and conduct problems subscales were analysed. It is worth noting that the "hyperactivity" scale contains both inattention and hyperactivity items. Both parent and teacher forms were completed during the course of the study. Parent and teacher SDQ scores correlated significantly

(conduct problems: r = 0.36 ; hyperactivity; r = 0.30). In order to reduce potential bias and also to take into account pervasiveness of symptoms, parent and teacher scores were combined to produce a mean score for both hyperactivity and conduct problems subscales. A copy of the parent SDQ is reported in Appendix F.

Analysis Plan

Where the data utilised did not meet the assumptions for the usage of parametric statistics and was not normally distributed, transformations were applied. Data was defined as abnormal when the skewness statistic was greater than two times the standard error. Three transformations were utilised where appropriate; logarithmic; square root; and reciprocal. The transformation which most significantly reduced the skewness was retained for later analyses. This resulted in square root transformations for: SDQ mean combined hyperactivity; SDQ mean combined conduct problems; BELT points; and BELT explosions. Across all variables, there were only two cases which were outliers (defined as over 3 standard deviations from the mean). These cases were altered so their scores fell exactly at three standard deviations from the mean [53].

Bivariate Pearson correlations were utilised to assess the relationships between demographic variables (age, gender), behavioural measures and neuropsychological measures. Where hyperactivity and conduct measures both correlated significantly with a particular executive function measure, regression analyses were performed to examine the relative strength of SDQ conduct problems and SDQ hyperactivity as predictors. Further analyses were conducted to investigate learning across both hot EF tasks (described below).

Results

Descriptive statistics for the sample are reported in Table 1, for both psychopathology and neuropsychological variables. Bivariate correlations between neuropsychological tasks are reported in Table 2.

Table 1.

Descriptive statistics of measures

Measures	Mean (SD)
SDQ Hyperactivity (Parent)	7.8 **(2.3)**
SDQ Hyperactivity (Teacher)	7.3 **(2.6)**
SDQ Conduct (Parent)	4.2 **(2.6)**
SDQ Conduct (Teacher)	3.4 **(2.6)**
Combined SDQ Hyperactivity	7.5 **(2.0)**
Combined SDQ Conduct	3.8 **(2.1)**
AWMA: Backward Digit Recall (standard score)	100.84 **(16.09)**
ANT ROO - Cognitive Flexibility (RT)	594.7 **(336.9)**
BELT Pumps	160.5 **(53.9)**
BELT Explosions	6.22 **(4.8)**
BELT Points	108.9 **(26.1)**
Hungry Donkey Net Score	12.8 **(16.5)**

Table 2.

Bivariate Pearson Correlations of Executive Function Measures

Measure	1	2	3	4	5	6	7	8
1. Age	1	.088	.068	-.075	.372**	-.099	-.180*	-.192*
2. Gender		1	-.132	-.162	.010	-.077	-.018	.177*
3. BELT Pumps			1	.909**	.453**	-0.093	-.185*	-0.139
4. BELT Explosions				1	0.084	-0.068	-0.163	-0.069
5. BELT Points					1	-0.128	-0.132	-.225*
6.Hungry Donkey Net Score						1	0.011	0.04
7.AWMA							1	0.059
8. ANT-ROO								1

* p <.05
** p <.001

BELT = Balloon Emotional Learning Task
AMWA = Automated Working Memory Assessment (standard score)
ANT-ROO = Amsterdam Neuropsychological Tasks, Response Organization Objects

Correlations indicated that older age was related to improved performance in three out of the four executive function tasks (with the exception of the Hungry Donkey task). Boys and girls did not perform differently on the tasks with the exception of the ANT-ROO where boys had greater cognitive flexibility than girls. Counter to expectations, there was no correlation between the two cool executive function tasks, or the two hot executive function tasks. BELT points and BELT explosions did not correlate, indicating that they are assessing different constructs. There were however some significant correlations reported across hot and cool domains. Specifically, riskier decision-making as measured by BELT pumps was related to poorer working memory as measured by the AMWA. Improved decision-making (as measured by BELT Points) was significantly correlated with poorer cognitive flexibility as assessed by the ANT-ROO.

Hypothesis 1.

Table 3.

Bivariate Pearson correlations with cool neuropsychological measures and hyperactivity and conduct symptoms

	AWMA: Backward Digit Recall, standard score	ANT-ROO Cognitive Flexibility RT
SDQ Hyperactivity Combined	-0.094	-0.096
SDQ Conduct Combined	-0.047	-0.040

* $p < .05$

There were no significant correlations reported with either hyperactivity or conduct symptoms and cool executive function measures, counter to our hypothesis.

Hypothesis 2.

Hot Executive Function

To test hypothesis 2, that performance on hot executive function tasks would be more strongly associated with conduct problem symptoms than inattention-hyperactivity symptoms, bivariate Pearson correlations with hyperactivity and conduct symptoms and hot executive function measures were conducted. The results are displayed in Table 4.

Table 4.

Bivariate Pearson correlations with hot executive function measures and hyperactivity and conduct symptoms

	BELT Pumps	BELT Explosions	BELT Points	Hungry Donkey Net Score
SDQ Hyperactivity Combined	0.178*	0.258**	-0.021	-0.030
SDQ Conduct Combined	0.186*	0.246**	-0.010	-0.037

* p <.05
** p <.001

BELT = Balloon Emotional Learning Task

Both hyperactivity and conduct symptom subscales did not correlate with performance on the Hungry Donkey task. However, both hyperactivity and conduct symptoms positively correlated with BELT explosions and BELT pumps. A regression analysis was therefore employed to understand the independent contributions of hyperactivity and conduct with BELT explosions and BELT pumps. A model-based approach was utilised to mitigate the high correlation between hyperactivity and conduct symptoms, the strength of which could potentially violate the assumption of non-collinearity of a multiple regression approach. In both models, age was entered in the first block as a control variable given earlier findings that age was related to overall improved performance on the BELT task (albeit in terms of BELT Points only). The second and third blocks were counterbalanced across models. In

the first model SDQ combined conduct symptoms subscale score was entered in the second block, whilst SDQ hyperactivity combined subscale score were entered in the third block. In the second model SDQ combined hyperactivity subscale score was entered in the second block, whilst SDQ conduct combined subscale score was entered in the third block. The regression analysis for BELT pumps is reported in Table 5, and the analysis for BELT explosions in Table 6.

Table 5.

Regression analysis of hyperactivity and conduct symptoms as predictors for BELT Pumps

		R	R Square	Sig. F Change
Model 1				
Block 1:	Age	0.068	0.005	0.439
Block 2:	Age, SDQ Conduct	0.192	0.037	0.040*
Block 3:	Age, SDQ Conduct, SDQ Hyperactivity	0.212	0.045	0.308
Model 2				
Block 1:	Age	0.068	0.005	0.439
Block 2:	Age, SDQ Hyperactivity	0.180	0.033	0.056
Block 3:	Age, SDQ Hyperactivity, SDQ Conduct	0.212	0.045	0.204

* $p < .05$

Within model 1, when age was entered in the first block, conduct symptoms were significantly associated with BELT pumps when entered into the second block. Hyperactivity symptoms did not significantly add to the model.

Within model 2, after age was accounted for, no variables were significantly related to BELT Pumps in either block two or three in terms of producing a significant F change within the model. Hyperactivity approached but did not meet significance within model 1 ($p = 0.056$). Taken together these models suggest that conduct symptoms were the most predictive of BELT pumps, when the two models were contrasted.

Table 6.

Regression analysis of hyperactivity and conduct symptoms as predictors for BELT explosions

		R	R^2	Sig. F Change
Model 1				
Block 1:	Age	0.020	0	0.817
Block 2:	Age, SDQ Conduct	0.222	0.049	0.011*
Block 3:	Age, SDQ Conduct, SDQ Hyperactivity	0.290	0.084	0.029*
Model 2				
Block 1:	Age	0.020	0	0.817
Block 2:	Age, SDQ Hyperactivity	0.274	0.075	0.002*
Block 3:	Age, SDQ Hyperactivity, SDQ Conduct,	0.290	0.084	0.259

* $p < .05$

Regarding BELT explosions, within model 1, after age was accounted for conduct symptoms were significantly related to explosions on the BELT task. Adding hyperactivity to the model produced a significant F change within model 1, suggesting that hyperactivity was a significant independent predictor of explosions on the BELT task. Within model 2, when age was entered in the first block, hyperactivity symptoms were significantly associated with BELT explosions, conduct symptoms did not significantly add to the model, suggesting that hyperactivity symptoms were the most predictive of BELT explosions, when the two models were contrasted.

Further Exploratory Analyses of Learning in the Hot Executive Function Tasks

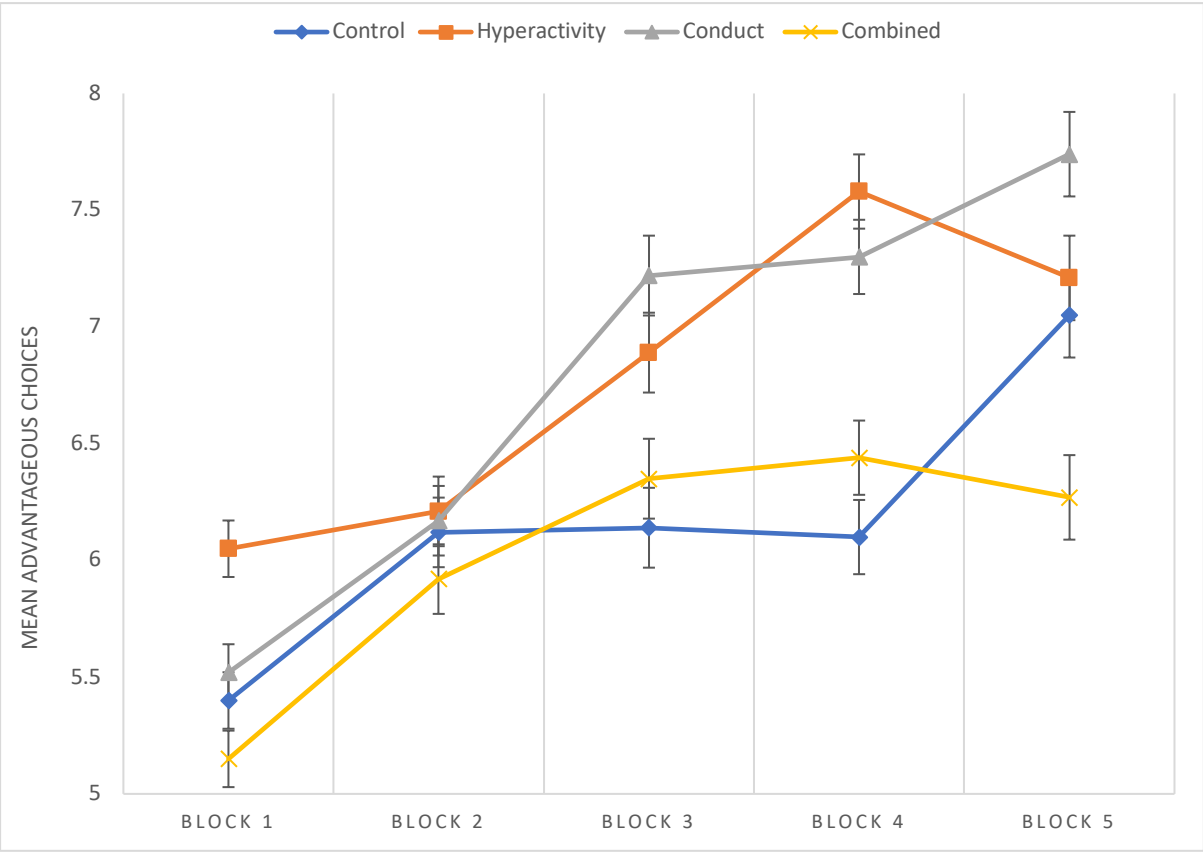
Hungry Donkey

In order to assess the effect of learning over time, further exploratory analyses were conducted on the Hungry Donkey task data. Groups based on the continuous SDQ variables were created in order to investigate learning. Specifically, the sample was divided by median combined SDQ scores on each subscale, creating four groups: 1) low hyperactivity, low conduct problems ("Control"; n = 42); 2) high hyperactivity, low conduct problems ("Hyperactivity only"; n = 19); 3) high conduct problems, low hyperactivity ("Conduct Problems only"; n = 23); and 4) high

hyperactivity, high conduct problems ("Combined"; n = 48). Prior to beginning the analysis Mauchly's test of sphericity indicated that the assumption of sphericity had been violated, $\chi^2(9) = 20.38$, $p = .016$, therefore the Greenhouser-Geisser correction was applied. There was no main effect of block (F (3.71, 471.48) = 2.25, p = .062, $\eta^2 = .01$), and there was no main interaction between block and age (F (3.71, 471.48) = 0.86, p = 0.47, $\eta^2 = .007$). There was a significant interaction between block and group (F(11.13, 471.48) = 2.00, p = .02, $\eta^2 = .045$). T-tests were used to explore the relationship between block and group, focussing on block 5 to assess learning effects. No significant effects were reported for; control and hyperactivity only groups, t(59) = -0.31, p = 0.75; control and conduct only groups, t(63) = -1.44, p = 0.15; control and combined groups, t(88) = 1.78, p = .078; hyperactivity only and conduct only t(40) = -0.86, p = 0.39; and hyperactivity only and combined groups, t(65) = 1.57, p = 0.12. A significant effect was observed only between the conduct only, and combined groups, t(69) = 2.67, p = .009). Mean advantageous choices across blocks are displayed in Figure 1.

Figure 1.

Hungry Donkey Learning across blocks, with standard error of mean displayed

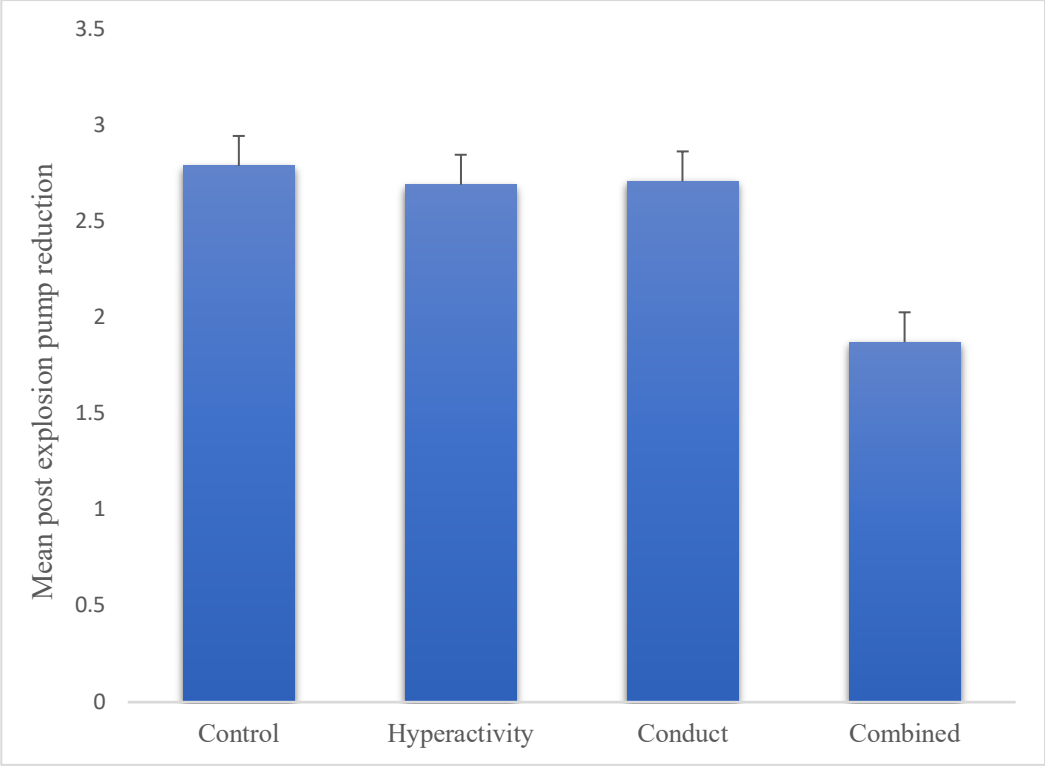


Balloon Emotional Learning Task (BELT)

A further variable produced by the BELT task is post-explosion reduction in pumps; a measure of sensitivity to negative feedback and thus indicative of using feedback to learn. This score was calculated by subtracting the mean of the number of pumps on trials where an explosion occurred, from the mean number of pumps in the trial which immediately preceded the explosion trial [54]. Positive scores indicated a reduction in the number of pumps in response to explosions; a measure of sensitivity to negative reinforcement. Eight participants were excluded from the analysis as they reported no explosions during the task. In a one-way ANOVA, a significant effect of group was found, controlling for age ($F(3) = 3.11$, $p = .029$, $\eta^2 = .07$), and as reported in Figure 2, was attributable to diminished sensitivity in the combined group.

Figure 2.

Post explosion reduction (sensitivity to negative reinforcement) across groups, with standard error



Discussion

The present study reports a novel exploration of the association of inattention-hyperactivity and conduct problem symptomatology with cool and hot executive function in a young sample of children.

The first hypothesis (that cool executive function would be more strongly related to inattention-hyperactivity and conduct problems) was not supported, in that neither hyperactivity or conduct symptoms correlated significantly with cool executive function measures (working memory or cognitive flexibility). This finding contrasts with previous literature which has reported consistent deficits in cool executive function in ADHD samples [20,21]. The lack of a significant relationship between conduct problems and cool executive function variables was partly consistent with the past literature where there have been mixed findings for ODD/CD samples, with some studies reporting an additive effect of ODD/CD symptoms [26], whilst others have not [55]. The present study diverges from several studies which have reported independent associations between both ADHD and ODD/CD with working memory deficits [26,30,31]. No further analyses were possible as regression models were not justified based upon the non-significant correlations. One possible explanation for the non-significant findings could be that in contrast to many previous studies, there was no healthy control group in the current sample, and the sample was not formally diagnosed with ODD/CD and ADHD. Hence extremes of strong and poor performance were potentially less in the current sample than previous studies, lessening the probability that significant effects would be found.

The second hypothesis (that hot executive function would be more strongly related to conduct problems than inattention-hyperactivity) was not fully supported. Initial correlational findings reported an association between both hyperactivity and conduct symptoms and BELT pumps and BELT explosions; measures of risky decision making and decision-making errors. Notably there was however no correlation between either hyperactivity or conduct symptoms on BELT points, a measure of successful decision making, or net-score on the Hungry Donkey task.

Regression analyses revealed a differential pattern of results. On the BELT pumps, only conduct symptoms were significant when age was taken into account, suggesting conduct was more

strongly related to risky decision making than hyperactivity, in partial support of the second hypothesis. Previous research has also highlighted the role of conduct symptoms in risky decision making on the BELT task. Humphreys and Lee [54] using the BART task, an early version of the BELT task, reported that risky-decision making (pumps) was more strongly associated with ODD than ADHD.

In relation to decision making errors (explosions), both hyperactivity and conduct symptoms were significantly associated with performance in the regression analyses after controlling for age. However, regression analyses indicated that only inattention-hyperactivity but not conduct problems independently added additional variance in decision making errors when controlling for conduct symptoms, which was in contrast to our second hypothesis.

Examining the sensitivity to punishment across groups split by high/low inattention-hyperactivity and conduct problems revealed a significant effect of group; with the comorbid hyperactivity and conduct problem group, displaying significantly diminished sensitivity, contrasted with the "control" group (low hyperactivity and conduct), hyperactivity only, and conduct only groups.

Whilst there was no relationship between the Hungry Donkey net score and symptom profiles, exploratory group analysis of learning across the five blocks of the task revealed significantly impaired learning in the group with high levels of hyperactivity and conduct problems. Both hyperactivity and conduct only groups displayed a significant learning effects over time, whilst the control group regained equivalence to the hyperactivity and conduct only group by the final group, suggesting that the difference seen in the final block, was largely derived through the impaired learning of the comorbid group. Counter to our hypothesis, conduct symptoms did not appear to be specifically related to impaired leaning on hot executive function tasks, with results suggesting that comorbidity of hyperactivity and conduct symptoms was the main factor related to poorer learning performance.

Taken together the findings from the hot executive functioning tasks did not support a clear differential effect of conduct and hyperactivity symptoms, in their association with hot executive

function. The evidence however supports the concept that higher levels of comorbidity are associated with poorer hot executive function performance; increased risk taking and diminished sensitivity to negative reinforcement. Our results show that in this sample of younger children, the differentiation of hyperactivity and conduct in their association with hot executive function is not as apparent as in older samples.

Strengths and Limitations

There are several limitations to the present design which should be considered when interpreting the study. First, the data presented is correlational and was taken at a single time point, therefore causality cannot be inferred. Similarly, smaller cross-sectional studies such as the one reported here do not help answer the crucial question of how the relationship between hyperactivity, conduct symptoms and executive function changes over time as children develop.

Second, given the nature of the sample (children referred by their teachers on the basis of emotional or behavioural concerns) there was a significantly high number of participants who reported high levels of hyperactivity symptoms, with the mean score of the sample equal to a score of 7.5 out of 10 when SDQ measures were combined. It is possible that this factor minimised the variance within the sample, explaining inconsistencies compared to some past studies, particularly with regard to cool executive function. However, this limitation is also a potential strength of the study, as links with hyperactivity/conduct and hot executive function were found even in a sample not based upon diagnostic criteria.

Third, counter to expectations, there were no significant correlations between the two cool executive function tasks and between the two hot executive function tasks. There were however correlations across hot and cool executive measures, with the BELT task variables correlating significantly with both working memory and cognitive flexibility variables. These findings suggest that there is some overlap in hot and cool executive functions, and that they were not clearly delineated within the present design. It is possible that this may be attributable to the young sample used in the current study. As noted by Welsh and Peterson (2014) [56], it is not uncommon within

samples of young children, for measures in both hot and cool domains, not to correlate with other measures within the hypothesised domain.

Fourth, one limitation is that the trial lengths of the hot executive function tasks utilised in this study might have been too short to enable a full examination of learning. Previous work utilising the Hungry Donkey task has also highlighted the importance of trial length in demonstrating clear learning responses over time. Cortes-Patino, Soares-Filho, and Acosta-Barreto (2017) [57], reported that up to 200 trials may be necessary to show a learning response, but typically only 100 trials are used within the literature. As the present study utilised the two door and 50 trial version, this may have been insufficiently sensitive to detect clearer effects for net-score.

Fifth, the implications of the present study in relation to the diagnoses of ODD/CD and ADHD are partially limited, as this design assessed only continuous scales relating to these diagnostic categories, and the sample was made up of many children who were at sub-diagnostic levels. However, this is still an important study in that it has revealed hot executive function difficulties related to hyperactivity and conduct problems in a young sample at risk of developing diagnostic-level difficulties.

Clinical Implications

The present study provides evidence on the relationship between hyperactivity and conduct symptoms in a sample of young children. Within the sample there was no evidence that these symptoms were related to cool executive function. There was however evidence that both hyperactivity and conduct symptoms were related to hot executive function. This finding suggests that a broader concept of externalising disorders may be more useful when considering relationships to hot executive function in young children. Poorer performance on hot executive function tasks may be a risk factor for externalising behaviour problems in young children. It may therefore be beneficial to encompass measures of hot executive function within neuropsychological assessments of young children, as there is a demonstrable link to externalising problems. This may provide additional tools for screening and early intervention in children at risk of developing ADHD or ODD/CD. Whilst there have been several studies which have examined cognitive training in children diagnosed with

ADHD, these have largely focussed upon cool executive function [58]. Recent evidence from typically developing children, has suggested attention training can improve delay gratification in young children [59], with research in adolescent samples suggesting that training using hot EF tasks may improve executive function skills, more than comparable tasks which do not have an affective component [60]. Taken together these findings provide tentative support for further intervention research in this area.

Future Research

Future research would benefit from utilising a wider battery of cool and hot executive function tasks to assess if the results found in the present study also persist across a broader range of tasks. Longitudinal research would also be useful in uncovering the relationship between ADHD and ODD/CD symptoms and hot and cool executive function over time. Finally, the use of diagnostic samples with healthy control groups would bolster the literature, as the current study is affected by the variety of psychopathology present across sample groups.

Conclusion

In summary, whilst a relationship between hot executive function and both inattention-hyperactivity and conduct symptoms was found in the present study, there was no evidence that conduct symptoms in young children were uniquely predictive of hot executive function in young children. Analyses suggested that hyperactivity is also associated with hot executive function, with comorbidity associated with poorer learning on hot executive function tasks. Counter to predictions, there was no difference in the relationship between cool executive function and hyperactivity or conduct problems, assessed by measures of working memory and cognitive flexibility. These study findings might offer support to developing early interventions that improve hot executive function in children with externalising behaviours.

Declaration

The present study was prepared as part of the lead authors Doctorate of Clinical Psychology (DClinPsy) thesis. The author reports no conflicts of interest to declare.

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Appendix A – Referencing and formatting guidance for the European Child & Adolescent Psychiatry journal.

European Child & Adolescent Psychiatry
Submission guidelines

Types of Papers

- Accepted article types: Original Contribution, Review Article, Brief Report, Letter to the Editors
- Declaration of Conflict of Interest is mandatory for all submissions. Please refer to the section "Integrity of research and reporting" in the Instructions for Authors.
- Original Papers must not exceed 20 manuscript pages of max. 32 lines each plus 8 figures, taking up no more than 3 printed pages altogether. Exceptions can be made only with the agreement of an Editor.
- Letters to the Editors and Brief Reports should not have more than 4 authors, and not contain more than 1000 words, 1 figure, 1 table (or 2 of either) and 10 references. Summary and key words are not required. Letters are subject to editorial review and may be peer-reviewed. When a submitted letter refers to an article published in a previous issue of the journal, the letter is sent to the authors of that article. Brief Reports are abbreviated research papers which should focus on a small number of principal findings. A Brief Report could be formatted as follows: Introduction, Methods, Results and Discussion.

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Manuscript Submission

Manuscript Submission

Submission of a manuscript implies: that the work described has not been published before; that it is not under consideration for publication anywhere else; that its publication has been approved by all co-authors, if any, as well as by the responsible authorities – tacitly or explicitly – at the institute where the work has been carried out. The publisher will not be held legally responsible should there be any claims for compensation.

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Please ensure you provide all relevant editable source files. Failing to submit these source files might cause unnecessary delays in the review and production process.

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Title page

Title Page

Please use this template title page for providing the following information.

The title page should include:

- The name(s) of the author(s)
- A concise and informative title
- The affiliation(s) of the author(s), i.e. institution, (department), city, (state), country
- A clear indication and an active e-mail address of the corresponding author
- If available, the 16-digit ORCID of the author(s)

If address information is provided with the affiliation(s) it will also be published.

For authors that are (temporarily) unaffiliated we will only capture their city and country of residence, not their e-mail address unless specifically requested.

Abstract

Please provide an abstract of 150 to 250 words. The abstract should not contain any undefined abbreviations or unspecified references.

For life science journals only (when applicable)

Trial registration number and date of registration

Trial registration number, date of registration followed by “retrospectively registered”

Keywords

Please provide 4 to 6 keywords which can be used for indexing purposes.

Declarations

All manuscripts must contain the following sections under the heading 'Declarations'.

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Funding (information that explains whether and by whom the research was supported)

Conflicts of interest/Competing interests (include appropriate disclosures)

Availability of data and material (data transparency)

Code availability (software application or custom code)

Authors' contributions (optional: please review the submission guidelines from the journal whether statements are mandatory)

To be used for life science journals + articles with biological applications

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Conflicts of interest/Competing interests (include appropriate disclosures)

Ethics approval (include appropriate approvals or waivers)

Consent to participate (include appropriate statements)

Consent for publication (include appropriate statements)

Availability of data and material (data transparency)

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Authors' contributions (optional: please review the submission guidelines from the journal whether statements are mandatory)

Please see the relevant sections in the submission guidelines for further information as well as various examples of wording. Please revise/customize the sample statements according to your own needs.

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Text

Text Formatting

Manuscripts should be submitted in Word.

- Use a normal, plain font (e.g., 10-point Times Roman) for text.
- Use italics for emphasis.
- Use the automatic page numbering function to number the pages.
- Do not use field functions.
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- Use the equation editor or MathType for equations.
- Save your file in docx format (Word 2007 or higher) or doc format (older Word versions).

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LaTeX macro package (Download zip, 188 kB)

Headings

Please use no more than three levels of displayed headings.

Abbreviations

Abbreviations should be defined at first mention and used consistently thereafter.

Footnotes

Footnotes can be used to give additional information, which may include the citation of a reference included in the reference list. They should not consist solely of a reference citation, and they should never include the bibliographic details of a reference. They should also not contain any figures or tables.

Footnotes to the text are numbered consecutively; those to tables should be indicated by superscript lower-case letters (or asterisks for significance values and other statistical data). Footnotes to the title or the authors of the article are not given reference symbols.

Always use footnotes instead of endnotes.

Acknowledgments

Acknowledgments of people, grants, funds, etc. should be placed in a separate section on the title page. The names of funding organizations should be written in full.

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Scientific style

Generic names of drugs and pesticides are preferred; if trade names are used, the generic name should be given at first mention.

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References

Citation

Reference citations in the text should be identified by numbers in square brackets. Some examples:

1. Negotiation research spans many disciplines [3].

2. This result was later contradicted by Becker and Seligman [5].

3. This effect has been widely studied [1-3, 7].

Reference list

The list of references should only include works that are cited in the text and that have been published or accepted for publication. Personal communications and unpublished works should only be mentioned in the text. Do not use footnotes or endnotes as a substitute for a reference list.

The entries in the list should be numbered consecutively.

- Journal article

  Gamelin FX, Baquet G, Berthoin S, Thevenet D, Nourry C, Nottin S, Bosquet L (2009) Effect of high intensity intermittent training on heart rate variability in prepubescent children. Eur J Appl Physiol 105:731-738. https://doi.org/10.1007/s00421-008-0955-8

  Ideally, the names of all authors should be provided, but the usage of "et al" in long author lists will also be accepted:

  Smith J, Jones M Jr, Houghton L et al (1999) Future of health insurance. N Engl J Med 965:325–329

- Article by DOI

  Slifka MK, Whitton JL (2000) Clinical implications of dysregulated cytokine production. J Mol Med. https://doi.org/10.1007/s001090000086

- Book

  South J, Blass B (2001) The future of modern genomics. Blackwell, London

- Book chapter

  Brown B, Aaron M (2001) The politics of nature. In: Smith J (ed) The rise of modern genomics, 3rd edn. Wiley, New York, pp 230-257

- Online document

  Cartwright J (2007) Big stars have weather too. IOP Publishing PhysicsWeb. http://physicsweb.org/articles/news/11/6/16/1. Accessed 26 June 2007

- Dissertation

  Trent JW (1975) Experimental acute renal failure. Dissertation, University of California

Always use the standard abbreviation of a journal's name according to the ISSN List of Title Word Abbreviations, see

ISSN.org LTWA

If you are unsure, please use the full journal title.

For authors using EndNote, Springer provides an output style that supports the formatting of in-text citations and reference list.

EndNote style (Download zip, 4 kB)

Authors preparing their manuscript in LaTeX can use the bibtex file spbasic.bst which is included in Springer's LaTeX macro package.

Tables

- All tables are to be numbered using Arabic numerals.
- Tables should always be cited in text in consecutive numerical order.
- For each table, please supply a table caption (title) explaining the components of the table.
- Identify any previously published material by giving the original source in the form of a reference at the end of the table caption.
- Footnotes to tables should be indicated by superscript lower-case letters (or asterisks for significance values and other statistical data) and included beneath the table body.

Appendix B – Ethical Approval Email for NDAU project.

From: psychethics <psychethics@cardiff.ac.uk>
Subject: Ethics Feedback - EC.16.10.11.4592GRA5
Date: 5 July 2018 at 10:34:22 BST
To: Stephanie Van Goozen <VangoozenS@cardiff.ac.uk>

Dear Steph,

The Ethics Committee has considered the amendment to your Staff project proposal: A Feasibility Study of a Neurodevelopmental Disorders Assessment Unit (EC.16.10.11.4592GRA5).

The amendment has been approved on the condition that a comment is added to the information, stating that if a child shows distress the monitor can be removed immediately.

Please note that if any changes are made to the above project then you must notify the Ethics Committee.

Best wishes,
Mark Jones

School of Psychology Research Ethics Committee

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http://psych.cf.ac.uk/aboutus/ethics.html

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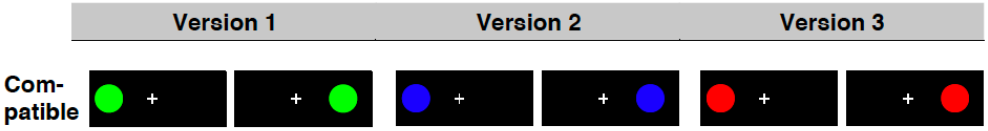
Appendix C – Amsterdam Neuropsychological Tasks – Response Organization Objects (ANT-ROO) instructions from ANT manual.

- **Response Organization Objects (ROO)**

The stimulus is a ball that is presented to the left or right of a fixation cross. The color of the ball determines the required type of stimulus-response mapping. Three parts, first part: compatible, second part: incompatible and third part: random (mixed) stimulus-response mapping.

- Part 1



In this task, you will always see a cross in the center of the screen [Instruct]. You will see a ball at the left or at the right of the screen, see? [Spacebar], [Spacebar]. In this part, the ball tells you exactly what you should do. So, you should press the left key when the ball is at the left side of the screen, and the right key when the ball is at the right side of the screen. [1] Any questions?

Practice
We will first practice. Put both index fingers on the response keys. *Try to respond as fast and as accurately as possible.* Attention… [Practice]

Test
Now the real test comes. Hold your fingers on the keys. *Try to respond as fast and as accurately as possible.* Attention … [Test]

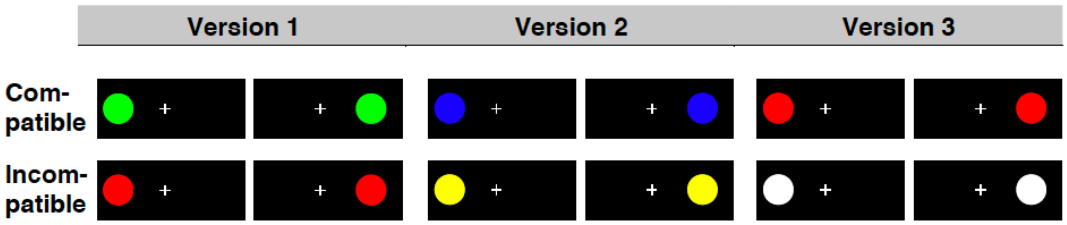
- Part 2

This time, we made it more difficult. [Instruct]. The balls have another color and therefore you should do exactly the opposite of what the ball tells you. So you should press the left key when the ball is at the right side of the screen, and the right key when the ball is at the left side of the screen, see? [Spacebar], [Spacebar]. [1] Any questions?

Practice
We will first practice. Put both index fingers on the response keys. *Try to respond as fast and as accurately as possible.* Attention… [Practice]

Test
Now the real test comes. Hold your fingers on the keys. *Try to respond as fast and as accurately as possible.* Attention … [Test]

- Part 3



This time, we made it even more difficult. [Instruct]. The balls will be shown at the left or at the right side of the screen and they will also change color. The color tells you what you should do: press the key at the same side, i.e. when the ball has this color [Spacebar], [Spacebar], or press the key at the opposite side, i.e. when the ball has this color [Spacebar], [Spacebar]. [1] Any questions?

Practice

We will first practice. Put both index fingers on the response keys. *Try to respond as fast and as accurately as possible*. Attention… [Practice]

Test

Now the real test comes. Hold your fingers on the keys. *Try to respond as fast and as accurately as possible*. Attention … [Test]

[1] In case of feedback: if you press the wrong key, you will hear a beep/see a red spot in the center of the screen.

Appendix D – Hungry Donkey task display, taken from Crone and van der Molen (2004)

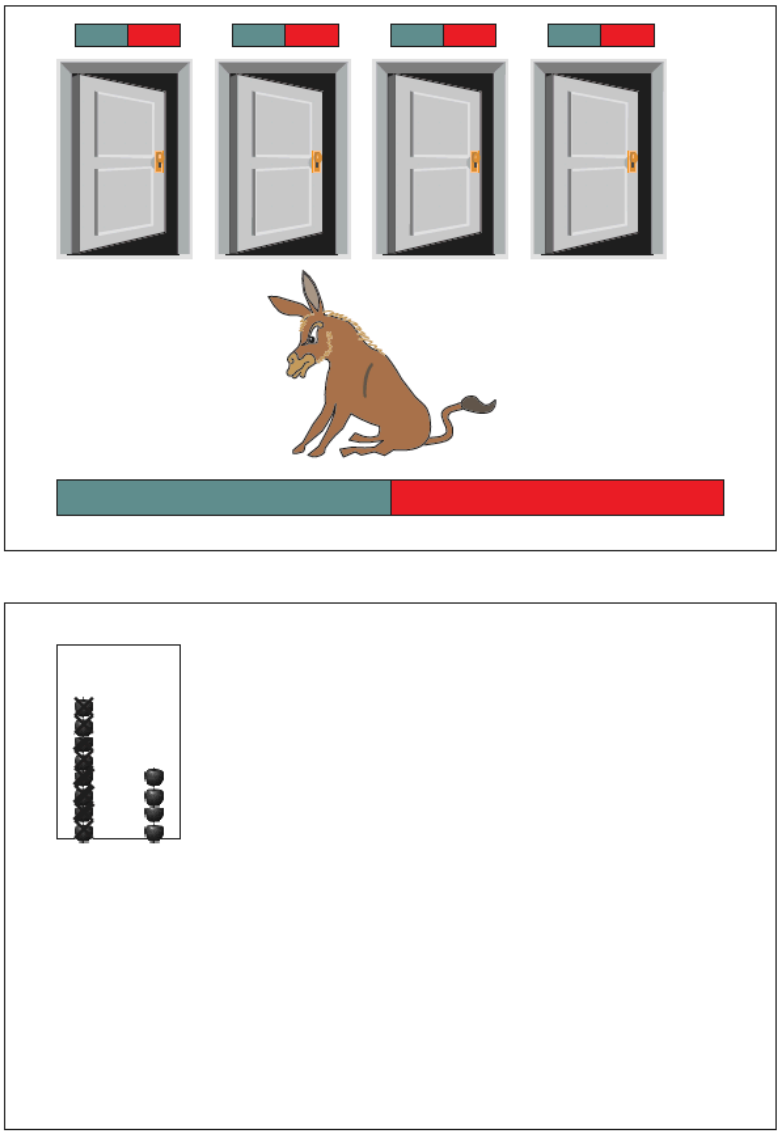
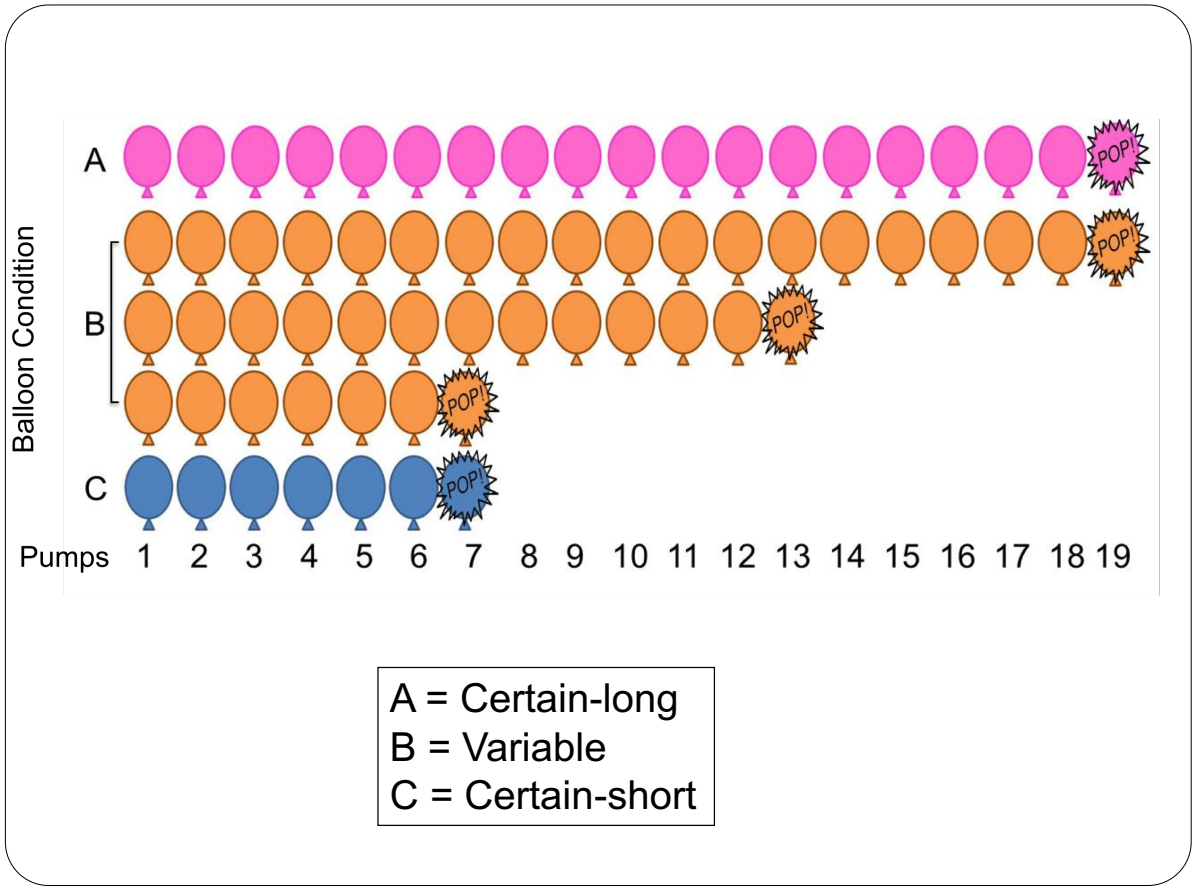


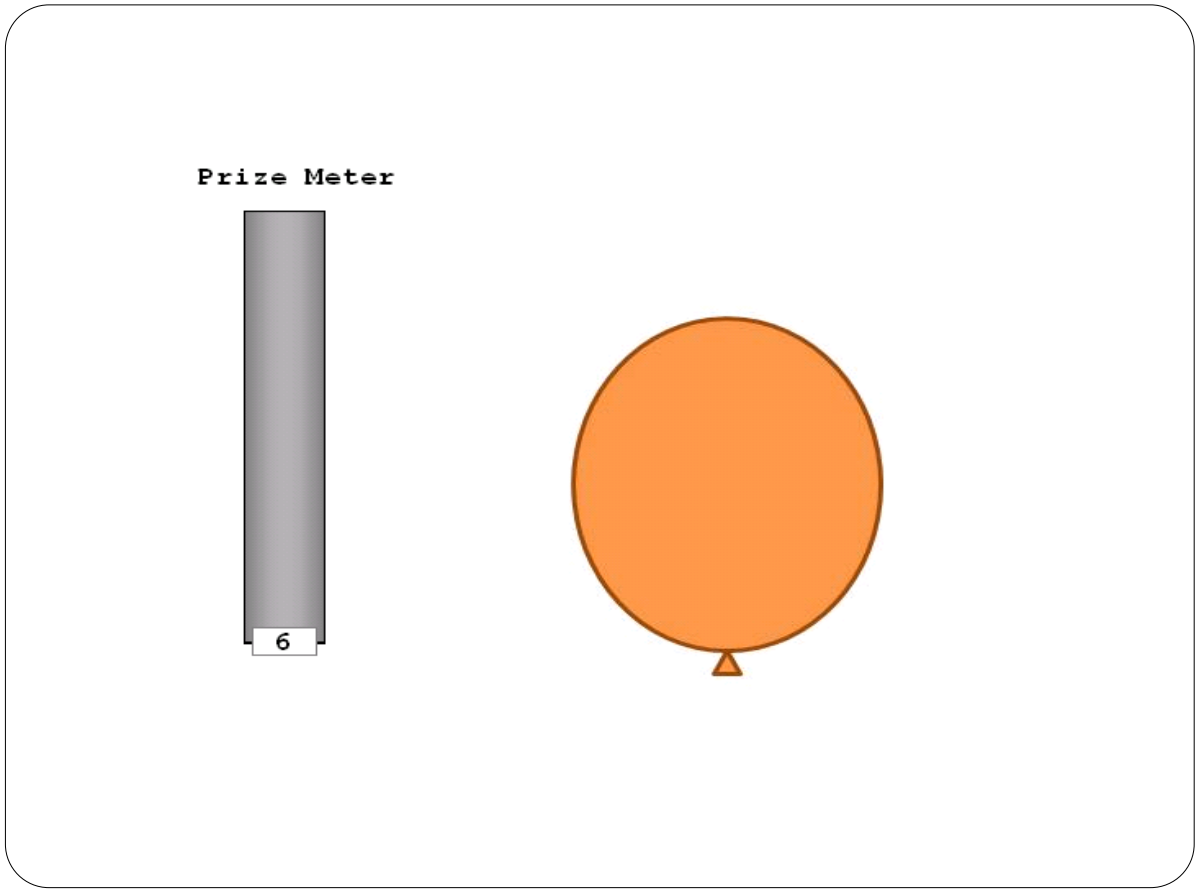
FIGURE 1 Example of stimulus display (top panel) and outcome display (bottom panel). The stimulus-screen consisted of four doors, a donkey, and bars that monitored performance (dependent on task condition). The doors were presented on the top half of the screen, approximately 2 cm from the top. They were equal in size, approximately 3 × 5 cm (length × height), and were located at a distance of approximately 2 cm from each other. The donkey (sized approximately 4 × 5 cm) was presented at the middle of the screen, approximately 1.5 cm below the doors and 2 cm above the bottom of the screen. The bars all were approximately 1 cm in height. The horizontal bar covered the whole length of the screen, until 1 cm from both sides. The bars above the doors covered the width of the doors (approximately 3 cm) and were presented approximately .5 cm above each door. Bar changes indexing the tally of wins and losses following each selection were computed using the formula: ([length/2] + [gain – loss]) / (12 × [length/2]).

Crone EA, van der Molen MW (2004) Developmental Changes in Real Life Decision Making: Performance on a Gambling Task Previously Shown to Depend on the Ventromedial Prefrontal Cortex. Developmental Neuropsychology 25 (3):251-279. doi:10.1207/s15326942dn2503_2

NB: The four-door version is displayed in the image above. The study utilized the two-door version

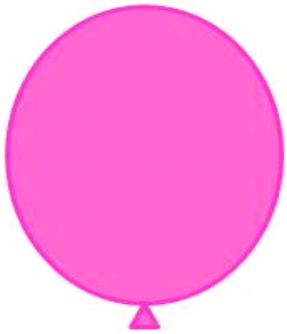
Appendix E – Balloon Emotional Learning Task (BELT) - Balloon conditions and example of task display.





Pop!

Prize Meter



You earned 12 points!

Appendix F – Strength and Difficulties Questionnaire (SDQ) – Parent version

Scoring the Strengths & Difficulties Questionnaire for age 4-17

The 25 items in the SDQ comprise 5 scales of 5 items each. It is usually easiest to score all 5 scales first before working out the total difficulties score. 'Somewhat True' is always scored as 1, but the scoring of 'Not True' and 'Certainly True' varies with the item, as shown below scale by scale. For each of the 5 scales the score can range from 0 to 10 if all items were completed. These scores can be scaled up pro-rata if at least 3 items were completed, e.g. a score of 4 based on 3 completed items can be scaled up to a score of 7 (6.67 rounded up) for 5 items.

Table 1: Scoring symptom scores on the SDQ for 4-17 year olds

Scale	Items	Not True	Somewhat True	Certainly True
Prosocial	**ITEM 1:** Considerate of other people's feelings (I try to be nice to other people)	0	1	2
Hyperactivity	**ITEM 2**: Restless, overactive… *(I am restless…)*	0	1	2
Emotional Problems	**ITEM 3:** Often complains of headaches… (I get a lot of headaches…)	0	1	2
Prosocial	**ITEM 4:** Shares readily with other children… *(I usually share with others)*	0	1	2
Conduct Problems	**ITEM 5:** Often has temper tantrums or hot tempers *(I get very angry)*	0	1	2
Peer Problems	**ITEM 6:** Rather solitary, tends to play alone *(I am usually on my own)*	0	1	2
Conduct Problems	**ITEM 7:** Generally obedient… *(I usually do as I am told)*	2	1	0
Emotional Problems	**ITEM 8:** Many worries… *(I worry a lot)*	0	1	2
Prosocial	**ITEM 9:** Helpful if someone is hurt… *(I am helpful is someone is hurt…)*	0	1	2
Hyperactivity	**ITEM 10:** Constantly fidgeting or squirming *(I am constantly fidgeting….)*	0	1	2

Peer Problems	**ITEM 11:** Has at least one good friend *(I have one goof friend or more)*	2	1	0
Conduct Problems	**ITEM 12:** Often fights with other children… *(I fight a lot)*	0	1	2
Emotional Problems	**ITEM 13:** Often unhappy, downhearted… *(I am often unhappy….)*	0	1	2
Peer Problems	**ITEM 14:** Generally liked by other children *(Other people my age generally like me)*	2	1	0
Hyperactivity	**ITEM 15:** Easily distracted, concentration wanders *(I am easily distracted)*	0	1	2
Emotional Problems	**ITEM 16:** Nervous or clingy in new situations… *(I am nervous in new situations…)*	0	1	2
Prosocial	**ITEM 17:** Kind to younger children *(I am kind to younger children)*	0	1	2
Conduct Problems	**ITEM 18:** Often lies or cheats *(I am often accused of lying or cheating)*	0	1	2
Peer Problems	**ITEM 19:** Picked on or bullied by other children… *(Other children or young people pick on me)*	0	1	2
Prosocial	**ITEM 20:** Often volunteers to help others… *(I often volunteer to help others)*	0	1	2
Hyperactivity	**ITEM 21:** Thinks things out before acting *(I think before I do things)*	2	1	0
Conduct Problems	**ITEM 22:** Steals from home, school or elsewhere *(I take things that are not mine)*	0	1	2

Peer Problems	**ITEM 23:** Gets on better with adults than with other children *(I get on better with adults than with people my age)*	0	1	2
Emotional Problems	**ITEM 24:** Many fears, easily scared *(I have many fears…)*	0	1	2
Hyperactivity	**ITEM 25:** Sees tasks through to the end… *(I finish the work I am doing)*	2	1	0

Total difficulties score: This is generated by summing scores from all the scales except the prosocial scale. The resultant score ranges from 0 to 40, and is counted as missing of one of the 4 component scores is missing.

'Externalising' and 'internalising' scores: The externalising score ranges from 0 to 20 and is the sum of the conduct and hyperactivity scales. The internalising score ranges from 0 to 20 and is the sum of the emotional and peer problems scales. Using these two amalgamated scales may be preferable to using the four separate scales in community samples, whereas using the four separate scales may add more value in high-risk samples *(see Goodman & Goodman. 2009 Strengths and difficulties questionnaire as a dimensional measure of child mental health. J Am Acad Child Adolesc Psychiatry 48(4), 400-403).*