**Hyper-persistence of attention-deficit/hyperactivity disorder (ADHD) in adult adoptees exposed to extreme early institutional deprivation: Clinical presentation, developmental continuities and functional impairment in the English and Romanian Adoptees study.**

Mark Kennedy1, Jana Kreppner1, Nicky Knights2, Robert Kumsta3, Barbara Maughan4, Michael Rutter4, Wolff Schlotz5 & Edmund Sonuga-Barke1

1. Developmental Brain-Behaviour Laboratory Psychology Academic Unit, University of Southampton.
2. The Amy Winehouse Foundation, London.
3. Department of Genetic Psychology, Faculty of Psychology, Ruhr-University Bochum, Germany.
4. MRC Social, Genetic & Developmental Psychiatry Centre, Institute of Psychiatry, Psychology and Neuroscience, King’s College London.
5. Max-Planck-Institute for Empirical Aesthetics, Frankfurt am Main.

**Correspondence: Edmund J S Sonuga-Barke, Psychology Academic Unit, University of Southampton, Southampton, SO17 1BJ. Email –** [**ejb3@soton.ac.uk**](mailto:ejb3@soton.ac.uk)**.**

**Submitted for JCPP Special Issue 2016**

**Word count**

Abstract- 288 words

Text - 4864

**Acknowledgements**

We would like to express our sincere gratitude to all the families and young people who have participated in this study over the many years it has been running. Without them none our work would be possible. We are also grateful to the comments from Dr Dennis Golm and Dr Graeme Fairchild during initial planning meetings and also to Helen Loader for her support. Thanks go to all the previous researchers on the project especially Dr Celia Beckett, Dr Jenny Castle, Dr Suzanne Stevens, Dr Emma Colvert and Amanda Hawkins who were involved in the collection of data during the mid-adolescent phase of the project. We thank our advisory board for all their useful suggestions especially Professor Megan Gunnar, Professor Jonathan Hill, Professor Trevor Robbins and Dr John Symonds. This work was funded by a project grant from the UK Economic Social Research Council (ESRC; RES-062-23-3300).

**Disclosures**

Over the last three years Professor Edmund Sonuga-Barke has received speaker fees, consultancy, research funding and conference support from Shire Pharma and speaker fees from Janssen Cilag, He has received consultancy fees from Neurotech solutions, Aarhus University, Copenhagen University and Berhanderling, Skolerne, Copenhagen, KU Leuven. Book royalties from OUP and Jessica Kingsley. He is the editor-in-chief of the Journal of Child Psychology and Psychiatry for which his University receives financial support. No other authors had conflicts of interest to declare.

**Abstract**

**Background: E**arly institutional deprivation is a putative social environmental cause of attention-deficit/hyperactivity disorder (ADHD). In childhood deprivation-related ADHD has a number of distinctive clinical features. In this paper, we examine, for the first time, the persistence of young adult ADHD in a sample of individuals adopted by UK families as young children after periods of up to 43 months in extremely depriving Romanian orphanages. **Methods:** DSM-IV symptom and impairment criteria were used to ascertain ADHD status for individuals when they were adolescents (age 15 years; 47 UK comparison and 146 Romanian adoptees) and young adults (age 22-25 years; 36 UK comparison and 101 Romanian adoptees). Data on co-occurring symptoms of disinhibited social engagement (DSE), autism spectrum disorder (ASD), cognitive impairment, conduct disorder (CD), anxiety and depression as well as young adult quality of life was also gathered. **Results:** In adolescence adoptees with more than 6 months institutional deprivation were nearly four times more likely to meet ADHD criteria than a combined group of UK controls and Romanian adoptees with less than 6 months deprivation (19% versus 5.6%). By young adulthood the difference had increased to more than seven fold (29.3% versus 3.8%). Young adult ADHD was equally common in males and females, was predominantly inattentive in presentation and co-occurred with high levels of DSE and ASD features. CD was strikingly absent from the group. ADHD was associated with reduced quality of life, high unemployment and low educational attainment. **Conclusion:** We provide the first evidence of the hyper-persistence into adulthood of a distinctively complex and impairing institutional deprivation related form of ADHD. Our results confirm the powerful impact of early experience on later development in a way that suggests a role for deep-seated alterations to brain structure and function.

Key words: attention deficit/hyperactivity disorder, institutional deprivation, Romanian adoptees, adult, longitudinal, adversity.

**Key points**

* Early severe institutional deprivation is associated with adverse long-term outcomes in groups of adopted children and adolescents in a substantial minority of cases.
* In the English and Romanian Adoptees study, ADHD-type problems are a characteristic feature of a distinctive pattern of deprivation-related childhood and adolescent problems also including quasi-autism, disinhibited social engagement and cognitive impairment.
* This study provides the first evidence of striking persistence of ADHD into adulthood following early institutional deprivation.
* Deprivation-related adult ADHD was distinctive in terms of its inattentive presentation, high proportion of females, co-occurrence with disinhibited social engagement and autism symptoms and low rates of conduct disorder.
* The hyper-persistence and complex nature of ADHD in adults exposed to severe early deprivation highlights the need to optimise continuity and cooperation between child to adult clinical services.

**Background**

Attention-deficit/hyperactivity disorder (ADHD), characterised by developmentally inappropriate and impairing symptoms of inattention, impulsivity and hyperactivity, is a childhood onset disorder with deleterious effects across the lifespan (Chorozoglou et al., 2015; Faraone & Biederman, 2005; Sonuga-Barke & Taylor, 2015). It is estimated to affect around five percent of children and adolescents worldwide (Polanczyk, Salum, Sugaya, Caye & Rohde, 2015). It is heterogeneous – with three clinical presentations defined in DSM-5 (American Psychiatric Association, 2013): predominantly inattentive (ADHD-PI), predominantly hyperactive/impulsive (ADHD-PHI) and combined types (ADHD-CT) affecting around 30, 10 and 60 percent of patients respectively (Willcutt, 2012). ADHD frequently co-occurs with conditions such as conduct, mood and anxiety disorders (Yoshimasu et al., 2012), as well as with learning (Plourde et al., 2015) and pervasive developmental difficulties (Jang et al. 2013). ADHD is associated with lower IQ scores (Frazier, Demaree & Youngstrom, 2004) and is common in children with intellectual disability (i.e., Ahuja, Martin, Langley & Thapar, 2013). Elevated rates of insecure attachment have been noted although the direction of causation remains to be established (Thorell, Rydell & Bohlin, 2012). ADHD is around 2.5 times more common in males than females in the general childhood population (Arnett et al., 2015; Willcutt, 2012) – although ADHD-PI is particularly common in girls (Biederman et al., 2014; Willcutt, 2012).

Although initially regarded as a child and adolescent condition, ADHD is now also established as an important source of mental ill health and impairment in adulthood (Garcia et al., 2012). While as few as 20% of patients with ADHD in childhood continue to meet the full child diagnostic criteria in adulthood, substantial continuity is observed in those who continue to experience ADHD-related impairment and sub-threshold symptoms (Faraone, Biederman & Mick, 2006). Comorbidities remain common but take on an adult form with antisocial personality disorder, anxiety, mood and substance use disorders especially common (Rasmussen & Levander, 2009). Males and females appear to be more equally represented in the adult ADHD population (Matte et al., 2015).

In keeping with its high heritability (Larsson, Chang, D’Onofrio & Lichtenstein, 2014) current aetiological models emphasize the role of genetic factors in ADHD (Thapar, Cooper, Eyre & Langley, 2013). Historically, the role of pre-natal environmental exposures such as those related to maternal smoking during pregnancy (Obel et al., 2015) and adverse intra-uterine environments marked by low birth weight have also been investigated (Pettersson et al., 2015). Recent longitudinal studies confirming the link between ADHD and socio-economic status suggest a role for post-natal social factors (Larsson, Sariaslan, Långström, D’Onofrio & Lichtenstein, 2014; Russell, Ford, Rosenberg & Kelly, 2014). However, such effects are non-specific, marking, as they likely do, a myriad adverse environmental exposures and familial genetic risks (Nigg & Craver, 2014). The same interpretational challenge is presented by studies linking ADHD to family conflict and maltreatment (McMillen et al., 2005; but see Harold et al., 2013). To date the most compelling evidence for a predominantly social/environmental pathway to ADHD comes from studies of children exposed to non-family related adversity (as found, for example, in institutional settings), and then placed with adoptive or foster families (van IJzendoorn et al., 2011). Elevated levels of inattention and hyperactivity/impulsivity have been reported in different institutionalised populations (Loman et al., 2013; McLaughlin et al., 2014; McLaughlin et al., 2010; Roy, Rutter & Pickles, 2004; Wiik et al. 2010), with effects growing stronger as a function of duration of institutional care and severity of deprivation experienced (Merz & McCall, 2010).

Evidence from the English and Romanian Adoptees study (ERA) is especially compelling in this regard (Rutter, Sonuga-Barke & Castle, 2010). ERA has followed a group of children who lived for up to the first three-and-a-half years of their lives in extreme deprivation in institutions in Romania during the final days of the Ceaușescu regime. Soon after the fall of the regime in 1989 they were adopted by families living in the UK. The conditions in the institutions varied from poor to appalling, with little or no personalised care or social or cognitive stimulation. Hygiene and food were also badly compromised. The initial effects of deprivation, seen immediately post-adoption, were profound and generalised, with extreme growth stunting and developmental delay (Rutter, 1998). By the age of six years there was evidence of substantial developmental and physical growth catch-up for many children indexed against a comparison group of non-deprived UK adoptees (O'Connor et al., 2000). However, a substantial minority of cases displayed persisting patterns of residual impairment in rather specific but overlapping domains (Kreppner et al., 2010; Kumsta et al., 2010), described previously as deprivation-specific problems (Rutter et al., 2010): quasi-autism (Rutter et al., 2007), disinhibited attachment (Rutter et al., 2007) and cognitive impairment (Beckett et al., 2006). Crucially, with regard to inferring a casual role for institutional exposures in the aetiology of such adverse outcomes, there was in each case a relationship between time spent in the institutions and severity of impairment. More specifically, there was a marked step-wise increase in problem severity for individuals who had spent more than 6-9 months in the institutions that emerged most clearly in assessments made at ages 11 and 15 years. In fact, those individuals in the institutions for less than 6 months were in many ways indistinguishable from typically developing peers.

A further distinctive element in the deprivation-specific profile is the elevated levels of ADHD symptoms consistently observed at the age 6, 11 and 15 year follow ups (Kreppner et al., 2001; Stevens et al., 2008; Stevens et al., 2009) – again showing the characteristic step wise increase with duration of deprivation. At age 15 years individuals who experienced more than 6 months of deprivation were four times more likely to meet DSM-IV diagnostic criteria for ADHD than those with less than 6 months deprivation (16% versus 4%). Although the symptom profile in these cases appeared typical of ADHD more generally, there were a number of features that appeared to distinguish deprivation-related from non-deprivation related ADHD: the sex difference was less marked; there was a striking absence of comorbid conduct problems, but high levels of social disinhibition and autistic features; and neuropsychological impairment was unusually severe (Sonuga-Barke & Rubia, 2008).

In this paper, we provide the first evidence relating to the persistence of deprivation-related ADHD into early adulthood using data from the recently completed ERA young adult follow up assessment carried out when the adoptees were aged 22-25 years of age. Our research questions were; (i) does a history of extended deprivation in institutions continue to place adoptees at risk for ADHD in early adulthood and, as previously found, does this manifest as a step-wise increase in those experiencing more than 6-9 months deprivation? (ii) Is there a drop in the proportion of adult cases meeting full DSM criteria similar to that seen in typical ADHD clinic samples? (iii) Is there a distinctive distribution of the three clinical presentations of ADHD, and does this change between adolescence and early adulthood? (iv) Is ADHD still equally likely in exposed males and females in adulthood? (v) Is there anything distinctive about the pattern of co-occurring disorders observed in deprivation-related adult ADHD? (vi) Is deprivation-related ADHD associated with adult life achievements and quality of life (QoL)?

**Methods:**

**Participants:** 165 Romanian adoptees and 52 comparison UK adoptees with no history of deprivation, and their families, initially entered the study in the mid-1990s. At age 15 some outcome data was available for 199 participants (48 UK comparison – 35.4% female; 151 Romanian adoptees - 54.3% female) while by young adult follow up (ages 22-25 years) this dropped to 164 (42 UK comparison – 38.1% female; 122 Romanian adoptees – 54.1% female). The average age at young adult follow-up for the UK comparison group was 23.2 (22-25, SD=.77) years and for the Romanian Adoptees 23.6 (22-26, SD=.81) years. Based on age 15 data there was no difference between those remaining in the study at the young adult follow up and those dropping out in terms of duration of deprivation, sex of child, age, IQ or the proportion of cases with deprivation-related problems (data available from authors).

**Measures**

The ERA study included a wide range of interview, questionnaire, cognitive and observational measures at the adolescent and young adult follow-ups. Only measures relevant to the current analysis are described here.

**ADHD**

**General strategy:** ADHD and associated impairment was assessed using different instruments in mid-adolescence and early adulthood. Working within these constraints, our aim was to assess levels of continuity and persistence across the adolescent-adult transition while anchoring our estimates to DSM definitions of clinical significance. To this end we adopted a categorical approach to defining ADHD using DSM-5 childhood diagnostic thresholds to identify the presence or absence of the disorder at both follow-ups.

**Adolescence:** Data on ADHD was collected from parents as part of a modified Child and Adolescent Psychiatric Assessment (CAPA) interview (Angold & Costello, 2000). This is a well-validated semi-structured interview covering a range of psychiatric disorders assessed over the last three months. Information was collected about the presence of nine of the 18 DSM-5 ADHD symptoms – 3 relating to inattention, 3 to hyperactivity and 3 to impulsivity. Each symptom was coded on a 0 to 3 severity scale by trained interviewers. Following the standard approach, a symptom was judged present when a score of 2 (definite) or more was recorded. ADHD was deemed to be present if either at least 2 of 3 symptoms of inattention (ADHD-PI), 4 of 6 symptoms of hyperactivity and/or impulsivity combined (ADHD-PH/I) or both (ADHD-CT) were reported along with “definite” levels of impairment in daily functioning. These thresholds represented pro-rated equivalents of the full DSM-5 18-item childhood criteria.

**Young adult:** Data on the 18 DSM ADHD symptoms were collected using both self- and parent-reported ratings from the Conner’s Comprehensive Behaviour Rating Scale (CBRS; Conners, Pitkanen & Rzepa, 2011). This is a well-validated scale covering a wide range of mental health and developmental disorders of childhood and adolescence. The version included items adapted for use with young adults following permission from the copyright holders. Items were rated in terms of “never, seldom” to “very often, very frequently” over the last month on a scale of 0 to 3. To optimise the equivalence between assessment waves, the early adult assessment of ADHD employed the same nine parent-rated symptoms that were available in the adolescent dataset. A symptom was deemed present using the standard approach (a score of 2 (often) or higher). The same diagnostic thresholds were employed as in adolescence, and a rating of “always” in at least two settings was required for impairment. The sub-scales for inattention and hyperactivity/ impulsivity both had acceptable reliability, with Cronbach’s alphas of .76 and .80 respectively.

**Co-occurring developmental and mental health problems**

**Autism Spectrum Disorder (ASD):** The Social Communication Questionnaire (SCQ; Rutter, Bailey & Lord, 2003) was completed by parents at both mid-adolescent and young adult follow ups. It is a widely used and clinically validated 35 item screen for ASD symptoms that maps onto DSM diagnostic criteria. In the current analysis the standard cut off of 15 was used as this has been shown to have good sensitivity and specificity as an ASD screen (Witwer & Lecavalier, 2007). This cut off identifies approximately 4-5 percent of the childhood population as ASD in childhood (Chandler et al., 2007). The scale has also been validated in adulthood (Chowdhury, 2007).

**Disinhibited Social Engagement (DSE, formerly known as disinhibited attachment):** This was assessed during both the mid-adolescent and young adult follow ups using three questions to parents based on those previously used during the ages 6 and 11 year follow ups, but adapted to be developmentally appropriate (Kreppner et al., 2010; Kumsta et al., 2010; Rutter et al., 2007). The interview questions were; “Seems too friendly with strangers or too eager to approach strangers?”; “Makes very personal comments or asked intrusive questions of others they’ve just met?”; and “Seems unaware of social boundaries, or the closeness of interaction with whom they are not familiar?”. In addition, as at the age 15 follow up, the researchers conducting the young adult assessment rated participants’ behaviour during the interview against observational codes: In this case the codes were “socially disinhibited”, “making socially intrusive spontaneous comments”, “unsolicited physical contact” and “committing physical violation of boundaries” as at age 15 DSE was deemed present when a positive response was made to at least one item (Kreppner et al., 2010).

**Cognitive Impairment (CI):** Shortened versions of the WISC-III, UK version (Wechsler, 1974) and WASI (two-subscale version, Wechsler, 1999) were administered in adolescence and early adulthood respectively. As in previous ERA follow-up waves CI was deemed present if an individual had a measured IQ of less than 80.

**Mood and conduct problems in young adulthood:** Generalised anxiety, major depression and conduct disorder (CD) were assessed using the self-rated CBRS in young adulthood using standardized T-scores. In line with established guidelines, the young person’s report, rather than the parent version was used (American Psychiatric Association, 2006).

**Quality of Life (QoL):** The Satisfaction with Life Scale (Diener, Emmons, Larsen & Griffin, 1985) is a widely used, reliable and well validated self-report measure of perceived QoL. On a scale from 1- Strongly Disagree, to 5 – Strongly Agree, participants were asked whether they; (i) see their current life as ideal; (ii) are satisfied with life; (iii) would live life again in the same way; (iv) find life excellent and; (v) think they have secured the important things in life. QoL is measured by a single sum score, with higher scores indicating better perceived QoL.

**Young adult life circumstance:** Key indicators of young adult life achievement and functioning were derived from young adult and parent reports. These were (i) currently being unemployed, (ii) having lower educational achievement (i.e., GCSE’s or less), and (iii) being married/ cohabiting. These were coded in a binary form (0 doesn’t apply, 1 applies).

**Procedure**

Ethical approval was received from the University of Southampton Research Ethics Committee. Informed consent was received from all participants. Assessment was carried out during face-to-face interviews in participants homes. Questionnaires were completed on line or returned via the post.

**Results**

Figure 1 compares the proportion of individuals meeting ADHD thresholds in adolescence and early adulthood in the UK adoptee group and three groups of Romanian adoptees using all available data: Those with less than 6 months institutional deprivation, those with between 6 and 24 months of institutional deprivation and those with over 24 months of deprivation. The under 6 months deprivation group contained a number of individuals who experienced no institutional care as they were adopted straight from family homes (17 in adolescence and 11 in young adulthood). These individuals did not differ in terms of levels of ADHD from the other members of this group (p=.71 for inattention; p=.39 for hyperactive/ impulsive). Data on ADHD symptoms were available for 193 individuals in adolescence and 137 individuals in young adulthood.

At both ages there was a similarly small proportion of ADHD cases in the UK comparison group and the group with less than 6 months’ deprivation (adolescence: 4.3% versus 6.6%- 2=0.27 (df=1), p=0.61; early adulthood: 2.8% versus 4.7% - 2=0.19 (df=1), p=0.66), with proportions in all cases being in line with population-based prevalence estimates (i.e., around 5%). There was no difference in the ADHD rates for the Romanian adoptees experiencing 6-24 month and over 24 months institutional deprivation (adolescence: 21.4% versus 16.7%- 2=0.31 (df=1), p=0.58; young adulthood 34.4% versus 23.1% –2=0.88 (df=1), p=0.35). There was however a highly significant difference between the combined group of UK and Romanian adoptees with less than 6 months institutional care (termed the *low-risk* group hereafter) and the combined group of those with more than six months in the institutions (termed the *high-risk* group henceforth) (adolescence 2=8.48 (df=1), p<.005; young adulthood 2=17.46 (df=1), p<.001). This confirmed the existence of the step-wise increase in risk associated with extended deprivation seen during previous follow-ups. In order to maximize statistical power all subsequent analyses compared these low- and high-risk groups.

Table 1 compares low- and high-risk groups in terms of the proportion of individuals with different ADHD presentations at the adolescent and young adult follow-ups. The effects of deprivation on ADHD appeared markedly stronger in early adulthood than adolescence, with a risk ratio of 1:3.9 for the former and 1:7.7 for the latter. In order to test whether this increase in risk over development was statistically significant we ran a generalized estimating equations model to test for effects of group (high versus low risk) as a between-subject variable and assessment age (adolescent versus early adult) as a repeated measure. Sidak-corrected pairwise comparisons of ADHD frequency between groups and assessment age were tested based on estimated marginal means. As shown above, ADHD was significantly more frequent in the high-risk group at both assessment waves (adolescent: difference = .14; SE = .048; p = .024; young adult: difference = .26; SE = .061; p = .001) but the interaction of group by age did not reach statistical significance (p = .12). However, when only those participants with data at both time points (low risk n = 78; high risk n = 53) were included in the analysis there was a significantly higher frequency of ADHD in the high-risk group in the young adult group only (adolescent difference = .11; SE = .062; p = .35; young adulthood difference = .24; SE = .066; p = .001). The group by age interaction term indicated a trend towards a significant increase in risk in young adulthood compared to adolescence (p = .053). There was also a high level of continuity at the individual case level with 70% of adolescent ADHD cases continuing to meet full diagnostic criteria in adulthood. ADHD-PI predominated at both follow up points; adolescence (2=8.62 (df=2), p<.05) and young adulthood (2=17.81 (df=2), p<.001). In adolescence, 75% of ADHD cases had a predominantly inattentive presentation while this declined to 59% in the young adult group.

Table 2 divides the young adult sample into three groups: (i) The low risk group (including the small number of ADHD), (ii) the high- risk group without ADHD and (iii) the high-risk group with ADHD. The groups are compared on a number of variables including sex, clinical engagement (e.g., diagnosed or treated specifically for ADHD), co-occurring problems and early adult circumstances. There was no difference between the three groups in ratio of males to females with roughly similar numbers of the sexes meeting diagnostic criteria for ADHD. The high-risk ADHD group were more likely than the low risk group and the high-risk group without ADHD to have received a clinical ADHD diagnosis and/ or treatment for ADHD (41%). The high-risk group with ADHD had the highest levels of both DSE and ASD. Nearly 60% of individuals with ADHD displayed the former and 15% the latter. In both cases this difference was statistically significant. Levels of DSE and ASD were also elevated in the high-risk non-ADHD group but the two high-risk groups did not differ statistically from each other. Compared to previous follow-ups the rates of CI were generally low and did not now vary as a function of ADHD group. On the basis of a comparison of dimensional measures the high-risk ADHD group also had significantly elevated levels of depression and anxiety compared to the low risk group but not compared to the high-risk non-ADHD group. Furthermore, the percentage of ADHD cases exceeding the standard CBRS cut-offs were approximately 45% and 55% for depression and anxiety respectively – around twice as many as found in the low risk group (26.4% and 23.3%). Strikingly, however, ADHD in the ERA sample was not associated with CD, with only 18.2% meeting standard CBRS cut offs compared to 9.7% in the high-risk non-ADHD group and 8.3% in the low risk group (p=.30 for high risk ADHD versus the low risk group). When considering current life circumstances, the very high rates of unemployment (almost 90%) and low educational achievement (over 70%) in the high risk ADHD group were significantly greater than in both other groups. Furthermore, QoL was lower in the high risk ADHD than the non-ADHD group.

**Discussion**

While adult ADHD following severe deprivation shares features with ADHD in typical clinical populations, it is also different in important ways. First, compared to ADHD found in typical clinical groups it appears to be strikingly persistent across the transition from adolescence to adulthood (Faraone et al., 2006) – a pattern of persistence previously seen in the ERA sample across childhood and early adolescence (Stevens et al., 2009). This is manifest both in the unusually high levels of continuity of ADHD at the level of the individual case and the very high rates of ADHD at the group level. Two aspects of this persistence are especially notable. First, it is observed at the level of symptoms and not just impairment, whereas in typical clinical populations symptoms often drop to sub-clinical levels although impairment remains common (Faraone et al., 2006). Second, the risk associated with deprivation in young adulthood was greater than in adolescence (although not statistically significant). We can draw a number of inferences from these observations. First, in general, they reinforce the notion that early exposure to severe adversity can have a powerful detrimental effect on long-term mental health and wellbeing (Rutter & O’Connor, 2004): In this case stretching across important periods of the lifespan - from the very earliest years of life to adulthood. Crucially these effects are found despite the fact that deprived individuals, post exposure, had spent the vast majority of their lives in loving, supportive and well-resourced families. Given this pattern of long term hyper-persistent risk it seems highly likely that deprivation-related ADHD results from early established deep-seated neurobiological alterations (see Nelson, Bos, Gunner & Sonuga-Barke, 2011 for a discussion). Whether the pathway from early deprivation to adult ADHD is mediated by structural and functional alterations in brain regions known to be involved in ADHD pathophysiology, or has its own neural signature, remains to be seen and is the subject of ongoing research. Second, and related to the above, the data provide the first suggestion in the literature that deprivation-related ADHD, perhaps as a particular example of an environmentally-driven variant of the condition, may represent a more persistent form of the condition than those seen in typical clinical practice. If true this would have both clinical and scientific implications. From a scientific perspective it may be that, somewhat paradoxically, ADHD established following early adversity may be less open to later operating genetic and environmental protective influences that can determine patterns of disorder offset in late childhood and early adolescence. Third, the data relating early institutional deprivation and adult ADHD may represent evidence of a distinctive developmental phenomenon linking environmental risk exposures and outcomes. Most longitudinal studies of social risk effects seem to conform to the rule that the further one moves in time away from the initial risk exposure, the smaller the impact on wellbeing. At least with regard to the adolescent-to-young adult transition, the opposite seems to be true here. Trying to tease apart the reasons for this is beyond the scope of the current paper, but there are a number of plausible possibilities. First, the persistence of risk could be due to the severity of the adverse experience implicated in early institutional deprivation and the extent of the associated “neural scarring”. Alternatively, it could be due to the early timing of the exposures. Finally, it could be the result of changing patterns of protective factors operating later in development. More specifically, it may that the buffering effect of high functioning family environments experienced by the adoptees supressed ADHD symptoms and/or associated impairments during childhood and adolescence so that the effects of early adversity only became apparent when individuals left home and moved away from these protective environments (an example of a sleeper effect).

In addition to elevated levels of persistence, deprivation-related ADHD seems to have other particular characteristics that mark it out from more common forms of ADHD. Four characteristics are particularly striking. First, and contrasting with the sex ratios found in population-based epidemiological studies, it seems equally common in males and females. Second, and perhaps relatedly, the predominantly inattentive presentation is unusually common in these ADHD cases. One hypothesis explaining the link between deprivation, sex and the inattentive presentation is that females compared to males are unusually susceptible to high doses of the sorts of environmental risk exposure (perhaps with a different risk threshold operating) that disrupt higher-level attentional control mechanisms. Third, deprivation-related ADHD has a characteristic pattern of comorbidities marked by persistently high DSE and ASD and low CD. Levels of depression and anxiety were also common in the ADHD sample but this is more typical of clinical ADHD samples in this age range. In the past, based on data from the age 11 and 15 years follow-ups, we have speculated about the existence of an institutional deprivation syndrome, the core of which is DSE and quasi-autism with deprivation-specific ADHD and CI being distinctive common associated features (Kreppner et al., 2010; Kumsta et al., 2010). Although this study was not designed to test this (and lacked sufficient statistical power to do so formally), the raised levels of ASD and DSE in the high risk ADHD group suggest a clustering of these problems in a way that is consistent with such a hypothesis. The pattern of results relating to CI, however, changed markedly between adolescence and young adulthood (Beckett et al., 2006) with no relationship observed between deprivation and CI in young adulthood. Since the time of the adoptees’ entry into the UK there has been a process of continual catch-up in IQ in the most deprived group – although a detailed examination of this is outside the scope of the current paper these findings suggest further catch-up between adolescence and young adulthood. The reason for the very low levels of conduct disorder (contrasted with more typical clinical populations) remains to be determined. It may be that CD is rare because of the specific presentation of ADHD found in the ERA sample (i.e., more inattentive and more female). Second, it may be rare because of the role of specific aetiological factors – ADHD forms resulting from environmental exposures, compared to genetic factors, might be less pleiotropic in their expression. Third, and related to the discussion above, growing up in a well- functioning family may buffer family-related risks that typically provoke the development of CD in ADHD individuals – such as high expressed emotion and harsh discipline (Scott et al., 2010) or peer-related deviance (Marshal, Molina & Pellham, 2003).

The findings have a number of clinical implications. First, it is clear that deprivation-related ADHD is associated with substantial clinical need, with poor long term outcomes (unemployment and poor educational attainment) and with reduced QoL. The clinical imperative is to ensure that deprived individuals with ADHD get the specialist services they need and that these continue into adult life. In this regard, at around 40% the life-time rate of clinical engagement in the ERA sample was relatively high. More generally, the current findings highlight the importance of measuring early life exposures to adverse environments in children with ADHD during clinical assessments in order to properly address the especially persistent and complex nature of the problems such individuals present.

The current study had significant strengths including its prospective nature and the stratification of deprivation-related risk to increase statistical power. There were also a number of limitations that need to be considered when interpreting its findings. One related to the way that ADHD symptoms were assessed. First, different approaches were used in adolescence and young adulthood to collect ADHD information – parental interview in adolescence and questionnaire in young adulthood. Second, information on only 9 ADHD items was collected during the adolescent interview. We addressed these constraints by (i) using a categorical approach to measuring ADHD – to reduce the impact of the different scaling properties of the two instruments on scores; (ii) restricting the young adult analysis to the same 9 symptoms assessed in adolescence and adjusting the diagnostic thresholds accordingly; (iii) employing the same DSM criteria at both ages (i.e., not employing the new DSM-5 adult criteria). This approach led to plausible estimates of ADHD rates in the low risk group. While we were initially concerned that use of questionnaires in the young adult sample would inflate rates, in fact the rates in the low risk group were lower in the young adult assessment than in the adolescent assessment. A second limitation concerned the level of attrition at the young adult assessment, which was much higher than the very low rates previously seen in the study. This although not surprising given the age range of the young adults limited the statistical power of the analysis especially in the comparison of ADHD and non-ADHD sub-groups. Crucially, however, attrition was not selective, with those retained and those lost to the study differing little on key measures.

In summary, the current study provides the first evidence of the hyper-persistence of a distinctively complex and impairing institutional deprivation-related form of ADHD into adulthood. This highlights the powerful impact of early experience on later development in a way that implicates deep-seated neurobiological alterations. Clinical services need to be especially mindful of the need to ensure an effective transition from adolescent to young adult services in ADHD individuals exposed to early adversity.

References

Ahuja, A., Martin, J., Langley, K., & Thapar, A. (2013). Intellectual disability in children with attention deficit hyperactivity disorder. *The Journal of pediatrics, 163*(3), 890-895. e891.

American Psychiatric Association. (2006). *American Psychiatric Association Practice Guidelines for the treatment of psychiatric disorders: compendium 2006*. American Psychiatric Pub.

American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed). Washington, DC: Author.

Angold, A., & Costello, E. J. (2000). The child and adolescent psychiatric assessment (CAPA). *Journal of the American Academy of Child & Adolescent Psychiatry, 39*(1), 39-48.

Arnett, A. B., Pennington, B. F., Willcutt, E. G., DeFries, J. C., & Olson, R. K. (2015). Sex differences in ADHD symptom severity. *Journal of Child Psychology and Psychiatry, 56*(6), 632-639.

Beckett, C., Maughan, B., Rutter, M., Castle, J., Colvert, E., Groothues, C., et al. (2006). Do the effects of early severe deprivation on cognition persist into early adolescence? Findings from the English and Romanian adoptees study. *Child development, 77*(3), 696-711.

Biederman, J., Kwon, A., Aleardi, M., Chouinard, V. A., Marino, T., Cole, H., et al. (2014). Absence of gender effects on attention deficit hyperactivity disorder: findings in nonreferred subjects. *American Journal of Psychiatry*.

Chandler, S., Charman, T., Baird, G., Simonoff, E., Loucas, T., Meldrum, D., et al. (2007). Validation of the social communication questionnaire in a population cohort of children with autism spectrum disorders. *Journal of the American Academy of Child & Adolescent Psychiatry, 46*(10), 1324-1332.

Chowdhury, M. (2007). *Course of Behavioral Change in High-functioning Young Adults on the Autism Spectrum* (Doctoral dissertation, The Ohio State University).

Conners, C. K., Pitkanen, J., & Rzepa, S. R. (2011). *Conners comprehensive behavior rating scale*: Springer.

Diener, E., Emmons, R. A., Larsen, R. J., & Griffin, S. (1985). The satisfaction with life scale. *Journal of personality assessment, 49*(1), 71-75.

Faraone, S. V., & Biederman, J. (2005). What is the prevalence of adult ADHD? Results of a population screen of 966 adults. *Journal of Attention Disorders, 9*(2), 384-391.

Faraone, S. V., Biederman, J., & Mick, E. (2006). The age-dependent decline of attention deficit hyperactivity disorder: a meta-analysis of follow-up studies. *Psychological medicine, 36*(02), 159-165.

Frazier, T. W., Demaree, H. A., & Youngstrom, E. A. (2004). Meta-analysis of intellectual and neuropsychological test performance in attention-deficit/hyperactivity disorder. *Neuropsychology, 18*(3), 543.

Garcia, C., Bau, C., Silva, K., Callegari-Jacques, S., Salgado, C., Fischer, A., et al. (2012). The burdened life of adults with ADHD: impairment beyond comorbidity. *European Psychiatry, 27*(5), 309-313.

Harold, G. T., Leve, L. D., Barrett, D., Elam, K., Neiderhiser, J. M., Natsuaki, M. N., et al. (2013). Biological and rearing mother influences on child ADHD symptoms: revisiting the developmental interface between nature and nurture. *Journal of Child Psychology and Psychiatry, 54*(10), 1038-1046.

Jang, J., Matson, J. L., Williams, L. W., Tureck, K., Goldin, R. L., & Cervantes, P. E. (2013). Rates of comorbid symptoms in children with ASD, ADHD, and comorbid ASD and ADHD. *Research in developmental disabilities, 34*(8), 2369-2378.

Kreppner, J., Kumsta, R., Rutter, M., Beckett, C., Castle, J., Stevens, S., et al. (2010). IV. Developmental course of deprivation-specific psychological patterns: early manifestations, persistence to age 15, and clinical features. *Monographs of the Society for Research in Child Development, 75*(1), 79-101.

Kreppner, J. M., O'Connor, T. G., Rutter, M., English, & Team, R. A. S. (2001). Can inattention/overactivity be an institutional deprivation syndrome? *Journal of abnormal child psychology, 29*(6), 513-528.

Kumsta, R., Kreppner, J., Rutter, M., Beckett, C., Castle, J., Stevens, S., et al. (2010). III. Deprivation‐specific psychological patterns. *Monographs of the Society for Research in Child Development, 75*(1), 48-78.

Larsson, H., Chang, Z., D'Onofrio, B. M., & Lichtenstein, P. (2014). The heritability of clinically diagnosed attention deficit hyperactivity disorder across the lifespan. *Psychological medicine, 44*(10), 2223-2229.

Larsson, H., Sariaslan, A., Långström, N., D'Onofrio, B., & Lichtenstein, P. (2014). Family income in early childhood and subsequent attention deficit/hyperactivity disorder: a quasi‐experimental study. *Journal of Child Psychology and Psychiatry, 55*(5), 428-435.

Loman, M. M., Johnson, A. E., Westerlund, A., Pollak, S. D., Nelson, C. A., & Gunnar, M. R. (2013). The effect of early deprivation on executive attention in middle childhood. *Journal of Child Psychology and Psychiatry, 54*(1), 37-45.

Marshal, M. P., Molina, B. S., & Pelham Jr, W. E. (2003). Childhood ADHD and adolescent substance use: an examination of deviant peer group affiliation as a risk factor. *Psychology of Addictive Behaviors, 17*(4), 293.

Matte, B., Anselmi, L., Salum, G., Kieling, C., Gonçalves, H., Menezes, A., et al. (2015). ADHD in DSM-5: a field trial in a large, representative sample of 18-to 19-year-old adults. *Psychological medicine, 45*(02), 361-373.

McLaughlin, K. A., Fox, N. A., Zeanah, C. H., Sheridan, M. A., Marshall, P., & Nelson, C. A. (2010). Delayed maturation in brain electrical activity partially explains the association between early environmental deprivation and symptoms of attention-deficit/hyperactivity disorder. *Biological psychiatry, 68*(4), 329-336.

McLaughlin, K. A., Sheridan, M. A., Winter, W., Fox, N. A., Zeanah, C. H., & Nelson, C. A. (2014). Widespread reductions in cortical thickness following severe early-life deprivation: a neurodevelopmental pathway to attention-deficit/hyperactivity disorder. *Biological psychiatry, 76*(8), 629-638.

McMillen, J. C., Zima, B. T., Scott, L. D., Auslander, W. F., Munson, M. R., Ollie, M.T., et al. (2005). Prevalence of psychiatric disorders among older youths in the foster care system. *Journal of the American Academy of Child & Adolescent Psychiatry, 44*(1), 88-95.

Merz, E. C., & McCall, R. B. (2010). Behavior problems in children adopted from psychosocially depriving institutions. *Journal of abnormal child psychology, 38*(4), 459-470.

Nelson, C. A., Bos, K., Gunnar, M. R., & Sonuga‐Barke, E. J. (2011). V. The neurobiological toll of early human deprivation. *Monographs of the Society for Research in Child Development, 76*(4), 127-146.

Nigg, J. T., & Craver, L. (2014). Commentary: ADHD and social disadvantage: an inconvenient truth?–a reflection on Russell et al.(2014) and Larsson et al.(2014). *Journal of Child Psychology and Psychiatry, 55*(5), 446-447.

Obel, C., Zhu, J. L., Olsen, J., Breining, S., Li, J., Grønborg, T. K., et al. (2015). The risk of attention deficit hyperactivity disorder in children exposed to maternal smoking during pregnancy–a reexamination using a sibling design. *Journal of Child Psychology and Psychiatry*.

O'Connor, T. G., Rutter, M., Beckett, C., Keaveney, L., Kreppner, J. M., English, et al. (2000). The effects of global severe privation on cognitive competence: Extension and longitudinal follow-up. *Child development*, 376-390.

Pettersson, E., Sjölander, A., Almqvist, C., Anckarsäter, H., D'Onofrio, B. M., Lichtenstein, P., et al. (2015). Birth weight as an independent predictor of ADHD symptoms: a within‐twin pair analysis. *Journal of Child Psychology and Psychiatry, 56*(4), 453-459.

Plourde, V., Boivin, M., Forget‐Dubois, N., Brendgen, M., Vitaro, F., Marino, C., et al. (2015). Phenotypic and genetic associations between reading comprehension, decoding skills, and ADHD dimensions: evidence from two population‐based studies. *Journal of Child Psychology and Psychiatry*.

Polanczyk, G. V., Salum, G. A., Sugaya, L. S., Caye, A., & Rohde, L. A. (2015). Annual Research Review: A meta‐analysis of the worldwide prevalence of mental disorders in children and adolescents. *Journal of Child Psychology and Psychiatry, 56*(3), 345-365.

Rasmussen, K., & Levander, S. (2009). Untreated ADHD in Adults Are There Sex Differences in Symptoms, Comorbidity, and Impairment? *Journal of Attention Disorders, 12*(4), 353-360.

Roy, P., Rutter, M., & Pickles, A. (2004). Institutional care: Associations between overactivity and lack of selectivity in social relationships. *Journal of Child Psychology and Psychiatry, 45*(4), 866-873.

Rucker, J. J., McGuffin, P., Bale, T. L., Baram, T. Z., Brown, A. S., Goldstein, J. M., et al. Rare gene variants in neurodevelopmental disorders.

Russell, G., Ford, T., Rosenberg, R., & Kelly, S. (2014). The association of attention deficit hyperactivity disorder with socioeconomic disadvantage: alternative explanations and evidence. *Journal of Child Psychology and Psychiatry, 55*(5), 436-445.

Rutter, M. & O'Connor, T. G. (2004). Are There Biological Programming Effects for Psychological Development? Findings From a Study of Romanian Adoptees. Developmental Psychology, Vol 40(1), Jan 2004, 81-94

Rutter, M. (1998). Developmental catch-up, and deficit, following adoption after severe global early privation. *Journal of Child Psychology and Psychiatry, 39*(04), 465-476.

Rutter, M., Bailey, A., & Lord, C. (2003). *The social communication questionnaire: Manual*: Western Psychological Services.

Rutter, M., Colvert, E., Kreppner, J., Beckett, C., Castle, J., Groothues, C., et al. (2007). Early adolescent outcomes for institutionally‐deprived and non‐deprived adoptees. I: Disinhibited attachment. *Journal of Child Psychology and Psychiatry, 48*(1), 17-30.

Rutter, M., Kreppner, J., Croft, C., Murin, M., Colvert, E., Beckett, C., et al. (2007). Early adolescent outcomes of institutionally deprived and non‐deprived adoptees. III. Quasi‐autism. *Journal of Child Psychology and Psychiatry, 48*(12), 1200-1207.

Rutter, M., Sonuga‐Barke, E. J., & Castle, J. (2010). I. Investigating the impact of early institutional deprivation on development: Background and research strategy of the English and Romanian Adoptees (ERA) study. *Monographs of the Society for Research in Child Development, 75*(1), 1-20.

Rutter, M., Sonuga-Barke, E. J., Beckett, C., Castle, J., Kreppner, J., Kumsta, R., et al. (2010). Deprivation-Specific Psychological Patterns: Effects of Institutional Deprivation. *Monographs of the Society for Research in Child Development, 75*(1), 1-252.

Scott, S., Sylva, K., Doolan, M., Price, J., Jacobs, B., Crook, C., et al. (2010). Randomised controlled trial of parent groups for child antisocial behaviour targeting multiple risk factors: the SPOKES project. *Journal of Child Psychology and Psychiatry, 51*(1), 48-57.

Sonuga‐Barke, E., & Rubia, K. (2008). Inattentive/overactive children with histories of profound institutional deprivation compared with standard ADHD cases: a brief report. *Child: care, health and development, 34*(5), 596-602.

Sonuga-Barke, E., & Taylor, E. (2015). Disorders of attention & activity *Rutter's Child and Adolescent Psychiatry*: Blackwells Oxford.

Stevens, S. E., Kumsta, R., Kreppner, J. M., Brookes, K. J., Rutter, M., & Sonuga‐Barke, E. J. (2009). Dopamine transporter gene polymorphism moderates the effects of severe deprivation on ADHD symptoms: developmental continuities in gene–environment interplay. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 150*(6), 753-761.

Stevens, S. E., Sonuga-Barke, E. J., Kreppner, J. M., Beckett, C., Castle, J., Colvert, E., et al. (2008). Inattention/overactivity following early severe institutional deprivation: presentation and associations in early adolescence. *Journal of abnormal child psychology, 36*(3), 385-398.

Thapar, A., Cooper, M., Eyre, O., & Langley, K. (2013). Practitioner review: what have we learnt about the causes of ADHD? *Journal of Child Psychology and Psychiatry, 54*(1), 3-16.

Thorell, L. B., Rydell, A.M., & Bohlin, G. (2012). Parent–child attachment and executive functioning in relation to ADHD symptoms in middle childhood. *Attachment & human development, 14*(5), 517-532.

van IJzendoorn, M. H., Palacios, J., Sonuga‐Barke, E. J., Gunnar, M. R., Vorria, P., McCall, R. B., et al. (2011). I. Children in institutional care: Delayed development and resilience. *Monographs of the Society for Research in Child Development, 76*(4), 8-30.

Wechsler, D. (1974). *WISC-R, Wechsler intelligence scale for children, revised*: Psychological Corporation.

Wechsler, D. (1999). *Wechsler abbreviated scale of intelligence*: Psychological Corporation.

Wiik, K. L., Loman, M. M., Van Ryzin, M. J., Armstrong, J. M., Essex, M. J., Pollak, S. D., et al. (2011). Behavioral and emotional symptoms of post‐institutionalized children in middle childhood. *Journal of Child Psychology and Psychiatry, 52*(1), 56-63.

Willcutt, E. G. (2012). The prevalence of DSM-IV attention-deficit/hyperactivity disorder: a meta-analytic review. *Neurotherapeutics, 9*(3), 490-499.

Witwer, A. N., & Lecavalier, L. (2007). Autism screening tools: an evaluation of the social communication questionnaire and the developmental behaviour checklist–autism screening algorithm. *Journal of Intellectual and Developmental Disability, 32*(3), 179-187.

Yoshimasu, K., Barbaresi, W. J., Colligan, R. C., Voigt, R. G., Killian, J. M., Weaver, A. L., et al. (2012). Childhood ADHD is strongly associated with a broad range of psychiatric disorders during adolescence: a population‐based birth cohort study. *Journal of Child Psychology and Psychiatry, 53*(10), 1036-1043.

Table 1: Proportion of individuals in low and high-risk groups meeting ADHD criteria

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | Adolescent | | Early adult | |
|  | Low Risk | High Risk | Low Risk | High Risk |
| Any ADHD | 6/108 (5.6%) | 16/84 (19.0%) | 3/79 (3.8%) | 17/58 (29.3%) |
| ADHD-CT | 1/108 (0.9%) | 4/84 (4.8%) | 2/79 (2.5%) | 7/58 (12.1%) |
| ADHD-PI | 5/108 (4.6%) | 12/84 (14.3%) | 1/79 (1.1%) | 10/58 (17.2%) |
| ADHD-PH/I | 0 | 0 | 0 | 0 |

Note: Low Risk was a group combining UK adoptees with Romanian Adoptees with less than 6 months of deprivation: High risk was a group containing all Romanian Adoptees with over 6 months deprivation. CT = combined type presentation; PI = predominantly inattentive presentation; PH/I = predominantly inattentive/hyperactive presentation.

Table 2: Demographic characteristics and clinical outcomes for low- risk and high-risk young adults with and without ADHD.

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
|  | **Low risk**  **(LR)**  **(n=79)** | **High risk** | | **Group contrasts** | | |
|  |  | **ADHD-**  **(n=41)** | **ADHD+**  **(n=17)** | **LR *vs* ADHD-** | **LR *vs* ADHD+** | **ADHD- *vs* ADHD+** |
| **Sex**  **(% female)** | 42.9 | 53.7 | 58.8 | 2=1.43, *p*=.23 | 2=1.53, *p*=.22 | 2=0.13, *p*=.72 |
| **Clinical diagnosis**  **(%)** | 6.7 | 12.2 | 41.2 | 2=1.22, *p*=.27 | **2=17.99, *p*<.001** | **2=6.15, *p*<.05** |
| **Co-occurring**  **disorder** |  |  |  |  |  |  |
| **DSE (%)** | 12.6 | 43.6 | 58.8 | **2=14.92, *p*<01** | **2=18.82, *p*<.001** | 2=1.10, *p*=.29 |
| **Autism (%)** | 2.8 | 6.7 | 15.4 | 2=0.85, *p*=.36 | **2=3.90, *p*<.05** | 2=0.82, *p*=.37 |
| **CI (%)** | 10.4 | 10.0 | 9.1 | 2=0.01, *p*=.95 | 2=0.02, *p*=.89 | 2=0.01, *p*=.93 |
| **CD** | 46.38 (10.78) | 48.45 (13.45) | 51.36 (11.16) | t(101)=-0.83, *p*=.41 | t(81)=-1.42, *p*=.16 | t(40)=-0.64, *p*=.52 |
| **Depression** | 54.29 (13.96) | 58.19 (14.97) | 65.00 (12.60) | t(101)=-1.27, *p*=.21 | **t(81)=-2.40, *p*=.02** | t(40)=-1.34, *p*=.19 |
| **Anxiety** | 54.15 (13.63) | 58.03 (14.03) | 62.73 (11.86) | t(102)=1.32, *p*=.19 | **t(82)=-2.00, *p*=.05** | t(40)=-0.99, *p*=.33 |
| **QoL** | 16.92 (5.38) | 18.19 (5.05) | 14.00 (5.33) | t(97)=-1.06, p=.29 | t(81)=1.68, p=.10 | **t(36)=-2.28, p<.05** |
| **Life circumstances** |  |  |  |  |  |  |
| **Unemployed (%)** | 12.0 | 24.4 | 88.2 | 2=3.30, *p*=.07 | **2=45.96, *p*<.001** | **2=19.97, *p*<.01** |
| **Low education (%)** | 26.4 | 31.7 | 76.5 | 2=0.39, *p*=.53 | **2=15.96, *p*<.001** | **2=9.74, *p*<.01** |
| **Married/cohabit (%)** | 22.8 | 39.0 | 17.6 | 2=3.71 ,*p*=.16 | 2=0.22, *p*=.64 | 2=2.49, *p*=.11 |

Note: Low Risk was a group combining UK adoptees with Romanian Adoptees with less than 6 months of deprivation: High risk was a group containing all Romanian Adoptees with over 6 months deprivation. ADHD- = High risk individuals not meeting ADHD criteria; ADHD+ = High risk individuals meeting ADHD criteria: DSE = disinhibited social engagement (formerly disinhibited attachment); CI = Cognitive Impairment; CD= conduct disorder (Self rated CBRS T score mean and (sd)); Depression = Self rated CBRS T score mean and (sd); Anxiety = Self-rated CBRS T scores mean and sd.; QoL = Self rated Satisfaction with Life score mean and (sd).

Figure 1: The proportion of ADHD cases in the UK comparison and the Romanian adoptees as a function of length of deprivation.

