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Full article title: Higher cognitive ability buffers stress-related depressive symptoms in adolescent girls

Abstract: Stress has been shown to have a causal effect on risk for depression. We investigated the role of cognitive ability as a moderator of the effect of stressful life events on depressive symptoms and whether this varied by gender. Data were analysed in two adolescent datasets: one representative community sample aged 11-12 years (n=460) and one at increased familial risk of depression aged 9-17 years (n=335). In both datasets, a three-way interaction was found whereby for girls, but not boys, higher cognitive ability buffered the association between stress and greater depressive symptoms. The interaction was replicated when the outcome was a diagnosis of major depressive disorder. This buffering effect in girls was not attributable to coping efficacy. However, a small proportion of the variance was accounted for by sensitivity to environmental stressors. Results suggest that this moderating effect of cognitive ability in girls is largely attributable to greater available resources for cognitive operations which offer protection against stress-induced reductions in cognitive processing and cognitive control which in turn reduces the likelihood of depressive symptomatology.

Key words: depression; cognitive ability; stress; resilience; cognitive reserve

Short title: Cognitive ability, stress and depression

Introduction

There is strong evidence for an association between stress and depression (Hammen, 2005). Indeed stressful life events exert a modest causal effect on the risk for major depressive disorder even when accounting for person effects on the environment (Kendler & Gardner, 2010). Stress appears to have a more important role in precipitating first episodes rather than recurrent episodes of depression (Kendler, Thornton, & Gardner, 2000; Monroe & Harkness, 2005; NICE, 2005). Adolescence is a period of vulnerability for depression with first episodes often emerging at this time and the prevalence of depressive symptoms increasing markedly, particularly among girls. The role of stress as a predictor of depression may therefore be of particular importance during this period. Indeed, it has been suggested that stress experienced during adolescence can have long-lasting effects on the development of brain areas involved in the regulation of stress via glucocorticoid exposure (Lupien, McEwen, Gunnar, & Heim, 2009). Adolescent depressive disorder and symptoms are associated with a range of contemporaneous and long-term difficulties including suicide attempts, hospitalisation, and impaired social, occupational and inter-personal functioning (e.g. Angold, Costello, Farmer, Burns, & Erkanli, 1999; Weissman et al., 1999). Taken together, these observations suggest that adolescence is an important period during which to assess the relationship between stress and depression.

Despite the strong associations between stress and depression, resilience work highlights that not all adolescents show negative outcomes following adversity. In particular, various authors suggest that higher cognitive ability (i.e. intelligence or executive processes) is associated with more positive social, academic and mental health outcomes following a range of stressors including adverse life events (Masten et al., 1999) and recurrent depression in a parent (Pargas, Brennan, Hammen, & Le Brocque, 2010; Tiet et al., 1998). Cognitive ability tests include tasks that assess verbal comprehension, abstract reasoning, working memory and processing speed (e.g. WISC; Wechsler, 2003) and thus index a wide-range of cognitive resources and processes including semantic memory, working memory and executive functioning. Individuals with higher cognitive ability may have resources such as greater cognitive reserve which serve to protect them against some of the adverse effects of stress whilst individuals with lower cognitive ability may be more vulnerable to depression following stressful life events because they are less equipped to cope with them (Barnett, Salmond,

Jones, & Sahakian, 2006; Koenen et al., 2009). Thus, cognitive ability may moderate the association between stress and depression. Specifically, higher cognitive ability may buffer (reduce) the association between stressful life events and depression (Figure 1, panel a). Stress exerts a modest causal effect on depression and has also been shown to impair memory for neutral material, executive processing and neuronal processes that subserve cognition such as dendritic arborization in the hippocampus (Lupien et al., 2009; Goodyer, 2008; Liston, McEwen, & Casey, 2009; Klein & Boals, 2001). Potential explanations of a possible interaction between cognitive ability and stress in predicting depression include that there is greater capacity and efficiency of the cognitive system in more able individuals which serves to protect them from some of the adverse effects of stress. Another is that more cognitively able individuals have greater capacity to profit from information acquired as a result of stressful situations, find meaning in them and positively reframing them - tendencies which have been associated with stress resilient outcomes (Southwick, Vythilingam, & Charney, 2005).

Studies generally report an inverse association between cognitive ability and depression both in childhood and adulthood (e.g. Collishaw, Maughan, & Pickles, 2004; Franz et al., 2011; Hartlage, Alloy, Vazquez, & Dykman, 1993), which is consistent with higher cognitive ability being a resilience factor against depression. Some research suggests that this association may be stronger in girls compared to boys (Glaser et al., 2011; Hatch et al., 2007). Regarding gender differences in depression, higher prevalence rates in girls compared to boys are well documented from mid-puberty onwards (Angold, Costello, & Worthman, 1998; Costello, Erkanli, & Angold, 2006; Green, 2005). Two explanations for the development of these gender differences are, firstly, that girls experience more negative life events than boys (Thapar, Collishaw, Pine, & Thapar, 2012), and secondly, that girls are more likely than boys to experience depression following these stressors (Hankin & Abramson, 2001). The buffering effect of higher cognitive ability proposed here may therefore be greater in girls than in boys (Figure 1, panel a). There is also some animal evidence that acute stress may impair learning in females while enhancing learning in males (Oldehinkel & Bouma, 2011). Cognitive ability has been suggested to play an important role in protecting against the damaging effects of stress (Boyce & Ellis, 2005) and girls appear to be more vulnerable to stress-related

depressive symptoms (Hankin & Abramson, 2001), Thus, girls with higher cognitive ability may benefit from a greater ability to process information under stress without impairing functioning, due to greater capacity and efficiency of the cognitive system.

Potential mechanisms through which higher cognitive ability in girls may buffer stress-related depressive symptoms include greater coping efficacy and lower sensitivity to environmental stressors (i.e. these mechanisms may mediate the moderating effect of cognitive ability on stress-related depressive symptoms in girls: mediated moderation; Figure 1, panel b). Cognitive processes such as active coping have been suggested to decrease the likelihood of developing disorders following stress, for example by preventing fear conditioning (Southwick et al., 2005). This is consistent with the elaborated cognitive vulnerability-transactional stress theory (Hankin & Abramson, 2001), which proposes that the likelihood of stress leading to depression is moderated by generic cognitive vulnerabilities. This theory also suggests that these cognitive vulnerabilities are greater in girls, making them more vulnerable to depression following stress. Therefore, coping efficacy may mediate the moderating effect of cognitive ability on stress-related depressive symptoms in girls. Another potential mechanism is environmental sensitivity. The biological-sensitivity-to-context theory (Boyce & Ellis, 2005) suggests that individuals differ in their biological sensitivity to the environment, with those of higher sensitivity (for example, those showing elevated cortisol response to stress) more likely to suffer negative outcomes following stress (Feder, Nestler, & Charney, 2009). Evidence from both animal and human studies suggests that females may perceive social stress as more stressful (Juster et al., 2011) and be more vulnerable to depression following social stress than males (McCormick & Mathews, 2007; Stroud, Salovey, & Epel, 2002). Thus, sensitivity to environmental stressors may be another mechanism through which cognitive ability buffers stress-related depressive symptoms in girls.

The aim of this study was to test the potential moderating effects of cognitive ability on the association between stress and depressive symptoms. Following some evidence that associations between cognitive ability and depressive symptoms may differ between high- and low-risk samples (Pargas et al., 2010), we included both types of sample in our study. Two datasets were analysed to

assess this question: a representative community based sample of 11 to 12 year olds (The School Transition & Adjustment Research Study: STARS) and a sample of the adolescent offspring of depressed parents aged 9 to 17 (The Early Prediction of Adolescent Depression Study: EPAD). Our main research hypotheses were: (1) cognitive ability would moderate the effect of stress on depressive symptoms; and, (2) the moderating effect of cognitive ability would be stronger in girls than boys. We also explored whether coping efficacy and sensitivity to environmental stressors were mediators of the predicted moderating effect of cognitive ability on the association between stress and depressive symptoms (i.e. mediated moderation).

Method

Samples

The STARS sample consists of data collected from pupils in year 7 (age 11-12 years old) attending ten mixed, non-selective secondary schools, in South-East England, UK. The schools were selected as their intake was representative of the English and Welsh population in terms of the proportion of pupils who were from economically disadvantaged households (indexed by eligibility for free school meals) and who had Special Educational Needs. The proportion of pupils from black and minority ethnic backgrounds and who did not have English as a first language was slightly higher than the general population, reflecting the inclusion of London schools. The study protocol was reviewed and approved by the university ethics committee. At each assessment, parents were given the opportunity to opt their children out of the study and informed pupil consent was obtained. Questionnaire data were collected at two stages: a postal survey during May 2012 (N=750; overall response rate=35%) and an in-school assessment during November 2012 (1712; overall response rate=87%). Cognitive ability data were collected in September of that year for six of the participating schools (N=1159). Of the 750 pupils for whom postal survey data was available, 663 (88%) completed the in-school assessments (reasons for non-completion were: pupil left the school; parent, pupil or school withdrawing from the study). Cognitive ability data were available for 460 (61% of those with baseline data), which formed our analysed sample (228 boys). The analysed sub-sample with complete data did not differ from the rest of the sample in terms of depressive symptoms

($t(1646)= 1.73$, $p=.08$) or negative life events ($t(743)= -0.36$, $p=.72$), but had higher cognitive ability scores ($t(1157)= -4.37$, $p<.001$).

The EPAD data consists of 337 children aged 9-17 years old of parents with a history of recurrent unipolar depression. Parents were recruited from general practices in South Wales UK (78%), from a previous database of adults with recurrent unipolar depression (19%) and from advertisements in primary care (3%). Families were excluded if parents had a previous bipolar or psychotic diagnosis, were not biologically related to the child, or the adolescent had an IQ < 50. A full description of the sample is given by Mars et al. (2012). Two families were later excluded as the affected parent was re-diagnosed with bipolar disorder. Data analysed in this report came from the first stage of the study. The analysed sample consisted of 335 offspring (139 boys). Mean age was 12.39 (SD=2.02).

As expected, the two datasets differed on a number of family stressors, as shown in Table 1. Specifically, the EPAD sample included higher proportions of participants that came from single parent households, had mothers with no formal educational qualifications and had a family income of below £10,000. In addition, the EPAD sample included a wider age and pubertal range (although modal pubertal status was the same).

Measures

Depression. Depressive symptoms in the STARS data were measured by self-report using the Short Mood and Feeling Questionnaire (Angold, Costello, Messer, & Pickles, 1995). The scale consists of 13 items designed to cover core symptoms of DSM-III-R depression (American Psychiatric Association, 1987) for children and adolescents. Items ask about symptoms during the past 3-months on a 3-point scale: true (2); sometimes true (1) and not true (0). Items are summed to produce a total score (possible range 0-26). A clinical cut point of 11 has been proposed for this measure (Angold, Erkanli, Silberg, Eaves, & Costello, 2002). Internal reliability was $\alpha=.89$, comparable to that reported by Angold et al. (1995) of $\alpha=.85$.

Depressive symptoms in the EPAD data were measured by parent and child ratings using the Child and Adolescent Psychiatric Assessment (CAPA; Angold & Costello, 2000), a semi-structured

diagnostic interview used to assess depressive disorders, as well as other psychiatric disorders. The total number of DSM-IV major depressive symptoms (possible range 0-9; American Psychiatric Association, 1994) was based on combined scores, whereby a symptom was coded as present if endorsed by either child or parent. For analyses focusing on Major Depressive Disorder (MDD), adolescents were classified as affected if this diagnosis was present at any of the 3 assessment stages (follow-up period 29 months, *SD*=5.39) of the EPAD study (7 boys; 30 girls met these criteria).

Cognitive ability. Cognitive ability in the STARS data was measured by the Cognitive Abilities Test (CAT3; Lohman et al., 2001), a standardised assessment which measures verbal reasoning, quantitative reasoning and non-verbal reasoning. The three scores were averaged to produce a cognitive ability score with higher scores indicating higher ability. CAT scores have been found to be highly reliable in terms of test-retest correlations and internal consistency estimates, and have strong predictive validity with later school performance (Strand, 2006). Cognitive ability in the EPAD data was analysed using the 10 subscales of the Wechsler Intelligence Scale for Children, which has also shown high reliability and validity (WISC-IV; Wechsler, 2003).

Stress. Stress was measured by negative life events for both the datasets using a version of the Life Events Checklist (Johnson & McCutcheon, 1980), listed in Appendix 1. The checklist for each consisted of 19 binary items (e.g. parents nagging/picking on you more, losing a close friend through arguments, doing badly in school work), which may have happened in the past year, summed to give a total score (possible range 0-19, higher scores indicating more negative life events). Life events were measured using child report for STARS and parent and child reports (coded as present if endorsed by either rater) for EPAD. Life events which could not be the result of characteristics of the participant (e.g. death of a grandparent) were coded as behaviour-independent (Appendix 1).

Additional measures for mediated moderation analysis.

Coping efficacy. Coping efficacy was measured in STARS using the Pearlin Mastery Scale (Pearlin & Schooler, 1978). Participants were asked to what extent seven statements describe themselves (e.g. There is really no way I can solve some of the problems I have; I have little control over the things that happen to me; I often feel helpless in dealing with the problems of life) on a 5-

point scale ranging from strongly disagree (1) to strongly agree (5). Items were reverse scored and summed to produce a total score (possible range 7-35, higher scores indicating greater coping efficacy, Cronbach's α=.60).

Coping efficacy was measured in EPAD using The General Self-Efficacy Scale (Schwarzer & Jerusalem, 1995). Participants were asked to what extent ten statements describe themselves (e.g. I can always manage to solve difficult problems if I try hard enough; I am confident that I could deal efficiently with unexpected events; I can remain calm when facing difficulties because I can rely on my coping abilities) on a 4-point scale ranging from not at all true (1) to exactly true (4). Items were summed to produce a total score (possible range 10-40, higher scores indicating greater coping efficacy, Cronbach's α=.85).

Sensitivity to environmental stressors. Sensitivity to environmental stressors was measured by sensitivity to the environment in STARS and stress sensitivity in EPAD. Sensitivity was measured in STARS using a self-report version of the Highly Sensitive Child Questionnaire, Short Form (Aron & Aron, 1997). The scale contained eight items (e.g. I am annoyed when people try to get me to do too many things at once; I don't like it when things change in my life; I don't like loud noises) on a 7-point scale: not at all (1); moderately (4); extremely (7). Items were summed to produce a total score (possible range 8-56, higher scores indicating greater sensitivity to the environment, Cronbach's α=.63).

Stress sensitivity was measured in EPAD using a version of the Life Events Checklist (see above, Johnson & McCutcheon, 1980). Participants were asked to rate the severity of each event which they stated having occurred on a 5-point scale from very unpleasant (1) to very pleasant (5). Scores were reversed so that a higher score indicates greater perceived severity and a score of 0 allocated if the event was not experienced. Items were summed to produce a total score (possible range 0-95, higher scores indicating greater stress sensitivity).

Statistical analysis

The association between cognitive ability and depressive symptoms was analysed using a series of multiple regression analyses with centred independent variables (Cohen, Cohen, Stephen, & Leona,

2003). The dependent variable was depressive symptoms. The first step entered stress as a single predictor variable, followed by cognitive ability in the second step. The interaction of cognitive ability x stress was added in the third step. Finally, the fourth step tested for moderation by stress and gender, which included gender, all possible additional two-way interaction terms and the interaction of cognitive ability x stress x gender. Gender was coded girls = 0, boys = 1. Significant interactions were followed-up with simple slopes analyses (Dawson & Richter, 2006) where low and high cognitive ability and stress were plotted as the sample mean ± one standard deviation. A final set of analyses exploring potential mediators of the hypothesised moderation was carried out with mediated moderation using Process, model 8, in SPSS (Hayes, 2013). Specifically, these analyses estimated the indirect effect of the cognitive ability x stress interaction term (*XW*) on depressive symptoms (*Y*) though the mediator (coping efficacy or sensitivity to environmental stressors; *M*). This is quantified as the effect of *XW* on *M* and of *M* on *Y*, keeping *X* and *W* (and *XW*) constant (Hayes, 2013). A 95% confidence interval (CI) was generated using N=5000 bootstrap samples. This tested the extent to which the hypothesised moderation of the association between stress and depressive symptoms by cognitive ability, was attributable to coping efficacy or sensitivity to environmental stressors.

Results

Table 2 presents descriptive data with gender differences and correlations by gender. Mean levels of negative life events were higher in EPAD than STARS (3.40 and 1.21 respectively). The proportion of participants meeting either the clinical cut-point for depression (STARS) or diagnostic criteria for depressive disorder (EPAD) was also higher in EPAD reflecting the fact that EPAD was selected on the basis of elevated familial risk for depression (13.6% in EPAD; 7.6% in STARS based on the suggested clinical cutpoint, Angold et al., 2002). Mean cognitive ability scores were 104 in STARS and 95 in EPAD. Independent-sample *t*-tests showed girls had higher levels of depressive symptoms than boys in both datasets, higher cognitive ability than boys in the EPAD data and more negative life events and sensitivity than boys in the STARS data. There were no gender differences for coping efficacy or independent life events. Bivariate correlations for each sample showed that depressive symptoms were associated with negative life events in both datasets (r=.34, p<.0001; r=.32, p<.0001)

and with cognitive ability in the EPAD data (r=-.13, p=.02) but not the STARS data (r=-.01, p=.88). Negative life events were also associated with cognitive ability in the EPAD data (r=-.16, p=.004) but not the STARS data (r=.04, p=.37).

Cognitive ability moderating the effect of stress on depressive symptoms and disorder

Findings testing the hypothesised moderating effects of cognitive ability on the association between stress and depressive symptoms are presented in Table 3. The first step of the analyses showed an association between stress and depressive symptoms (STARS β=.34, p<.0001; EPAD β=.30, p<.0001). The second step showed that this association remained when cognitive ability was entered into the model (STARS β=.34, p<.0001; EPAD β=.23, p<.0001) with no main effect of cognitive ability on depressive symptoms (STARS β=.0004, p=.92; EPAD β=-.07, p=.21). The third step showed no evidence of cognitive ability moderating the effects of stress on depressive symptoms, (interaction terms: STARS β=-.04, p=.34; EPAD β=-.07, p=.18) with a main effect of stress (STARS β=.33, p<.0001; EPAD β=.29, p<.0001) but not cognitive ability (STARS β=.004, p=.94; EPAD β=-.07, p=.21) on depressive symptoms. Further analyses revealed a three-way interaction between stress, cognitive ability and gender (Model 4; three-way-interaction terms: STARS β=.13, p=.03; EPAD β=.17, p=.01). When the samples were split by gender, two-way interactions of stress and cognitive ability were present in girls (STARS β=-.12, p=.05; EPAD β=-.17, p=.02) but not in boys (STARS β=.08, p=.21; EPAD β=.10, p=.27).

Simple slopes analyses were used to follow-up the three-way interaction and revealed a buffering effect of higher cognitive ability for girls (Figure 2, panels a and b) with a significantly stronger association between stress and depressive symptoms for girls of lower cognitive ability compared to girls of higher cognitive ability (STARS t=-2.18, p=.03; EPAD t=-2.86, p=.01). For boys, there was no significant difference between lower and higher cognitive ability in the association between stress and depressive symptoms (Figure 1, panels d and e; STARS t=1.10, p=.27; EPAD t=1.23, p=.22). Associations between stress and depression were stronger for girls with lower cognitive ability compared to boys of lower cognitive ability (STARS t=-2.38, p=.02; EPAD t=-1.96, p=.05). In contrast, there was some indication that associations between stress and depression were

marginally stronger for boys of higher cognitive ability compared to girls of higher cognitive ability in the high risk sample only (STARS t=-.88, p=.38; EPAD t=1.88, p=.06). There was no difference between slopes comparing boys of high cognitive ability and girls of low cognitive ability (STARS t=-.31, p=.76; EPAD t=.58, p=.56) or comparing boys of low cognitive ability and girls of high cognitive ability (STARS t=-1.16, p=.25; EPAD t=-.52, p=.61). In summary, higher cognitive ability was found to buffer the association between stress and depressive symptoms in girls, but not in boys.

Logistic regression analyses (EPAD only) showed that the three-way cognitive ability x stress x gender interaction replicated when a diagnosis of MDD was the outcome variable (*Exp(B)*=1.04, p=.04), along with main effects of stress (*Exp(B)*=1.36, p=.002) and gender (*Exp(B)*=.06, p=.02), but not cognitive ability (*Exp(B)*=.97, p=.09), and no significant two-way interactions (cognitive ability x stress: *Exp(B)*=.99, p=.38; cognitive ability x gender: *Exp(B)*=.86, p=.05; stress x gender: *Exp(B)*=1.55, p=.14); results for girls are shown in Figure 1 panel c and for boys in Figure 1 panel f. When the sample was split by gender, two-way interactions of stress and cognitive ability were not significant, although these showed the same direction of associations as was found for depressive symptoms in EPAD (girls: *Exp(B)*=.99, p=.38; boys: *Exp(B)*=1.04, p=.06).

Behaviour-independent life events

Restricting the analysis to independent life events outside the control of the individual (Appendix 1) allowed us to test the possibility that the interaction was driven by cognitive ability influencing levels of stress exposure. Tests of the moderating effect of cognitive ability on the association between stress and depressive symptoms replicated when restricting the measurement of stress to behaviour-independent life events (Table 4).

Mediated Moderation analyses

Follow-up mediated moderation analyses were conducted to test whether the observed moderating effect of cognitive ability on the association between stress and depressive symptoms for girls was accounted for by coping efficacy or sensitivity to environmental stressors. There was no evidence of mediated moderation for coping efficacy in either sample (STARS b=-.001, SE=.004, CI [-.01, .01];

EPAD *b*=-.001, SE=.001, CI [-.004, .0002]). There was some indication of mediated moderation for sensitivity to environmental stressors in both samples (STARS *b* =-.01, SE=.004, CI [-.02, -.001]; EPAD *b*=-.002, SE=.001, CI [-.01, -.0001]). That is, there was a small but significant indirect effect of the interaction between cognitive ability and stress on depressive symptoms for girls through environmental sensitivity. This suggests that higher cognitive ability in girls may be associated with fewer depressive symptoms partly via decreased sensitivity to environmental stressors when exposed to stressful life events. However, this only accounted for a small proportion of the observed interaction.

Discussion

We set out to examine the relationship between stress, cognitive ability and depressive symptoms, with the specific prediction that higher cognitive ability promotes resilience to stressful life events. We examined the role of cognitive ability as a moderator of the association between stressful life events and depressive symptoms in two adolescent samples where the degree of stress exposure was expected to differ. Based on the suggestion that individuals of lower cognitive ability may be less equipped to cope with stressful life events and thus be more vulnerable to their depressogenic effects (Barnett et al., 2006; Koenen et al., 2009), we predicted that higher cognitive ability would buffer the effect of stress on depressive symptoms. Given the increased vulnerability to depression following stress found in adolescent girls (Hankin & Abramson, 2001), we also predicted that the buffering effect of cognitive ability would be stronger in girls than boys.

The data supported our first hypothesis that higher cognitive ability moderated the effect of stress on depressive symptoms, although this was specific to girls. The observation that higher cognitive ability buffers against the depressogenic effects of stressful life events at least in adolescent girls is consistent with previous work which has found that those with higher cognitive ability show more positive outcomes such as academic, behavioural, social and psychiatric competence following stress (e.g. Masten et al., 1999; Pargas et al., 2010; Tiet et al., 1998). Our findings illustrated that higher cognitive ability was associated with resilience to life stress both in a sample of adolescents at increased familial risk of depression and a community sample of adolescents. One possible

explanation is that those of higher cognitive ability may show greater cognitive self-regulation under stress. Indeed, it has been suggested that cognitive ability may enable faster and more flexible responses to the environment (Boyce & Ellis, 2005). For instance, a range of cognitive processes which are at least partly indexed by the broad measure of cognitive ability, (such as working memory capacity and executive functions such as cognitive flexibility) may enable individuals to utilise controlled, effortful processing and thus generate strategic, flexible emotional responses under conditions of stress (Barrett, Tugade, & Engle, 2004). Thus, those of higher cognitive ability may be better able to inhibit negative behavioural and cognitive responses to stress. Consistent with resilience work which highlights cognitive ability as a predictor of better mental health particularly in those at high familial risk (Pargas et al., 2010), we found bivariate associations between cognitive ability and depression in our sample of children of depressed parents, but not in our community sample. Nevertheless, this association did not remain when controlling for exposure to stressful life events. Thus, rather than having a direct association, higher cognitive ability appears to be a protective factor for depression in the context of stressful life events. This is important when considering the role of cognitive ability in the aetiology of depression, suggesting that lower cognitive ability is not in itself a risk factor for depressive symptoms. It also suggests that girls of lower cognitive ability are a vulnerable group that may merit special consideration for supportive interventions.

Exposure to stressful life events can be controllable and partly dependent on behaviour (e.g. getting into a fight and being injured) or uncontrollable and independent of behaviour (e.g. death of a loved one) (Kendler & Baker, 2007). Thus, it is possible that individuals influence stress exposure through their own behaviour. We tested whether results replicated when stress exposure was restricted to independent life events and found the same pattern of results. The fact that results replicated gives greater confidence in our interpretation that cognitive ability modifies the influence of stress on the individual as opposed to influencing stress exposure (Hammen, 1991; Rice, Harold, & Thapar, 2003).

Our data also supported our second hypothesis that the moderating effect of cognitive ability would be greater for girls than boys. The finding that higher cognitive ability buffers the association between stress and depressive symptoms in girls only, is somewhat consistent with previous studies showing associations between higher cognitive ability and fewer depressive symptoms in girls but not

boys (e.g. Hatch et al., 2007). It is also consistent with data showing that from adolescence onwards, girls experience more social stressors and are more likely to develop depressive symptoms following social stressors (Thapar et al., 2012). We also observed suggestive evidence of an opposite effect in boys (that higher cognitive ability may be associated with increased stress-related depressive symptoms), at least in those at high risk. Some researchers have suggested that higher cognitive ability may be associated with a greater sensitivity to the environment, including stressors, although those studies did not test that explanation (Luthar, 1991; Zigler & Farber, 1985). It may be that the effect of cognitive ability on stress processing is different in boys and girls and that different cognitive processes may need to be targeted in boys and girls. These results are preliminary and will require replication, particularly given the small number of boys with major depressive disorder in the EPAD sample (6.4%). Nonetheless, prevalence rates were higher than typically reported (Costello et al., 2006), given that the analysis of MDD focussed on a high-risk sample.

We tested coping efficacy as a possible mediator for the moderating effect found in girls. This was based on reports of associations between cognitive reappraisal and resilience to stress-related depression (Southwick et al., 2005), and of suggestions that such cognitive vulnerabilities result in an increased risk of depression in girls (Hankin & Abramson, 2001). We found no evidence of mediated moderation. We also explored sensitivity to environmental stressors as a possible mediator of this moderation following suggestions that girls may find social stress more stressful, and be more vulnerable to depression following social stress than males (Juster et al., 2011; McCormick & Mathews, 2007; Stroud et al., 2002). We found some evidence of mediated moderation, with higher cognitive ability in girls leading to fewer depressive symptoms under stress at least partly via reduced environmental sensitivity. Thus, girls of higher cognitive ability may be better able to process information without impairing functioning under stress, due to greater capacity and efficiency of the cognitive system compared to girls of lower cognitive ability. In particular, our findings are consistent with 'hardware' interpretations that the observed buffering effect of higher cognitive ability may be due to greater cognitive capacity and efficiency (Brewin & Smart, 2005; Ellis, 1990). Under stress, controlled attentional/cognitive resources are reduced. However higher cognitive ability, in particular executive processes including working memory capacity, mental flexibility and inhibition, may

increase resistance to the attentional capture of negative information (Barrett et al., 2004; Cohen-Gilbert & Thomas, 2013; Joormann & Gotlib, 2010). It is also possible that girls of higher cognitive ability show lower biological stress responses but evidence to date is inconclusive (Flegr et al., 2012; Power, Li, & Hertzman, 2008; Slattery, Grieve, Ames, Armstrong, & Essex, 2013; Stawski et al., 2011). Moreover, the mediation effect was small, which may be partially due to measurement characteristics of our indices of environmental sensitivity. Despite being well established, internal validity was relatively low for the measure used in STARS and relied on having been exposed to stressful life events in EPAD. We therefore encourage future work looking at mechanisms of this association which would inform prevention programmes.

Our study has a number of strengths; particularly the use of independent datasets which differed on background stress levels. One sample included the offspring of depressed parents, a group known to differ from the general population on exposure to stress (Goodman & Gotlib, 1999). Findings replicated across both samples, with varying age ranges, measures of cognitive ability and measure of depressive symptoms and disorder, suggesting our findings are reliable. Although modal pubertal status was equivalent in both samples, the wider age range of the high risk sample could have been an issue, as this represents the entire range of puberty, whereas important gender differences in depression emerge at the onset of puberty (Thapar et al., 2012). However, replication of our findings in this sample suggests that the observed buffering effect of higher cognitive ability against stress-related depressive symptoms is not limited to this pubertal period. A limitation is that we were unable to investigate the possibility of shared genetic risk between cognitive ability, stress and depression. However, the pattern of results replicated when using behaviour-independent negative life events. It is therefore unlikely that the interaction we observed is due to life events exposure arising from the characteristics of the individual, including genetically influenced characteristics (e.g. Hammen, 1991; Rice et al., 2003). Thus, observed results for independent stressful life events are inconsistent with shared genetic risk for stress and either cognitive ability or depressive symptoms. An alternative possibility is that associations are due to the confounding effects of deprivation, which may be associated with cognitive ability, stress and depression (Collishaw et al., 2004). However, our results replicated when we repeated our analyses controlling for socioeconomic status (indexed by free-

school meals eligibility in STARS and the 2000 Standard Occupational Classification for EPAD) suggesting that this is not the case (results available from the first author). Another consideration is the inference of causality. We make a case for girls of higher cognitive ability being of lower risk of increased depressive symptoms following stress. Our results do not exclude the possibility of reverse causation. However, as noted, our results replicated for behaviour-independent life events. Cognitive ability shows high stability from childhood to old age (Deary, Whalley, Lemmon, Crawford, & Starr, 2000) and thus lower cognitive ability is a better candidate for an antecedent of, rather than a result of, depressive symptoms. Finally, our findings provide useful insight into the aetiology of depression during adolescence. This is particularly important given that this is a period of vulnerability for depression and long-lasting effects of stress (Lupien et al., 2009; Thapar et al., 2012). To investigate whether the buffering effects of higher cognitive ability are specific to this developmental period, future work could investigate the relationship between cognitive ability, stress and depression during childhood and adulthood.

Our findings suggest that low cognitive ability is not a significant vulnerability factor for depressive symptoms in itself. Instead, cognitive ability acts indirectly, with higher cognitive ability showing a buffering effect in girls consistent with the view that cognitive ability is one resilience promoting factor in the context of stress both in adolescents at low and high familial risk for depression.

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Table 1:

Sample characteristics

	STARS	EPAD
Age	11-12 years old	9-17 years old
Modal pubertal status (range)	Late pubertal (Pre pubertal – late pubertal)	Late pubertal (Pre pubertal – post pubertal)
Single parent household	14.7%	28.8%
Mothers with no formal educational qualifications	4.7%	16.0 %
Gross annual family income below £10,000	8.5%	13.6%

STARS = School Transition & Adjustment Research Study; EPAD = The Early Prediction of Adolescent Depression Study.

Table 2:

Descriptive statistics and correlations: depressive symptoms, cognitive ability, negative life events, coping efficacy and sensitivity to environmental stressors

	Whole sample Mean (SD)	Boys Mean (SD)	Girls Mean (SD)	Gender differences *t* (*df*)	Correlations 1.	2.	3.	4.	5.	6.
STARS (School Transition & Adjustment Research Study)										
1. Depressive symptoms (SMFQ)	3.70	3.08	4.30	3.00**		.04	.26***	-.24***	.28***	.07
	(4.35)	(3.69)	(4.83)	(446)						
2. Cognitive ability	103.51	103.71	103.32	-.35	-.04		-.08	.28***	.19*	.07
	(11.98)	(12.48)	(11.48)	(458)						
3. Negative life events	1.21	1.08	1.35	2.12*	.36***	-.01		-.25***	.20**	.65***
	(1.39)	(1.20)	(1.54)	(456)						
4. Coping efficacy	27.24	27.45	27.04	-.95	-.35***	.20**	-.45***		-.07	-.02
	(4.65)	(4.70)	(4.61)	(453)						
5. Sensitivity to the environment	33.08	31.62	34.43	3.68***	.29***	.05	.16*	-.26***		.15*
	(7.65)	(7.49)	(7.57)	(389)						

6. Behaviour-independent life events	.62	.55	.69	1.93		.19**	.08	.76***	-.26***	.09	
	(.78)	(69)	(.86)	(456)							
					1a.	1b.	2.	3.	4.	5.	6.
EPAD (Early Prediction of Adolescent Depression Study)											
1a. Depression (MDD)	.14	.06	.18	2.86**		.60***	-.24*	.29**	-.26*	.33**	.24*
	(.34)	(.25)	(.39)	(270)							
1b. Depressive symptoms (CAPA)	1.69	1.38	1.91	2.59*	.38***		-.09	.37***	-.20	.29**	.16
	(1.86)	(1.54)	(2.04)	(329)							
2. Cognitive ability	94.92	93.22	96.15	2.05*	-.19*	.17*		-.20*	.24*	-.15	-.15
	(12.86)	(11.34)	(13.75)	(328)							
3. Negative life events	3.40	3.35	3.44	.33	.31***	.29***	-.14		-.03	.79***	.69***
	(2.37)	(2.44)	(2.33)	(316)							
4. Coping efficacy	27.73	27.82	27.67	-.23	-.19*	-.25**	.21*	-.14		-.06	-.002
	(4.78)	(4.74)	(4.82)	(245)							
5. Sensitivity to the environment	8.28	7.37	9.00	1.70	.34***	.36***	-.21**	.69***	-.09		.65***
	(8.10)	(7.50)	(8.47)	(295)							

6. Behaviour-independent life events	1.25	1.22	1.28	.46	.20*	.11	-.07	.70***	-.02	.53***
	(1.21)	(.10)	(1.26)	(316)						

NB. Correlations for boys lie above the diagonal, correlations for girls lie below the diagonal. *p<.05, **p<.01, ***p<.001.

Table 3:

Associations between cognitive ability and depressive symptoms: total negative life events

	STARS (School Transition & Adjustment Research Study)					**EPAD (Early Prediction of Adolescent Depression Study)**				
	Model change		Coefficients			Model change		Coefficients		
	R^2 change	*p*	*β*	*B (S.E.)*	*p*	R^2 change	*p*	*β*	*B (S.E.)*	*p*
Step I: stress										
Intercept				3.70 (.19)	<.0001				1.69 (.10)	<.0001
Stress			.34	1.04 (.14)	<.0001			.30	.24 (.04)	<.0001
Step 2: stress and cognitive ability	<.0001	.93				.01	.213			
Intercept				3.70(.20)	<.0001				1.69 (.10)	<.0001
Stress			.34	1.04 (.14)	<.0001			.29	.23 (.04)	<.0001
Cognitive ability			.0004	.002 (.02)	.92			-.07	-.01 (.01)	.21
Step 3: moderation by stress	.002	.34				.01	.18			
Intercept				3.69 (.20)	<.0001				1.67 (.10)	<.0001

Stress			.33	1.03 (.14)	<.0001			.29	.23 (.04)	<.0001
Cognitive ability			.004	.001 (.02)	.94			-.07	-.01 (.01)	.21
Cognitive ability x Stress			-.04	-.01 (.01)	.34			-.07	-.004 (.003)	.18
Step 4: Moderation by stress and gender	.03	.01				.04	.004			
Intercept				4.12 (.27)	<.0001				1.87 (.13)	<.0001
Stress			.35	1.10 (.18)	<.0001			.32	.25 (.06)	<.0001
Cognitive ability			-.03	-.01 (.02)	.62			-.13	-.02 (.01)	.05
Gender			-.10	-.90(.39)	.02			-.12	-.44 (.21)	.03
Cognitive ability x Stress			-.13	-.04 (.02)	.03			-.17	-.01 (.004)	.01
Cognitive ability x Gender			.06	.03 (.03)	.39			.07	.02 (.02)	.28
Stress x Gender			-.06	-.30 (.29)	.30			-.002	-.002 (.09)	.98
Cognitive ability x Stress x Gender			.13	.06 (.03)	.03			.17	.02 (.01)	.01

Table 4:

Associations between cognitive ability and depressive symptoms: behaviour-independent negative life events

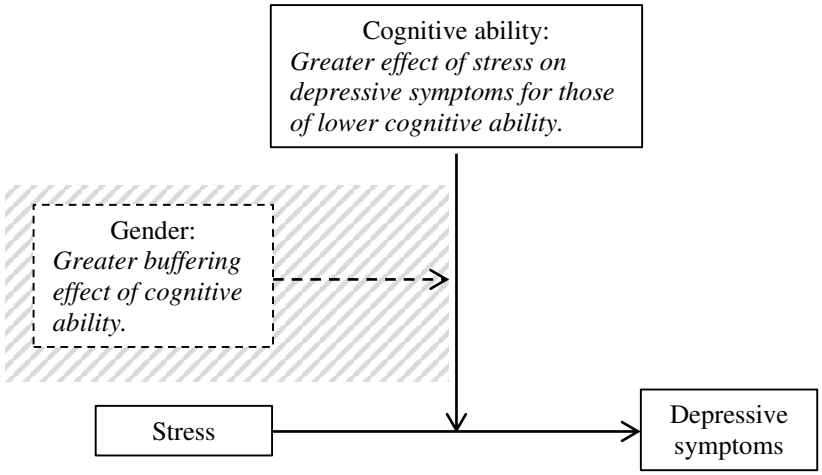
	STARS (School Transition & Adjustment Research Study)					EPAD (Early Prediction of Adolescent Depression Study)				
	Model change		Coefficients			Model change		Coefficients		
	R^2 change	*p*	*β*	*B (S.E.)*	*p*	R^2 change	*p*	*β*	*B (S.E.)*	*p*
Step I: stress										
Intercept				3.70 (.20)	<.0001				1.68 (.11)	<.0001
Stress			.15	.85 (.26)	.001			-.11	.17 (.09)	.06
Step 2: stress and cognitive ability	.0004	.670				.01	.06			
Intercept				3.70(.20)	<.0001				1.67 (.11)	<.0001
Stress			.16	.86 (.26)	.001			.10	.16 (.09)	.08
Cognitive ability			-.02	-.01 (.02)	.67			-.11	-.02 (.01)	.06
Step 3: moderation by stress	.01	.05				.01	.11			
Intercept				3.72 (.20)	<.0001				1.67 (.11)	<.0001

Stress			.17	.93 (.26)	.0004			.10	.16 (.09)	.08
Cognitive ability			-.02	-.01 (.02)	.61			-.11	-.02 (.01)	.06
Cognitive ability x Stress			-.09	-.04 (.02)	.05			-.09	-.01 (.01)	.11
Step 4: Moderation by stress and gender	.03	.01				.04	.01			
Intercept				4.25 (.28)	<.0001				1.91 (.14)	<.0001
Stress			.20	1.12 (.33)	.001			.10	.16 (.11)	.17
Cognitive ability			-.06	-.02 (.03)	.35			-.17	-.02 (.01)	.02
Gender			-.13	-1.16 (.41)	.01			-.14	-.52 (.21)	.02
Cognitive ability x Stress			-.16	-.08 (.03)	.01			-.17	-.02 (.01)	.01
Cognitive ability x Gender			.06	.03 (.04)	.35			.07	.02 (.02)	.34
Stress x Gender			-.09	-.81 (.54)	.14			.04	.10 (.19)	.62
Cognitive ability x Stress x Gender			.12	.09 (.05)	.05			.14	.03 (.02)	.03

Figure 1:

Conceptual figure of the tested models

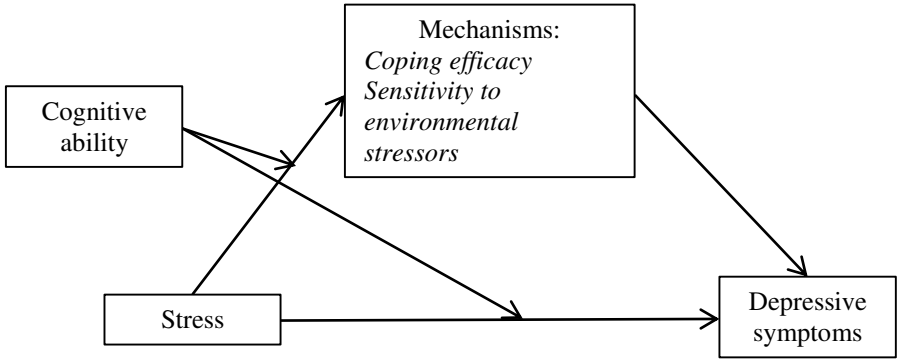
a) Cognitive ability moderates the effect of stress on depressive symptoms



i) Moderation (Hypothesis 1)

ii) Moderation greater in girls than boys (Hypothesis 2)

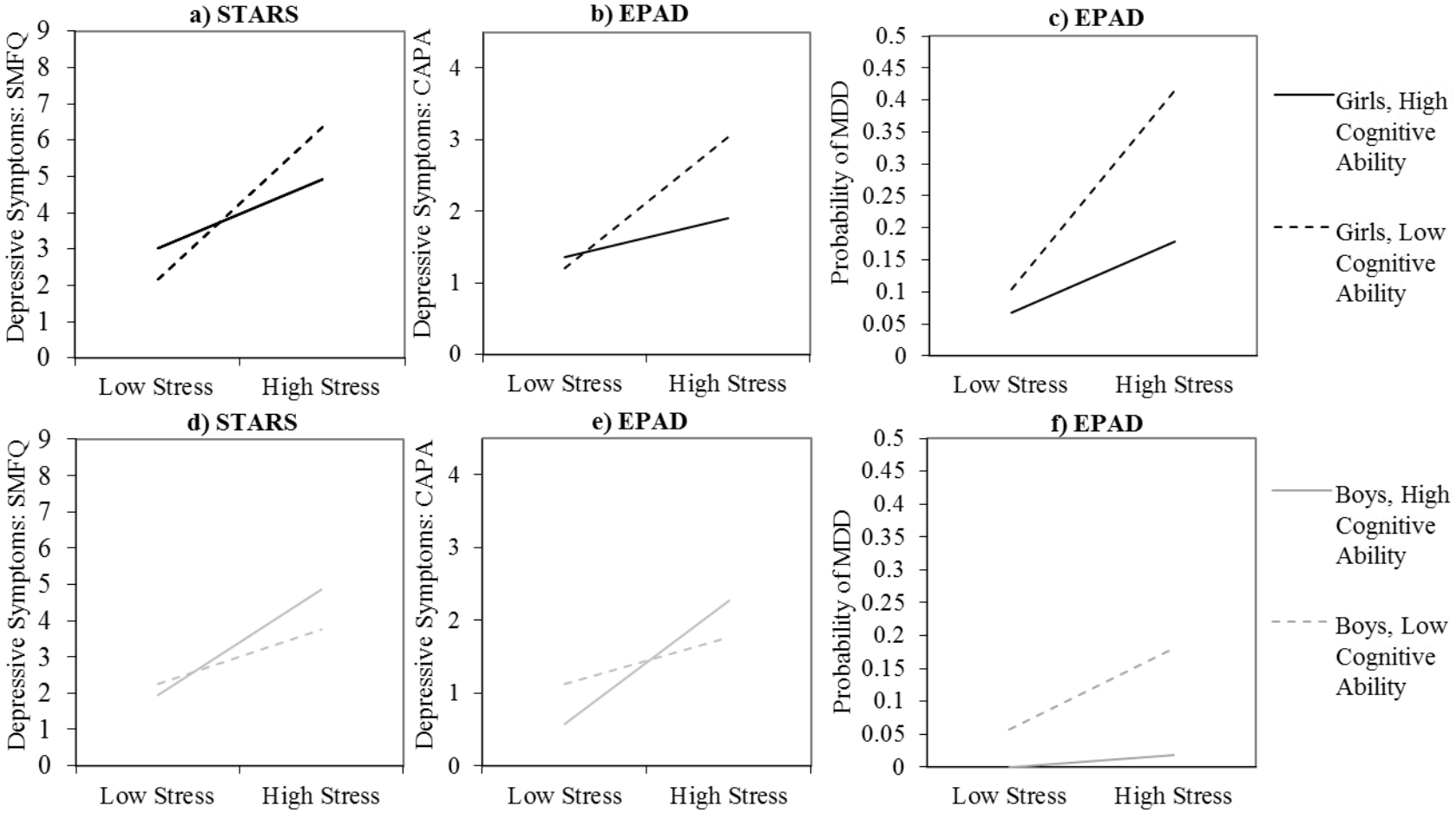
b) Mechanisms of the moderating effect of cognitive ability on stress-related depressive symptoms in girls



Mediated moderation (following Hypothesis 2)

Figure 2:

Simple slopes analysis: associations between cognitive ability and depressive symptoms/MDD for girls and boys


a) STARS
b) EPAD
c) EPAD
d) STARS
e) EPAD
f) EPAD
Depressive Symptoms: SMFQ
Depressive Symptoms: CAPA
Probability of MDD
Low Stress
High Stress
Girls, High Cognitive Ability
Girls, Low Cognitive Ability
Boys, High Cognitive Ability
Boys, Low Cognitive Ability

NB. Low and high stress and cognitive ability and are plotted as the mean ± 1SD for the two datasets. All predictor variables are centred. STARS = School Transition & Adjustment Research Study; EPAD = The Early Prediction of Adolescent Depression Study.

Appendix 1:

Negative life events

1. Serious illness in family member*
2. Increased quarrelling between parents
3. Death of parent/brother/sister*
4. Death of grandparent*
5. Death of close friend*
6. Serious illness/injury to close friend*
7. Parent in trouble with police*
8. Parent going to prison*
9. Doing badly in an exam
10. Parents being less interested/loving
11. Parents nagging/picking on you more
12. Serious illness to you*
13. Doing badly in (school) work
14. Close friend moves away*
15. Losing a close friend through arguments
16. Death of a pet*
17. Mother losing job*
18. Father losing job*
19. Being bullied

*Coded as behaviour-independent life events