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Objective: We examined whether an empirically based, randomised controlled trial of a preventive intervention for divorced mothers and children had a long-term impact on offspring cortisol regulation.
Design: Divorced mothers and children (age 9–12) were randomly assigned to a literature control condition or the 11-week New Beginnings Program, a family-focused group preventive intervention for mothers and children in newly divorced families.
Main Outcome Measures: Fifteen years after the trial, offspring salivary cortisol ($n = 161$) was measured before and after a social stress task.
Results: Multilevel mixed models were used to predict cortisol from internalizing symptoms, externalizing symptoms, group assignment and potential moderators of intervention effects. Across the sample, higher externalizing symptoms were associated with lower cortisol reactivity**.** There was a significant group-by-age interaction such that older offspring in the control group had higher reactivity relative to the intervention group, and younger offspring in the control group exhibited a decline across the task relative to younger offspring in the intervention group.
Conclusions: Preventive interventions for youth from divorced families may have a long-term impact on cortisol reactivity to stress. Results highlight the importance of examining moderators of program effects.

Keywords: parental divorce; cortisol; intervention; externalizing

Children in divorced families may experience a number of adversities, including interparental conflict, financial stressors, parental depression and separation from a parent. A considerable literature documents health risks associated with such family-related adversities (Miller, Chen, & Parker, 2011). Meta-analyses find that youth in divorced families have more conduct, internalizing, social and academic problems than those in nondivorced families (e.g. Amato, 2001). Parental divorce has also been empirically linked to tobacco use, physical symptoms in children and adolescents (Troxel and Matthews, 2004), health problems in adolescence and adulthood (Fabricius & Luecken, 2007; Fuller-Thomson & Dalton, 2012; Roustit et al., 2011), and decreased longevity (Larson & Halfon, 2013).

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The impact of adverse experiences during a sensitive period of neurological development on the calibration and function of biological stress response systems is a commonly theorised mechanism linking early adversity and health (e.g. Luecken & Lemery, 2004; Miller et al., 2011; Repetti, Taylor, & Seeman, 2002). The hypothalamic-pituitary-adrenal (HPA) axis, which regulates the production and release of the hormone cortisol, undergoes extensive development during childhood and adolescence, and is highly sensitive to environmental influences (Gunnar, Talge, & Herrera, 2009). A large research literature demonstrates that adverse childhood experiences can significantly modify HPA activity, but yields conflicting results about the form or direction of effects. Prolonged or intense childhood stress can lead to exaggerated or attenuated reactivity to psychosocial stress, higher or lower basal cortisol, or flattened diurnal slopes (Gunnar & Quevedo, 2007; Koss et al., 2013; Miller, Chen, & Zhou, 2007). Both exaggerated and attenuated cortisol reactivity to stress and daily cortisol output have been linked to long-term mental and physical health problems, including affective and externalizing disorders, heart disease and infectious diseases (Heim, Ehlert, & Hellhammer, 2000; Miller et al., 2007; Shirtcliff, Peres, Dismukes, Lee, & Phan, 2014). Research suggests complex relations between childhood adversity and associated cortisol alterations, with exaggerated reactivity as an outcome for some vulnerable youth, and attenuated reactivity for others.

The few studies that have examined the impact of parental divorce and interparental conflict on offspring cortisol reactivity find similarly complex results. Kraft and Luecken (2009) found lower cortisol reactivity to a videotaped lab-based social challenge task (a 4-min preparation period followed by a 4-min speech defending oneself from a false accusation of shoplifting; Saab, Matthews, Stoney, & McDonald, 1989) in emerging adults from divorced families compared to young adults from married families. Bloch, Peleg, Koren, Aner, and Klein (2007) reported that parental divorce in childhood predicted lower cortisol response to a corticotropin-releasing hormone stimulation test. Although not specific to parental divorce, both exaggerated and attenuated cortisol reactivity to simulated conflict between parents have been reported in grade-school-aged children (Davies, Sturge-Apple, Cicchetti, 2008, 2011; Koss et al., 2013). Higher cortisol reactivity to a 10-min role-play peer conflict task was exhibited among young adults exposed to high interparental conflict during childhood relative to those who experienced moderate conflict (Hagan, Roubinov, Purdom Marreiro, & Luecken, 2014). The investigation of predictors of individual differences in outcomes is a critical direction for future research.

Concurrent or life course psychological symptomatology may help explain the impact of parental divorce on cortisol regulation. A recent study of adults exposed to childhood adversity found divergent patterns of cortisol reactivity depending on whether or not the participant also experienced recurrent psychological problems (Goldman-Mellor, Hamer, & Steptoe, 2012). Children who experience parental divorce and interparental conflict are at elevated risk of internalizing and externalizing problems (Amato, 2001; Kim, 2011), and a number of studies document cortisol dysregulation in children and adults with psychological disorders. Internalizing symptoms, for example, have been associated with higher baseline cortisol and cortisol stress reactivity (Lopez-Duran, Kovacs, & George, 2009), while reduced basal cortisol and lower cortisol reactivity have been associated with externalizing problems (Alink et al., 2008; Hagan, Roubinov,

Mistler, & Luecken, 2014). Offspring of parental divorce who develop internalizing or externalizing problems may show diverging patterns of cortisol reactivity.

Given clear evidence that childhood adversity can impact health across the lifespan, the development of interventions capable of mitigating negative effects is critical (Shonkoff, Boyce, & McEwen, 2009). As a potential mediator, the HPA axis represents an intriguing target of interventions for at-risk youth (e.g. Bruce, Gunnar, Pears, & Fisher, 2013; Cicchetti & Gunnar, 2008). Randomised controlled trials (RCTs) offer a powerful means to assess the ability of interventions to alter biological functions because the process of randomisation minimises the possibility that programme effects are due to pre-existing biological or individual characteristics. Several studies provide intriguing evidence of short- and long-term adrenocortical benefits of interventions for children in foster care and parentally bereaved children (Brotman et al., 2007; Fisher, Stoolmiller, Gunnar, & Burraston, 2007; Fisher, Van Ryzin, & Gunnar, 2011; Luecken et al., 2010; O'Neal et al., 2010).

Although recent findings are promising, it remains an open question whether the long-term impact of parental divorce on neuroendocrine response systems can be modified, and whether there are individual differences in intervention effects – a task that requires consideration of programme moderators. Examination of programme moderators has considerable public health significance as it can help identify who is most likely to benefit from intervention, the conditions under which benefits are maximised, and individual differences in treatment response (Kraemer, Wilson, Fairburn, & Agras, 2002; Kraemer, Frank, & Kupfer, 2006; MacKinnon, Lockhart, Baraldi, & Gelfand, 2013). Ideally, moderators are baseline characteristics (e.g. age and sex) that are uncorrelated with programme condition (Kraemer et al., 2002). Moderators of intervention effects on psychological outcomes are commonly examined, but little is known about moderators of programme effects on neurobiological systems.

The New Beginnings Program

The New Beginnings Program (NBP) is an 11-week family-focused intervention for recently divorced mothers and children. The NBP was designed to promote children's positive adaptation by minimizing risk factors such as exposure to interparental conflict and maximizing protective factors such as high-quality parent–child relationships. An RCT of the NBP conducted when children were 9–12 years old revealed programme benefits on several indices of mental health at post-test, six years and 15 years later (Wolchik et al., 2000, 2002, 2013; Wolchik, Sandler, Weiss, & Winslow, 2007).

The current study investigated NBP effects on cortisol 15 years later. Drawing from research about intervention effects on psychosocial functioning in at-risk youth, we theorised that age and gender would moderate NBP effects on neurobiological systems. Sensitive periods for environmental influences on brain development occur at different ages for varied aspects of cognitive, emotional and biological development, and children are particularly receptive to negative experiences during sensitive periods. The peripubertal period represents one such sensitive period. The effects of parenting interventions on child psychosocial functioning have been found to vary by age (e.g. Jayson, Wood, Kroll, Fraser, & Harrington, 1998; Sandler et al., 1992). For example, the Incredible Years Programme found stronger effects on conduct problems for younger children (Gardner, Hutchings, Bywater, & Whitaker, 2010). Further, age effects on

HPA activity and the neurobiological consequences of stress have been reported (Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004; Miller et al., 2011). Sex differences in children's mental health outcomes have been reported for interventions after parental bereavement or divorce (e.g. Sandler et al., 2003; Wolchik et al., 2013). Moreover, sex differences in HPA activity, the impact of childhood adversity on HPA activity and the impact of parental divorce on adult physical health outcomes have been noted (Doom, Cicchetti, Rogosch, & Dackis, 2013; Fuller-Thomson & Dalton, 2012; Trickett, Gordis, Peckins, & Susman, 2014).

The current study examined the impact of NBP participation on cortisol reactivity to a psychosocial challenge in young adulthood. Prior research finds evidence of both blunted and exaggerated reactivity in youth affected by adversity relative to a non-affected group. The current sample was all affected by parental divorce, and we expected that those who received the preventive intervention would exhibit less evidence of dysregulation (exaggerated or blunted responses) relative to the control group. We also examined the relation of concurrent mental health to cortisol reactivity. Finally, we evaluated child age and gender as moderators of intervention effects on cortisol.

Materials and methods

Participants

Participants included 161 young adults from divorced families who participated in a RCT of the NBP 15 years earlier. Demographic information is displayed in Table 1 and a CONSORT flowchart is shown in Figure 1. Most families (80%) were recruited from randomly selected court records of divorce decrees within two years of the intervention's start; the remainder responded to media advertisements. Eligibility criteria included: (1) primary residential parent was female, (2) a 9–12 year-old child who resided at least 50% of the time with the mother, (3) neither mother nor any child was in treatment for mental health problems and (4) mother had not remarried, did not plan to remarry and did not have a live-in boyfriend (see Wolchik et al., 2000 for complete eligibility criteria). Children signed informed assent forms; parents and youth older than 18 signed informed consent.

Assessments were conducted in participants' homes at baseline (prior to assignment to condition, W0), post-test (W1) and at 3-month (W2), 6-month (W3), 6-year (W4) and 15-year (W5) follow-ups. The current analyses include W0 and W5 data. At W0, families were randomly assigned to one of three conditions: Mother Program ($n = 81$), Mother and Child Programme ($n = 83$) or Literature Control (LC; $n = 76$). Prior analyses indicated that the child component did not increase programme benefits on mental health outcomes beyond the mother component (Wolchik et al., 2002, 2013)[1]; therefore, the Mother and the Mother and Child intervention groups were combined for analyses, hereafter labelled the NBP.

Of the 240 youth assigned to condition, 194 (89.6% of the families; NBP $n = 134$; LC $n = 60$) completed the 15-year follow-up (see Figure 1). Attrition analyses showed that the rate of attrition did not differ significantly across NBP (9.8%) and LC (11.8%) (χ^2 [1, $N = 240$] = .24, $p = .65$) conditions. We found no differential attrition or attrition by group interaction effects on demographic or psychosocial risk variables from W0.

Table 1. Sample characteristics.

	Full sample	NBP	LC	*p*-value
Age at W0 (study entry; *M*, *SD*)	10.76 (1.4)	10.77(1.1)	10.67 (1.1)	.62
Age at W5 (*M*, *SD*)	25.6 (1.2)	25.6 (1.2)	25.5 (1.2)	.73
Age at parental divorce (*M*, *SD*)	9.7 (1.3)	9.8 (2.3)	9.7 (1.2)	.63
Sex (*N*, %)				
Male	84 (52%)	61 (54%)	23 (51%)	.49
Female	77 (48%)	53 (46%)	24(49%)	
Ethnicity (*N*, %)				
Hispanic	18 (11%)	14 (12%)	4 (9%)	.68
Anglo/Caucasian	132 (82%)	92 (81%)	40 (85%)	
African American	5 (3%)	5 (4%)	0 (0%)	
Native American	3 (2%)	0 (0%)	3 (7%)	
Asian/Pacific Islander	3 (2%)	3 (3%)	0 (0%)	
Hormonal contraceptive use (*N*, %)	24 (31%)	17 (32%)	7 (29%)	.80
Regular Smoker (*N*, %)	50 (31%)	35 (31%)	15 (33%)	.82
Time from divorce to study entry (*M*, *SD*)[1]	12.5 (6.3)	12.4 (6.3)	12.5 (6.5)	.89
Use of medications (*N*, %)[2]	37 (23%)	26 (23%)	11 (23%)	.80
W0 Psychosocial risk (*M*, *SD*)[3]	0 (1.0)	.081 (.99)	−.15 (.69)	.15
W5 Internaliz. Symptoms (*M*, *SD*)[4]	56.4 (6.8)	56.54 (6.5)	55.8 (6.4)	.63
W5 Externaliz. Symptoms (*M*, *SD*)[4]	49.2 (10.8)	48.8 (10.9)	49.2 (9.3)	.80
W5 Total Problems Index (*M*, *SD*)[4]	50.2 (9.2)	50.5 (9.4)	49.4 (8.6)	.51
Pre-task cortisol (nmol/L; *M*, *SD*)[5]	2.41 (1.3)	2.37 (1.3)	2.53 (1.3)	.77
Post-task cortisol (nmol/L; *M*, *SD*)[5]	2.34 (1.5)	2.29 (1.6)	2.49 (1.4)	.65
20-min post cortisol (nmol/L; *M*, *SD*)[5]	2.48 (1.9)	2.46 (2.0)	2.61 (1.7)	.84
40-min post cortisol (nmol/L; *M*, *SD*)[5]	2.02 (1.3)	1.98 (1.3)	2.14 (1.3)	.66

Note: "NBP" = intervention participants; "LC" = participants in the control condition.
[1]In months.
[2]Includes allergy, antidepressant, antianxiety or other medications that could potentially affect cortisol.
[3]*z*-score.
[4]*t*-score.
[5]Non-transformed data.

Cortisol samples were not obtained from 12 participants (eight refused and four lived outside the country). Two participants had cortisol levels outside normal physiological levels (>50 nmol/L; Nicolson, 2008), indicating interference in the assay. An additional nineteen (11 from the NBP group and eight from the LC group) were excluded a priori from analyses due to pregnancy ($n = 8$), thyroid or other medications that affect glucocorticoids ($n = 9$), or cortisol levels >4 SD's from the mean ($n = 2$; Nicolson, 2008).[2] The final sample consisted of 115 participants in the NBP and 46 in the LC group.

Procedure

All components of the assessment were conducted at participants' homes. Saliva samples were collected before, immediately after, and 20 and 40-min after a modified Trier Social Stress task (TSST; Kirschbaum, Pirke, & Hellhammer, 1993) that included 3 min of mental arithmetic and an 6-min video-recorded speech about personal strengths and weaknesses (2 min preparation, 4 min speech). The mental arithmetic task included three trials of serial subtraction problems performed out loud. A new starting number

Assessed for eligibility (n=1331)

Excluded (n= 1091)
- Not meeting inclusion criteria (n=709)
- No recruitment visit (n=218)
- Declined to participate (n= 112)
- No pretest (n=26)
- Terminated between pre-test and assignment (n= 26)

Randomized (n= 240)

Wave 0 (pre-test)

Assigned to intervention (NBP) **(n=164)**
- Received intervention (n= 164)

Assigned to control (LC) **(n=76)**
- Received control condition (n=76)

Wave 1 (post-test)

Lost to follow-up: Refused (n= 4)

Lost to follow-up (n = 0)

Wave 2 (3 mos.)

Lost to follow-up: Refused (n= 3)

Lost to follow-up: Refused (n = 1)

Wave 3 (6 mos.)

Lost to follow-up: Refused (n= 4)

Lost to follow-up: Refused (n = 2)

Wave 4 (6 years)

Lost to follow-up: Refused (n= 14)

Lost to follow-up: Refused (n= 6)
Unable to Locate (n = 2)

Wave 5 (15 years)

Lost to follow-up: Refused (n=14),
Unable to reach/locate (n=13),
Deceased (n=3)

Refused cortisol sampling (n=5)
Out of country (n=2)
Cortisol outside physio range (n=1)

Lost to follow-up: Refused (n=5),
Unable to reach/locate (n=11)

Refused cortisol sampling (n=3)
Out of country (n=2)
Cortisol outside physio range (n=1)

Analyses

Cortisol ≥ 4 SD from mean (n=1)
Pregnant/medications: (n=10)
Data Analysed: (**n=115**)

Cortisol ≥ 4 SD from mean (n=1)
Pregnant/medications: (n= 7)
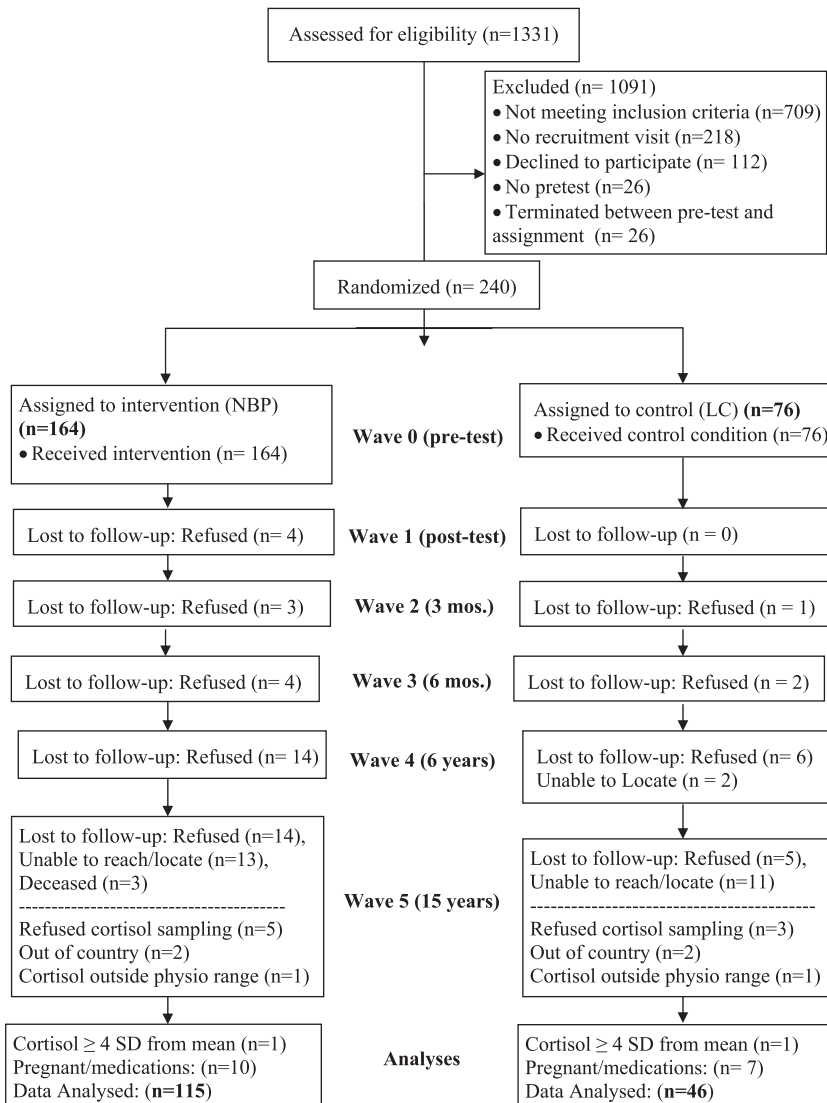Data Analysed: (**n=46**)

Figure 1. Flow of study participants.

was provided each minute, adjusted for difficulty to hold effort constant across participants. It was conducted under time pressure, with prompting from the research assistant (e.g. 'faster', 'incorrect, begin again with ___'). The speech was video-recorded and given in front of the experimenter who displayed a neutral expression and took notes on a clipboard throughout the speech. In addition, prior to the speech task, participants were told their speech would be evaluated and graded by a team of experts. An a priori start time for cortisol samples was 2–8:30 PM (range = 1:54–8:27 PM; mean = 5:55 PM).

Intervention and control conditions

Intervention condition (NBP)

The Mother Program consisted of 11 group sessions (1.75 h each; average group size = 9) and two individual sessions. Groups were led by two master's level clinicians using structured manuals. Mother Program sessions focused on skills to improve mother–child relationship quality and effective discipline, decrease barriers to child contact with the father and decrease interparental conflict. In the Mother and Child Program, children participated in separate groups that aimed to increase active coping, decrease avoidant coping and negative appraisals of divorce stressors, and improve mother–child relationship quality. Clinical methods were based on social learning and cognitive behavioural theories. As noted earlier, the two intervention conditions were combined in the analyses, and the combined group is referred to as NBP. See Wolchik et al. (2000) for a detailed description of the clinical methods.

Control condition (LC)

In the LC, mothers and children each received three books about children's divorce adjustment, along with a syllabus to guide their reading. Families received their first books a week after assignment to condition, their second books, three weeks later and their last books, six weeks after assignment to condition (see Wolchik et al., 2000 for details).

Measures

Mental health problems

Mental health problems during the past six months were assessed at W5 using the Adult Self Report form (Achenbach & Rescorla, 2003). Subscale scores included internalizing problems ($\alpha = .90$), externalizing problems ($\alpha = .84$) and a total problems index ($\alpha = .90$).

Baseline psychosocial risk

A baseline risk index served as a control variable in the prediction of cortisol reactivity. All measures in the risk index were completed by mothers at W0, prior to assignment to condition. The risk index was the composite of standardised scores on child externalizing problems (Child Behaviour Checklist; Achenbach, 1991; $\alpha = .88$) and divorce-related adversity, including interparental conflict (Children's Perception of Interparental Conflict, Grych, Seid, & Fincham, 1992; $\alpha = .88$), post-divorce negative events (Negative Life Events Scale; Sandler, Wolchik, & Braver, 1988), maternal mental health problems (Psychiatric Epidemiology Research Interview; Dohrenwend, Shrout, Egri, & Mendelsohn, 1980; $\alpha = .91$), low contact with non-residential father (mother reported visits with father in the past month) and low per capita income (total income divided by the number of household members). The index was predictive of mental disorder and substance use in the LC group six years later (Dawson-McClure, Sandler, Wolchik, & Millsap, 2004).

Cortisol sampling

Samples were obtained with the Salivette (Sarstedt, Rommelsdorf, Germany) and shipped on dry ice to Salimetrics (State College, PA) for analysis of free cortisol using high-sensitive enzyme immunoassay. The test has a range of sensitivity from .007 to 1.8 μg/dl, and mean intra-and inter-assay coefficients of variation 4.13 and 8.89%. Cortisol values were transformed from μg/dl to nmol/L and then log-transformed to correct for deviations from normality.

Statistical analyses

Preliminary analyses

We tested group equivalence on W0 demographic variables (ethnicity, sex, income, psychosocial risk, age at the divorce and months since the divorce) using χ^2 or *t*-statistics. Next, we evaluated covariates and confounders. Covariates do not explain relations between the independent variable (IV: group assignment) and dependent variable (DV: cortisol reactivity) because they are not in a causal pathway between them (MacKinnon & Luecken, 2008). Potential covariates included W0 demographics (e.g. sex, income and ethnicity) as well as information from the day of testing (W5; time of day, recent meals, exercise, caffeine consumption, medications or hormonal contraceptives). It is not necessary to control for covariates, but doing so may increase efficiency in estimation (Sauer, Brookhart, Roy, & Vanderweele, 2013). Confounders are variables potentially associated with both the IV and the DV that could introduce bias into estimations (e.g. smoking). Covariates and confounders were selected empirically with the same criteria: any variable significantly related to the intervention condition and/or cortisol reactivity was selected for inclusion in analyses.

Analyses of outliers, conducted by evaluation of Cook's D, studentized residuals and leverage scores, identified two influential cases. The first, from the NBP group, self-reported heavy non-prescription drug use (99th percentile) and had cortisol levels >3 SD from the mean. The second, from the LC group, began cortisol sampling at 1:54 PM, had smoked 15 min prior to the first sample, and reported heavy alcohol use and binge-drinking (95th percentile). Although the statistical significance of the results was not affected by the removal of these cases, the graphical pattern of results was influenced by their extreme values. These two cases were not considered reliable and were removed from the analyses.

A preliminary analysis was conducted to evaluate whether the challenge task resulted in significant cortisol reactivity across the sample. A multilevel model revealed a significant quadratic pattern (capturing cortisol's rise and fall across the task), $p = .008$, indicating reactivity to the task across the sample.

Primary analyses

We used multilevel modelling (MLM) to evaluate group differences in cortisol reactivity. The data were modelled using SPSS MIXED, with the repeated cortisol measures forming the within-person dimension. A variable 'time' reflected within-person cortisol sampling order ('0' = pre-task, '1' = immediately post-task, '2' = 20 min post-task, '3' = 40 min post-task), and was included as a random linear effect. 'Reactivity' was

modelled with the quadratic term 'time X time'. Group assignment served as the between-persons dimension, coded as NBP group = 1 and LC group = −1. Sex was coded as 0 = male and 1 = female. Continuous variables were centred at the sample mean. Significant models were re-estimated with the inclusion of concurrent mental health symptoms.

Results

Evaluation of group equivalence

The NBP and LC groups did not significantly differ on W0 measures of age, ethnicity, sex, income, psychosocial risk, age at the divorce, months since the divorce or concurrent internalizing symptoms, externalizing symptoms or total behavioural problems (see Table 1). On the day of testing (W5), the groups did not differ with regard to session start time, time of saliva sampling, cigarettes smoked, caffeine or energy drinks, BMI, exercise, medications, time of most recent meal or negative mood before or after the task (all p's > .18).

Selection of covariates and confounds

Zero-order correlations are shown in Table 2, and demonstrate significant relations among time of day and participant sex with cortisol levels at individual time points. Additional analyses were conducted using MLM to evaluate effects of covariates or confounds on cortisol reactivity. Men showed stronger cortisol reactivity (quadratic pattern of response) than women ($p = .01$). The time of day predicted cortisol intercept ($p = .02$), but did not predict reactivity ($p = .25$). Age, ethnicity, W0 income, W0 psychosocial risk, child age at divorce and time since the divorce did not predict cortisol intercept, slope or reactivity, nor did smoking, caffeine or energy drinks, BMI, exercise, pre- or post-task negative mood, medications or recent meals on the day of testing (all p's ≥ .10). Therefore, time of day and sex were included as covariates in all models.

Mental health symptoms and cortisol reactivity

A multilevel model controlling for time of day and sex found that higher externalizing symptoms were associated with lower cortisol reactivity (quadratic effect), $\beta = .001$, 95% CI [.0001, .001], $p = .047$, and a declining linear slope, $\beta = −.003$, 95% CI [−.005, −.001], $p = .017$ (see Figure 2). Internalizing symptoms did not predict cortisol reactivity (quadratic effect), $p = .22$, linear slope, $p = .12$ or intercept, $p = .16$. The total mental health problems index was a statistically significant predictor of cortisol linear slope, $\beta = −.004$, 95% CI [−.006, −.001], $p = .003$, such that more problems were associated with a more steeply declining slope across the task. Total mental health problems did not predict cortisol reactivity ($p = .06$) or intercept ($p = .53$).

Main effect of intervention on cortisol reactivity

A multilevel model controlling for time of day and sex did not find significant prediction of cortisol reactivity by group, $p = .57$. The linear slope, $p = .67$, and intercept,

Table 2. Zero-order correlations[1].

	1	2	3	4	5	6	7	8.	9	10
(1) W0 age	1.0									
(2) Sex	−.047	1.0								
(3) Time of day	.044	.021	1.0							
(4) Time since divorce	−.047	.013	.053	1.0						
(5) W5 Internalizing	−.089	−.038	.020	−.019	1.0					
(6) W5 Externalizing	−.020	.050	.042	−.030	.74**	1.0				
(7) W5 Total Problems	−.066	−.064	−.003	−.032	.65**	.73**	1.0			
(8) Baseline cortisol	−.026	−.055	−.20**	−.068	−.037	−.021	−.036	1.0		
(9) Post-task cortisol	−.046	−.16*	−.13***	−.006	−.033	−.019	−.028	.81**	1.0	
(10) 20-min post cortisol	−.050	−.25**	−.08	−.006	−.11	−.13***	−.22**	.62**	.83**	1.0
(11) 40-min post cortisol	−.044	−.13	−.14*	−.027	−.054	−.045	−.12	.66**	.82**	.83**

[1]Non-transformed cortisol (nmol/L); sex is coded male = 0, female = 1; Time of day is in minutes past midnight; Time since divorce is in months; Internalizing symptoms, Externalizing symptoms and Total Problems are *t*-scores.
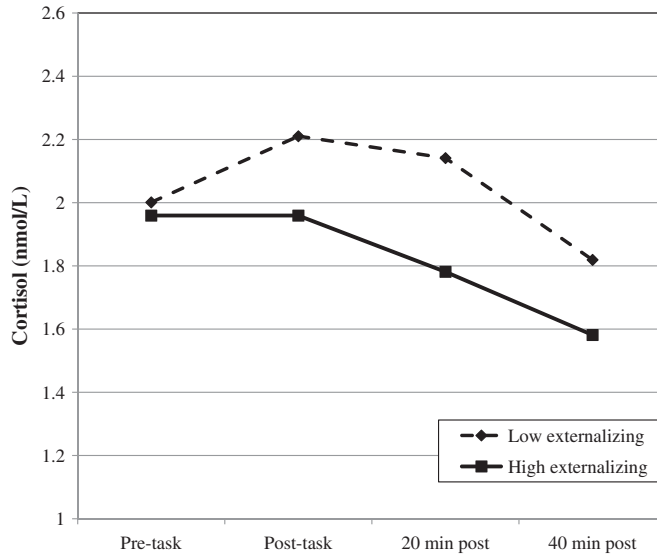*$p < .05$; **$p < .01$; ***$p < .10$.

Figure 2. Cortisol reactivity by externalizing symptoms.
Note: Covariates = time of day and sex. Externalizing symptoms was analysed as a continuous variable but set to one SD above and below the mean for display purposes. Predicted log-transformed cortisol values were subjected to antilog-transformation in order to display cortisol values in an interpretable metric.

$p = .51$, were also not statistically significant. Inclusion of externalizing symptoms or total mental health problems in the models did not significantly change the results.

Moderation of intervention effect by youth age

We next examined individual differences in response to the intervention. The group-by-age interaction was a statistically significant predictor of cortisol reactivity, $\beta = .011$, 95% CI [.004, .018], $p = .004$ and linear slope, $\beta = -.040$, 95% CI [−.064, −.016], $p = .001$, but did not predict the intercept, $p = .56$. The group-by-age interaction remained statistically significant after adjusting for externalizing symptoms, $\beta = .012$, 95% CI [.005, .019], $p = .002$ and total mental health problems, $\beta = .011$, 95% CI [.004, .019], $p = .003$. As shown in Figure 3, the NBP participants showed similar reactivity and recovery regardless of age, while older LC participants had elevated reactivity and younger LC participants exhibited declining cortisol across the task.

Probing the group*age interaction

The significant group-by-age interaction was probed by dividing the sample into younger (age ≤ 10.5 at program entry; $n = 79$) and older (age > 10.5; $n = 82$) groups using a median split and evaluating each age group separately. Older participants across both the intervention and control conditions exhibited significant cortisol reactivity to the task ($p = .001$); however, older LC participants showed exaggerated reactivity compared
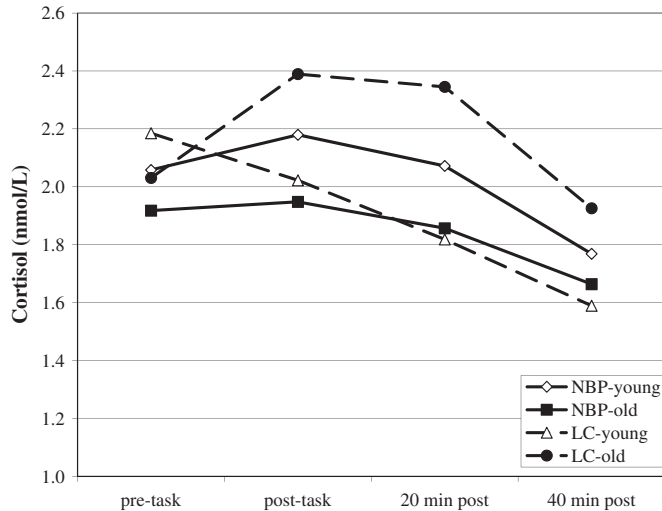
Figure 3. Intervention X age effects on cortisol reactivity; comparison of NBP to the literature control condition (LC).
Note: Age groupings represent median split. Covariates = time of day, sex and externalizing symptoms. Predicted log-transformed cortisol values were subjected to antilog-transformation in order to display cortisol values in an interpretable metric.

to older NBP participants, $\beta = .016$, 95% CI [.004, .028], $p = .010$. Among older participants, the NBP and LC groups did not differ on pre-task cortisol ($p = .71$), sex ($p = .33$), time of day ($p = .37$), externalizing symptoms ($p = .13$) or total mental health problems ($p = .13$), and these factors did not explain the group effect on cortisol reactivity.

When examined separately, younger LC participants did not show cortisol reactivity ($p = .99$), whereas younger NBP participants did exhibit reactivity ($p = .006$). When directly compared, the group difference in reactivity did not reach statistical significance, $\beta = .011$, 95% CI [−.022, .0009], $p = .072$. However, younger LC participants' cortisol response showed a steeper linear slope relative to younger NBP participants, $\beta = .045$, 95% CI [.007, .084], $p = .021$. Among younger participants, the NBP and LC groups did not differ on pre-task cortisol ($p = .91$), sex ($p = .93$), time of day of testing ($p = .94$), externalizing symptoms or total mental health symptoms (p's $> .36$), and these factors did not explain the group difference in slope.

Moderation of intervention effect by sex

A multilevel model predicting cortisol reactivity from group, sex and the group-by-sex interaction, controlling for time of day found that the group-by-sex interaction was not significant in the prediction of cortisol reactivity, $p = .69$, linear slope, $p = .44$ or intercept, $p = .86$. Inclusion of externalizing symptoms or total mental health symptoms in the models did not change the results.

Discussion

Our results suggest that an evidence-based intervention for recently divorced families may influence offspring cortisol stress responses 15 years later. Compared to youth in families who received the NBP, participants in the LC group showed diverging patterns of cortisol stress response depending on their age: older LC participants had elevated reactivity relative to NBP participants, whereas younger LC participants showed declining cortisol across the task relative to younger NBP participants. Higher externalizing symptoms predicted lower cortisol reactivity in both the NBP and LC groups, but did not explain the age effects on cortisol reactivity. The study is strengthened by the use of a randomised experimental design and an evidence-based intervention with documented mental health benefits. We found no evidence of pre-intervention group differences that could explain the current results. The use of a randomised design is critical because it minimises the possibility that third variables (e.g. genetic or other pre-intervention factors) accounted for intervention effects (Cicchetti & Gunnar, 2008).

The study is unique in finding evidence of both exaggerated and attenuated cortisol reactivity in the LC group, similar to the sensitisation and attenuation patterns of dysregulation following childhood adversity that have been noted in other studies (e.g. Miller et al., 2007), and suggest that age at the time of exposure to divorce-related stressors may have influenced long-term neuroendocrine response patterns in the control group. Relative to older participants in the NBP and younger LC participants, older LC participants had significantly elevated reactivity. Although pubertal status was not assessed, older participants were 11 or 12 years old during the program and likely transitioning into puberty. The peripubertal period is a sensitive period of development marked by increasing parent–child conflict and decreasing parent–child closeness (Steinberg & Morris, 2001), as well as heightened emotional and hormonal reactivity to stress (Gunnar, Wewerka, Frenn, Long, & Griggs, 2009; Shirtcliff et al., 2012; Sumter, Bokhorst, Miers, Van Pelt, & Westenberg, 2010). Hormonal activity during puberty also influences parent–child relationships and youth psychological health (Marceau, Dorn, & Sussman, 2012). This transitional period is particularly challenging for youth coping with parental divorce (Anderson, Hetherington, & Clingempeel, 1989). As a result, older youth may have been particularly susceptible to the effects of parental divorce and related stressors, and program-induced improvements in family factors or sensitivity to stress may have contributed to lower cortisol reactivity for NBP participants.

We also found evidence of attenuated reactivity among younger participants in the LC group. Younger participants in the intervention and control conditions did not differ in pre-task cortisol, thus the declining cortisol in the young LC participants is unlikely to be a result of higher anticipatory cortisol. Although speculative, participants who were younger during their exposure to the stressors in the early part of the divorce process may have initially exhibited high reactivity, but continuing stress across puberty may have promoted blunted reactivity, as suggested by theories of the long-term impact of chronic stress across development (e.g. Miller et al., 2007; Ruttle et al., 2011). Pathways by which parental divorce may affect HPA regulation include exposure to interparental conflict, parental mental health problems, offspring risky health behaviours, decreased father–child relationship quality or impairments in academic, social or occupational competence (Luecken & Lemery, 2004; Troxel & Matthews, 2004). The processes by which the NPB affected young adult HPA regulation are likely to be complex

and involve cascading effects of changes across time in environmental, family and individual-level processes. Future studies evaluating mechanistic pathways will be critical for testing theories of age-related effects of interventions on biological processes.

Interpretation of the health implications of different cortisol response patterns is complicated, as both higher and lower cortisol reactivity have been linked to psychological or physical health problems. In addition, interpretation of what constitutes a response that is too high or too low is necessarily context dependent. In general, an adaptive response rises to the extent needed to meet the unique demands of the situation and decreases upon cessation of the stressor. However, both high and low cortisol reactivity could confer advantage in a given situation. Evolutionary models theorise that early life conditions prepare a child's HPA axis to respond optimally to an idiosyncratic environment, and both exaggerated and blunted reactivity could have short-term survival advantages in adverse environmental conditions (Pluess & Belsky, 2013; Shirtcliff et al., 2014). Indeed, emerging research points to nonlinear relations between childhood adversity and HPA outcomes, in which characteristics of the child (e.g. age, temperament, mental health and personality), the caregiving environment (e.g. warmth and harsh discipline) and/or environment (e.g. severity and type of stressors, cumulative stressors) may influence the HPA stress response.

However, what is adaptive in childhood may be maladaptive later in development and in other environmental conditions (Shirtcliff et al., 2014). Both sensitisation and attenuation patterns of cortisol stress response have been linked to maladaptive psychosocial outcomes in children and young adults exposed to family adversity (Hagan, Roubinov, Mistler, et al., 2014; Koss et al., 2013), and poor long-term physical health outcomes (Heim et al., 2000). Higher reactivity may indicate an overly sensitive HPA axis or disruptions in the HPA axis negative feedback system, whereas attenuated responses may reflect a failure of the HPA to initiate a response or an overactive negative feedback system. In the current study, the higher reactivity of older LC participants could be considered an adaptive response in that moment, but might also reflect heightened sensitivity to stress relative to other participants. However, even if adaptive in a specific situation, consistent higher reactivity across time and in other contexts may confer future health risk. The association of concurrent mental health problems (externalizing symptoms and total problem index) with steeply declining cortisol responses suggests that the response pattern seen in younger LC participants likely indicates poor adaptation.

There are limitations to the analyses. We did not assess cortisol reactivity prior to assignment to condition, thus we cannot distinguish between whether the NBP prevented or alleviated adversity-induced alterations to cortisol. However, the randomised design of the trial is a 'gold standard for establishing causation because randomisation creates probabilistically equivalent treatment and control groups' (Mercer, DeVinney, Fine, Green, & Dougherty, 2007, p. 143), and we found no evidence of group differences in attrition or pre-intervention demographic or psychosocial factors that might suggest the intervention and control groups differed on cortisol at baseline. Because cortisol reactivity was not assessed at waves before the 15-year follow-up, we cannot examine cortisol reactivity patterns over time. It was necessary for the study that assessments be conducted in participants' homes, and the average cortisol reactivity was smaller than responses to the TSST commonly found in lab-based studies in front of 'audiences' (e.g. Dickerson & Kemeny, 2004). Further, a larger, more diverse sample would allow examination of additional moderators of intervention effect, such as

socio-conomic status or ethnicity. Finally, because the intervention was specifically for children from divorced families, all participants experienced parental divorce. It cannot be determined if results generalise to youth experiencing other forms of adversity.

Conclusions

Alterations in the neuroendocrine stress response system may represent a mechanism linking childhood parental divorce to adult mental and physical health outcomes. This study demonstrated age-related effects on cortisol reactivity of a preventive intervention for children who experienced parental divorce. Older youth in the control group displayed higher cortisol reactivity 15 years later relative to the intervention group, whereas younger participants in the control group showed a declining response to the task relative to the intervention group. Across the sample, higher externalizing symptoms and more mental health problems were associated with lower cortisol reactivity, but did not explain intervention effects. The findings contribute to a growing literature documenting varied patterns of cortisol response among those who experience childhood adversities and suggest that preventive interventions may alter the potential long-term neuroendocrine consequences.

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Notes

1. Preliminary analyses also confirm that the two intervention groups did not differ in cortisol response to the task ($p = .17$).
2. Primary analyses were repeated without excluding these 19 participants, as recommended by Cohen, Cohen, West and Aiken (2013) The results retained statistical significance. However, because the identified factors are known to alter cortisol levels (e.g. Jung et al., 2011; Walter et al., 2012), these cases were excluded from analyses.

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