The co-development of internalising symptoms, externalising symptoms and cognitive ability across childhood and adolescence

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Short title: Behaviour problems and cognitive ability trajectories

The co-development of internalising symptoms, externalising symptoms and cognitive ability across childhood and adolescence

Abstract

Cognitive ability, externalising symptoms and internalising symptoms are correlated in children. However, it is not known why they combine in the general child population over time. To address this, we used data on 17,318 children participating in the UK Millennium Cohort Study and followed-up five times between ages 3 and 14 years. We fitted three-parallel process latent growth curve models to identify the parallel unfolding of children's trajectories of internalising symptoms, externalising symptoms and cognitive ability across this period. We also examined the effects of time-invariant (ethnicity, birth weight, maternal education and age at birth, and breastfeeding status) and time-varying covariates (maternal psychological distress and socioeconomic disadvantage) on the growth parameters of the trajectories. The results showed that the intercepts of the trajectories of cognitive ability and, particularly, externalising symptoms were inversely correlated. Their linear slopes were also inversely correlated, suggesting parallel development. Internalising symptoms were correlated positively with externalising symptoms and inversely (and more modestly) with cognitive ability at baseline, but the slope of internalising symptoms correlated -positively- only with the slope of externalising symptoms. The covariates predicted 9% to 39% of the variance in the intercepts and slopes of all domains, suggesting they are important common risk factors. Overall, it appears that externalising symptoms develop in parallel with both cognitive ability and internalising symptoms from early childhood through to mid-adolescence. Children on an increasing trajectory of externalising symptoms are likely both increasing in internalising symptoms and decreasing in cognitive skills as well, and are thus an important group to target for intervention.

Keywords: Internalising symptoms, externalising symptoms, cognitive ability, trajectories, childhood, adolescence

Introduction

Children with low cognitive ability are disproportionally more likely to exhibit mental health difficulties such as internalising (emotional) and externalising (behavioural) symptoms, compared to those without (Cheng, Palta, Kotelchuck, Poehlmann, & Witt, 2014; de Ruiter, Dekker, Verhulst, & Koot, 2007; Emerson, 2003; Emerson & Hatton, 2007). The substantial body of developmental and educational psychology research to date on the links between mental health difficulties and cognitive ability in children is perhaps equally divided between observational studies examining mental health difficulties as 'predictors' of cognitive skills and those examining the latter as predictors of the former. Causal links are certainly plausible. For example, elevated levels of behavioural problems interfere with a child's normative development and consequently with the acquisition of age-appropriate cognitive skills (Campbell, 2002). Children with high levels of externalising problems are also more difficult to teach than their peers because they are not interested in learning, have trouble following directions, and often lack the self-control to cooperate (Rimm-Kaufman, Pianta, & Cox, 2000), and so have fewer opportunities to strengthen a broad range of cognitive abilities. Children with internalising difficulties may also have fewer opportunities to strengthen their cognitive abilities. Socially withdrawn and anxious children, for example, tend to take fewer risks which affects learning negatively. At the same time, there is much evidence in support for the opposite direction of the link between cognition and mental health in children. For example, there is strong evidence for the causal role of primarily frontally-mediated deficits in executive functions (e.g., attention, planning, working memory and response inhibition) in a range of externalising behaviours or disorders (Sergeant, Geurts, & Oosterlaan, 2002; Van der Meere, Marzocchi, & De Meo, 2005). There is also evidence for

the role of memory dysfunction and poor language skills in internalising problems (Price & Drevets, 2010; Toren et al., 2000).

Longitudinal evidence suggests dynamic associations too, especially between cognitive and behavioural difficulties (Glaser et al., 2011; Van der Ende, Verhulst, & Tiemeier, 2016). The notion that cognitive and mental health difficulties may be mutually reinforcing is further supported by evidence suggesting that neurocognitive deficits can be both risk factors (Koenen et al., 2009; Moffitt, 2003; Zammit et al., 2004) and outcomes of psychopathology (Wood et al., 2007). There is comparatively more longitudinal research into how internalising and externalising symptoms codevelop in children (Boylan, Georgiades, & Szatmari, 2010; Flouri et al., 2018; Gooren, van Lier, Stegge, Terwogt, & Koot, 2011; Herrenkohl et al., 2010; Morin et al., 2017; Rogosch, Oshri, & Cicchetti, 2010; van Lier & Koot, 2010; Wiesner, 2003). For example, many studies - usually situated in social developmental psychology - have shown positive longitudinal effects of externalising on internalising symptoms in childhood, in line with expectations from the failure theory whereby noxious behaviours and lack of social skills alienate peers which, in turn, increases vulnerability to internalising symptoms (Gooren et al., 2011). In general, most studies exploring the developmental cascades of internalising and externalising symptoms in childhood show positive unidirectional effects of externalising on internalising symptoms or positive reciprocal associations (Boylan et al., 2010; Flouri et al., 2019; Gooren et al., 2011; Moilanen, Shaw, & Maxwell, 2010; Morin et al., 2017; Van der Ende et al., 2016).

Although to our knowledge only two studies to date have examined how all three domains (internalising symptoms, externalising symptoms and cognitive ability) are inter-related over time in the general child population (Flouri et al., 2018; Flouri et al., 2019), many studies have examined longitudinal links between internalising and externalising symptoms and constructs related to cognitive ability such as academic competence (Weeks et

al., 2016) or, usually, academic performance. Most have shown a negative direct link from externalising problems to later academic performance but also a mixed picture of how academic performance is associated with externalising and internalising problems longitudinally (Englund & Siebenbruner, 2012; Masten et al., 2005; Moilanen et al., 2010; Riglin, Frederickson, Shelton, & Rice, 2013; Vaillancourt, Brittain, McDougall, & Duku, 2013; Van der Ende et al., 2016; van Lier et al., 2012; Verboom, Sijtsema, Verhulst, Penninx, & Ormel, 2014). In addition, by focussing on academic performance these studies have excluded the early years, when knowledge about causal processes, and therefore recommendations about interventions, may be particularly important. Finally, although related, cognitive ability and academic performance are distinct constructs (Johnson, McGue, & Iacono, 2006). Cognitive ability is one of the stronger predictors of academic performance, but the latter is also independently associated with other genetic and environmental factors, including executive functioning, self-regulation, socioeconomic and schooling characteristics and the home learning environment (Blair, McKinnon, & Family Life Project, 2016).

Of course, cognitive ability, internalising symptoms and externalising symptoms may be inter-linked in children simply because they share causes. Among the most powerful risk factors for all three are low socioeconomic status (Christensen, Schieve, Devine, & Drews-Botsch, 2014; Flouri & Midouhas, 2017; Hair, Hanson, Wolfe, & Pollak, 2015; Hanson et al., 2017), low parental education (Noble et al., 2015; Ormel et al., 2015), maternal depression (Bjornebekk et al., 2015; S. H. Goodman et al., 2011), low birth weight (Anderson, Doyle, & Victorian Infant Collaborative Study, 2003; Farajdokht et al., 2017) and being non-breastfed (Oddy et al., 2010; Park et al., 2014). The proposed mechanisms via which these risk factors can impact on cognitive ability and internalising or externalising symptoms in children are also similar. Socioeconomic status and parental education are thought to exert their impacts directly on brain development but also via their effects on parenting style, quality of parent-

child interactions, parent involvement in learning and parent stress (Flouri & Midouhas, 2017; Guo & Harris, 2000; Linver, Brooks-Gunn, & Kohen, 2002; Ormel et al., 2015; Tong, Baghurst, Vimpani, & McMichael, 2007). Breastfeeding is thought to enhance mother-infant interactions and can be considered an indicator of secure attachment status, an established predictor of a child's behavioural and emotional development (Crowell & Waters, 2005). The maternal contact occurring during breastfeeding has also been shown in animal models to have a beneficial effect on the development of neuroendocrine aspects of stress response (Liu et al., 1997), which can, in turn, affect later mental health and cognition (Guerry & Hastings, 2011; Juster, McEwen, & Lupien, 2010; Weinstock, 2005). Finally, low birth weight and maternal mental illness are risk factors of both poor cognitive skills (Bjornebekk et al., 2015; Farajdokht et al., 2017) and poor mental health (Chou, Wu, Chen, & Yang, 2016; Weinstock, 2005), via several routes including by their direct impacts on brain development and morphology. For example, a systematic review by Farajdoht et al. (2017), provides evidence for a delay in the cortical thinning among preterm children, possibly due to disturbance in the neuronal development in the third semester of pregnancy. Children born to mothers with mental health problems, on the other hand, have been shown to have smaller putamen volume on average relative to controls (Bjornebekk et al., 2015).

From a methodological point of view, the available studies to date on the associations over time between mental health difficulties and academic or cognitive skills in children have followed statistical analysis techniques - most commonly cross-lagged panel modelling (CLPM) when reciprocal associations are of special interest - which do not allow for the differentiation of intra-individual patterns of change over time (within-variation) from inter-individual (between-variation) differences in this co-development [for example, (Boylan et al., 2010; Gooren et al., 2011; Morin et al., 2017)]. Albeit informative, the developmental cascades described in these studies do not reflect "pure" longitudinal change because of the

conflation of within-child changes with between-child differences across the measures over time (Berry & Willoughby, 2017). Recent critiques of CLPM suggest the introduction of random intercepts as a way to segregate the between-person, "trait-like" aspects of a behaviour from the within-person deviations from one's own overall longitudinal trajectory (Hamaker, Kuiper, & Grasman, 2015). This approach was successfully implemented in a recent study of developmental cascades of cognitive ability and problem behaviour across childhood (Flouri et al., 2019) which found bidirectional associations between externalising symptoms and cognitive ability in males and between externalising and internalising symptoms in females. Yet, the interpretation of such effects is limited to claims about how one variable is associated with change in another variable measured at the subsequent assessment, without allowing for inferences about the overall longitudinal development across assessments. An additional limitation of CLPM is that the effects of the time-varying covariates are conditioned on the growth of the outcome variables, but not on their own overall growth, as this is not directly modelled (Muniz-Terrera et al., 2017). [Note: it is possible to model such effects in CLPM but computationally such models are extremely intensive and thus, most commonly, fail to converge]. Finally, models that include lagged effects can be sensitive to the time elapsed between measurement occasions.

Given the above, in this study we examined for the first time how cognitive, emotional and behavioural difficulties combine in the general child population over time using a statistical technique that allows for the separation of the between- from the within-person variation, i.e., parallel-process latent growth curve modelling. Additionally, we attempted to quantify more adequately the impact of key time-varying and time-invariant covariates on this parallel unfolding. Based on findings from studies employing CLPM to study developmental cascades of internalising symptoms, externalising symptoms and cognitive ability (Flouri et al., 2019), we hypothesised that externalising symptoms at

baseline will be cross-sectionally correlated with internalising symptoms (positively) and cognitive ability (negatively). We also expected an inverse correlation, albeit of smaller size, between internalising symptoms and cognitive ability at baseline. We also hypothesised that the slope of externalising symptoms would be the strongest predictor of the slope of both internalising symptoms (positively) and cognitive ability (negatively). Moreover, we theorised that the time-invariant covariates would have a significant impact on the growth parameters of all three outcome measures. Finally, in the absence of evidence about the associations, modelled this way, between our time-varying covariates and outcomes, we expected that the estimates of the covariance between the growth parameters reflecting intra-individual change would become weaker after adjustments, albeit they would remain significant.

Methods

Sample

The data for this study came from the first six sweeps of the Millennium Cohort Study (MCS), an ongoing population-based cohort study following children born in the UK in 2000 or shortly thereafter. The children were on average 9 months old at Sweep 1, and 3, 5, 7, 11 and 14 years old at Sweeps 2, 3, 4, 5 and 6, respectively. At the six sweeps, the number of participating families was 18,522, 15,590, 15,246, 13,857, 13,287 and 11,714, respectively. Our analytic sample included children (singletons and first-born twins or triplets) with valid data on externalising symptoms, internalising symptoms and cognitive ability in at least one of Sweeps 2 to 6 (N=17,318; 51% male), when MCS had data on all three outcomes. Ethical approval was gained from NHS Multi-Centre Ethics Committees, and parents and children gave informed consent before interviews took place.

Measures

Cognitive ability at ages 3, 5, 7, 11 and 14 years

Cognitive ability was calculated for each age by using the age-adjusted ability assessments that were available in MCS. At age 3, there were two cognitive assessments, the Bracken School Readiness Assessment-Revised, measuring children's 'readiness' for formal education by testing their knowledge and understanding of basic concepts (Bracken, 1998), and the second edition of the British Ability Scales (BASII)(C. D. Elliott, Smith, & McCulloch, 1996) for Naming Vocabulary, which measures expressive language. At age 5, ability was assessed with three scales, BAS Naming Vocabulary, BAS Pattern Construction (measuring spatial problem-solving) and BAS Picture Similarities (measuring non-verbal reasoning). At age 7, it was measured with BAS Pattern Construction, BAS Word Reading (measuring educational knowledge of reading) and the National Foundation for Educational Research Progress in Maths. At age 11, it was measured with BAS Verbal Similarities, which assesses verbal reasoning and verbal knowledge. Finally, at age 14 it was measured with a word activity task assessing the understanding of the meaning of words. This task, used in other general population studies in the UK (e.g., at the age 16 sweep of the 1970 British Birth Cohort Study), is based on standardised vocabulary tests devised by the Applied Psychology Unit at the University of Edinburgh in 1976 (J. Elliott & Shepherd, 2006).

When multiple cognitive assessments were available (i.e., at ages 3, 5 and 7), we measured cognitive ability by using the scores derived from a principal components analysis of the various assessment scores. Each component score was then transformed into a standardised score with a mean of 100 and a standard deviation of 15 (Hanscombe et al., 2012). These multiple well-validated assessments are thought to be able to capture a general cognitive ability factor, which is not dependent on the use of specific mental ability tasks (Johnson, Bouchard Jr, Krueger, McGue, & Gottesman, 2004). (For ages 11 and 14, when

only one measure of ability was available in MCS, we transformed the age-adjusted ability score into a standardised cognitive ability score.)

Internalising and externalising symptoms at ages 3, 5, 7, 11 and 14 years

Internalising and externalising symptoms were measured with the parent-reported Strengths and Difficulties Questionnaire (SDQ), a short behavioural screening tool for children aged 2 to 17 years old (R. Goodman, 1997). The 20 difficulties and symptoms assessed by the SDQ are equally divided in four subscales, emotional symptoms, conduct problems, hyperactivity/inattention and peer problems. In line with recommended practice for community samples (A. Goodman, Lamping, & Ploubidis, 2010), the internalising problems scale comprised the 10 items from the emotional and peer problems subscales, and the externalising problems scale the 10 items from the hyperactivity and conduct problems subscales. Scores on each of these two scales range from 0 to 20, with higher scores indicating more problems or symptoms. In the analytic sample the Cronbach's alphas for the internalising and externalising problem scales ranged from 0.61 (age 3) to 0.77 (ages 11 and 14) and from 0.78 (ages 3 and 5) to 0.81 (ages 11 and 14), respectively, suggesting adequate reliability. In this study we considered internalising and externalising symptom scores as continuous variables, yet we also used the widely-used banding by cut-off score proposed by Goodman (1997). According to this, children's scores are 'borderline' if they lie in the upper 80-90% of the distribution (internalising symptom scores >= 7; externalising symptom scores >= 9) and 'abnormal' if they are in the upper decile (internalising symptom scores >= 9; externalising symptom scores >= 11).

Covariates

We evaluated the effect of several time-varying and time-invariant factors which are known to be associated with both cognitive ability and externalising and internalising

symptoms. The time-invariant covariates were *birth weight* (dummy coded as <2.5 kg or not), *breastfeeding status* (dummy coded as yes or no), *ethnicity* (one dummy variable for each of the following, UK Census classified, ethnic groups: white, Indian, Pakistani/Bangladeshi, black, mixed, and other), *maternal education* (dummy coded as having obtained a university degree or not) and *maternal age at birth*. The time-varying (at ages 3, 5, 7, 11 and 14) covariates were *maternal psychological distress* [measured using the Kessler K6 (Kessler et al., 2002)] and s*ocioeconomic disadvantage.* This was measured using a 4-item summative index comprising overcrowding (>1.5 people per room excluding bathroom and kitchen), lack of home ownership, receipt of income support, and income poverty (equivalised net family income below 60% of the national median household income) (Malmberg & Flouri, 2011).

Statistical analysis

All models were stratified by sex to account for differences in the childhood developmental trajectories of the three main measures between males and females (Carter et al., 2010; Douma, Dekker, de Ruiter, Tick, & Koot, 2007; Leve, Kim, & Pears, 2005; Richer, Lachance, & Côté, 2016). Our analytic approach was as follows. First, we examined sex differences in the main measures and the covariates at baseline (age 3 years). Next, we used parallel-process latent growth curve modelling (LGCM) to describe the parallel unfolding of cognitive ability and mental health from ages 3 to 14 years. The basic LGCM estimates intra-individual patterns of change over time (within-variation) but also inter-individual heterogeneity in growth parameters (between-variation), by yielding standard errors and significance levels for the variance estimates of the intercept and slope. The parallel-process LGCM, however, can also estimate the covariance between the growth parameters of the different outcomes, which provides information on their parallel development (Bollen & Curran, 2006; Curran, Obeidat, & Losardo, 2010; Wickrama, Lee, O'Neal, & Lorenz, 2016).

We ran three parallel-process LGCMs. A baseline model estimated model fit to the data prior to including any covariates. The second model adjusted for the time-invariant covariates. For each of the ethnic groups considered we created a dummy-coded variable, as explained above, and we did not include in the models the variable for white ethnic group which, thus, served as the reference category. The third model made further adjustments for the time-varying covariates of maternal psychological distress and socioeconomic disadvantage. We parametrised the latter two models in such a way that we allowed time-invariant covariates to predict the growth parameters directly. For the time-varying covariates, we modelled their growth trajectories and estimated the regression paths between their growth parameters and those for the three outcomes (internalising symptoms, externalising symptoms and cognitive ability). Using this model specification, the correlations between the intercepts and slopes of the three trajectories represented intra-individual developmental changes. The residual variance of the intercepts and slopes of the trajectories as well as the residual correlations between the growth parameters reflected inter-individual differences.

All analyses were performed in Stata/SE 14.2 (StataCorp, 2011) and Mplus 7.4 (Muthén & Muthén, 2009). LGCMs were run using the maximum likelihood with robust standard errors (MLR) estimator which provides maximum likelihood parameter estimates with robust standard errors and takes into account the skewed distributions of variables. Model fit was assessed using three indices: the comparative fit index (CFI), the root mean square error of approximation (RMSEA) and the standardised root mean square residual (SRMR). CFI values >=0.95, RMSEA values <0.06 and SRMR values <0.05 are indicative of good model fit (Hooper, Coughlan, & Mullen, 2008). All missing data were handled using full information maximum likelihood which estimates parameters using any available information that is contained in the analytic model. The MCS stratum was controlled to

account for the disproportionate stratification of the MCS survey design (Plewis, Calderwood, Hawkes, Hughes, & Joshi, 2007). Attrition and non-response were taken into account by using weights.

Results

Table 1 summarises the characteristics of the sample stratified by sex. At baseline, males scored significantly higher in internalising and externalising symptoms compared to females, had lower cognitive ability and were less likely to have been of low birth weight, but did not differ with respect to the remaining characteristics considered.

"TABLE ONE HERE"

Model fit was relatively poor for the baseline LGCM (CFI=0.88; RMSEA=0.05; SRMR=0.08) but improved for the model including the time-invariant covariates (CFI=0.89; RMSEA=0.04; SRMR=0.05) and especially for the one including both the time-invariant and the time-varying covariates (CFI=0.91; RMSEA=0.03; SRMR=0.04). Using Wald tests we tested whether the growth parameters (intercepts and slopes) of internalising symptoms, externalising symptoms and cognitive ability differed between sexes in the fully adjusted model. The results showed that there were no sex differences in the growth parameters of the internalising and externalising symptom trajectories. However, the intercept of the cognitive ability trajectory was significantly lower in males [intercept=96.30(SE=0.33)] compared to females [intercept=100.17(SE=0.36); $\chi^2(1)=7.92$, p=0.005], while the slope was positive in males [slope=0.47(SE=0.07)] and negative in females [slope=-0.42(SE=0.07); $\chi^2(1)=12.07$, p<0.001].

As can be seen in Table 2, the positive correlations between the intercepts of internalising and externalising symptoms for both males and females in the fully adjusted model (rs around .60 for both sexes) suggest that children with more internalising symptoms

are also more likely to present with externalising symptoms at baseline, and vice versa. In addition, the positive correlations between their slope estimates (rs around .75 for both sexes) suggest that the two symptom types develop in parallel. Higher levels of symptoms at baseline were additionally associated with lower cognitive ability at baseline although the correlations were lower (rs. around .40 for both sexes and across both symptom types). Nonetheless, only the slope of externalising symptoms, but not that of internalising symptoms, was associated with the slope of cognitive ability (rs around .30 and .20 for males and females, respectively).

"TABLE TWO HERE"

Table 3 summarises the standardised residual variance estimates of the growth parameters, the correlations between the three intercepts and the correlations between the three slopes, before and after adjustments for covariates (Models A-C). The significant variance estimates of the intercepts and slopes as well as the significant correlations in the fully-adjusted model, with the exception of the correlation between the slopes of the trajectories of internalising symptoms and cognitive ability, suggest that inter-individual differences in the growth parameters as well as in the co-development of the outcomes persist even after adjustments for covariates. Nevertheless, a significant amount of variance in the growth parameters was explained by the covariates. This ranged, in males, from 9% for the slope of the externalising symptom trajectory to 41% for the slope of the cognitive ability trajectory. The estimates in females were 21% and 39%, respectively. Noticeably, the time-invariant covariates - ethnicity, breastfeeding status, birth weight, maternal education and maternal age at birth - explained much of the between-child variance in the growth parameters of cognitive ability (females: 26% of the variance in the intercept and 35% of the variance in slope; males: 24% and 29%, respectively). By contrast, the proportion of variance explained in the intercepts and slopes of the internalising and externalising symptom

trajectories increased substantially after adjustments for the time-varying covariates, i.e. socioeconomic disadvantage and maternal psychological distress. For example, the between-child variance in the intercept of internalising changed from 11% (in the model adjusting for time-invariant covariates only) to 37% (in the model adjusting for both time-invariant and time-varying covariates) in males, and from 12% to 35% in females. The between-child variance in the slope of internalising changed from 2% to 20% in males, and from 4% to 23% in females. For externalising, the between-child variance in the intercept changed from 10% to 28% in males and from 13% to 27% in females; the between-child variance in the slope changed from 1% to 9% and from 2% to 21% in males and females, respectively.

"TABLE THREE HERE"

Table 4 shows the unstandardised regression coefficients of the covariates on the growth parameters of the trajectories stratified by sex. Ethnic minority children had lower cognitive ability at baseline than their white counterparts but their cognitive skills changed at a faster pace throughout childhood and adolescence. Additionally, children of mothers with a university degree, the breastfed and those of normal birth weight had fewer internalising and externalising symptoms and higher cognitive ability. Breastfeeding status was also related to the rate of change in internalising and externalising symptoms in females, while maternal age at birth only in internalising symptoms in females. With respect to the time-varying covariates, their effects on the growth parameters of the outcomes were comparable between sexes. At baseline, higher levels of maternal psychological distress and socioeconomic disadvantage were correlated with higher levels of internalising and externalising scores and with lower levels of cognitive ability. The slope of maternal psychological distress was significantly predictive of the slopes of internalising and externalising symptoms, but not cognitive ability, suggesting that increases in maternal psychological distress during childhood and adolescence are associated with increases in externalising and internalising

symptoms in children. By contrast, the slope of socioeconomic disadvantage was significantly predictive of the slope of cognitive ability - and internalising symptoms in females only - but not externalising symptoms, suggesting that increases in the level of socioeconomic disadvantage are associated with decreases in cognitive ability in both sexes and with increases in internalising symptoms in females.

"TABLE FOUR HERE"

To illustrate the cumulative effect of the covariates that are arguably modifiable risk factors, we plotted the predicted values of the trajectories of the three domain scores after restricting covariate values to their mean, their minimum and their maximum. We considered modifiable risk factors to be maternal education, birth weight, breastfeeding status, maternal psychological distress and socioeconomic disadvantage. Plotted this way, the trajectories for which covariates were held at their extreme high and low values illustrate, respectively, the effects of absence and accumulation of risk. Figures 1 and 2 illustrate these trajectories for males and females, respectively. Children scoring high on all risk factors (termed high-risk in the figure) show high and increasing levels of internalising and externalising symptoms as well as low and decreasing cognitive ability. By contrast, children scoring low on all risk factors (termed low-risk in the figure) are characterised by a near absence of symptoms and by cognitive ability scores almost 1 standard deviation above the population mean at baseline, which in fact increased even further during the study period.

"FIGURE ONE HERE"

"FIGURE TWO HERE"

Bias analysis

We performed a bias analysis to test whether the three domain scores follow non-linear trajectories by fitting a fully adjusted (as in Model C) LGCM including a quadratic

term for age. Visual inspection of the data showed that the linear and non-linear LGCMs extracted almost identical trajectories for the three outcomes (Figures 3 and 4). In addition, the non-linear LGCM ran into convergence problems, including the identification of a linear relationship between the slope estimates of internalising and externalising symptoms, suggesting model inadmissibility and therefore preference for the better-fitting and more parsimonious linear LGCM.

"FIGURE THREE HERE"

"FIGURE FOUR HERE"

Discussion

This study adds to the evidence that internalising symptoms, externalising symptoms and cognitive ability are inter-related in the general child population. However, it also provides support for important specificity in these associations. We showed that although all inter-domain associations at baseline were significant, those with externalising symptoms were clearly stronger. In addition, increasing levels of externalising symptoms throughout childhood and until mid-adolescence were associated both with increases in internalising symptoms and with declines in cognitive ability. Together, these findings suggest that children on an increasing trajectory of externalising symptoms are likely both increasing in internalising symptoms and decreasing in cognitive skills as well, and are thus an important group to target for intervention. Of course, since the relationships we identified are associative and not causative, it is not clear if declines in cognitive skills and/or increases in internalising symptoms cause increases in externalising symptoms, if increases in externalising symptoms cause declines in cognitive skills (Glaser et al., 2011; Van der Ende

et al., 2016) and increases in internalising symptoms, or if third variables are responsible for externalising symptoms changing in parallel with both internalising and cognitive difficulties.

Another important finding, in particular from a public health perspective, is that factors easily identified in the early years, such as birthweight, maternal education, maternal age and breastfeeding status, explain much of the between-child variation in the trajectory of cognitive ability from the preschool period to mid-adolescence. As discussed in the introduction, low birth weight and not being breastfed are independently associated with the child's neurological development and might explain their strong effects on cognitive ability (Farajdokht et al., 2017; Juster et al., 2010; Liu et al., 1997). A mother's educational attainment, on the other hand, is a proxy of her cognitive ability and thus also a proxy of genetic influences on a child's ability in biological mother-child pairs, the vast majority of mother-child pairs in MCS. At the same time, maternal education is typically a very reliable indicator of parental human capital. Greater levels of parental human capital are, in turn, linked to more favourable cognitive outcomes for children. For example, more educated parents are able not only to afford higher-quality education but also to invest more time and effort in basic care and play (Kalil, Ryan, & Corey, 2012), thus creating and fostering more cognitively stimulating environments for their children. By contrast, between-child differences in externalising and internalising symptom trajectories were mostly explained by the two time-varying covariates we considered, maternal psychological distress and socioeconomic disadvantage. Regarding maternal psychological distress, we would argue that, due to the genetic basis of psychopathology, a large part of its effect on child emotional and behavioural symptoms in our study captures genetic influences. Nonetheless there are also several other, environmental and neurobiological, pathways through which maternal depression is associated with child psychopathology (S. H. Goodman et al., 2011) and which, as will be discussed later in detail, we could not test. Socioeconomic disadvantage, on the

other hand, likely impacts on children's internalising and externalising symptoms via its effects on parental mental health but also more proximally via the environmental influences of poor parenting and psychosocial stressors (Roubinov & Boyce, 2017). Interestingly, there was some evidence for specificity in the effects of the time-varying covariates on slopes. We found that increasing levels of maternal psychological distress during childhood and adolescence were associated with increasing levels of both internalising and externalising symptoms, but not with decreased cognitive ability. Increases in socioeconomic disadvantage decreased cognitive skills, but did not change externalising symptoms. This risk specificity notwithstanding, our study established the importance of risk accumulation for all three domains. As we showed, the trajectories of children exposed to all the risk factors we considered diverged significantly from those of the children exposed to some or no risk factors. In fact, the predicted scores were above the 'abnormal' cut-off for SDQ scores, thus suggesting that such children would represent an ultra high-risk group for later adverse outcomes, including poor decision-making skills, low self- esteem, engagement in antisocial behaviours, and psychiatric morbidity (Flouri et al., 2018; Lancefield, Raudino, Downs, & Laurens, 2016).

Nonetheless, even after accounting for the effects of the covariates, the initial score (intercept) as well as the rate of change (slope) of trajectories differed significantly between children. This unexplained variation is likely due to other covariates, not controlled here. Likely candidate variables include peer relationships (particularly important in adolecsence) and schooling characteristics, such as school quality (Sisco et al., 2015) or academic perfomance (Metsäpelto et al., 2015; Moilanen et al., 2010), which we did not consider given that we started following our sample at preschool age. Likely candidate variable of course include also family and parenting (Flouri & Midouhas, 2017; Guo & Harris, 2000; Linver et al., 2002; Ormel et al., 2015; Tong et al., 2007) characteristics which we could not consider

in as much detail as we would wish, as will be discussed below. Genetic factors are also likely candidate variables as explained (Hagenaars et al., 2016; Hill, Davies, Liewald, McIntosh, & Deary, 2016; Martin, Hamshere, Stergiakouli, O'donovan, & Thapar, 2015; McGrath et al., 2016). Anatomical factors too could be important (Kanai & Rees, 2011; Whittle, Vijayakumar, Simmons, & Allen, 2019). A review of MRI studies of the brain emphasised their potential for formulating a neural basis of human behaviour and cognition (Kanai & Rees, 2011). As a case in point, inter-individual variability in intelligence has been demonstrated to correlate with cortical thickness and white matter integrity (Kanai & Rees, 2011), also associated with internalising and externalising problems: increasing levels of internalising symptoms in childhood are associated with reduced thinning in the orbitofrontal cortex, whereas increases in levels of externalising symptoms are associated with reduced thinning in the postcentral gyrus (Whittle et al., 2019).

Our study has limitations, too. First, we could not determine the direction of the associations we established. Second, the fully-adjusted LGCM was a computationally intensive model and in order to achieve convergence we had to omit inclusion of several family characteristics, such as parental involvement, discipline and warmth, all of which could have been covariates. Third, SDQ-based internalising and externalising symptoms were parent (overwhelmingly mother) reported, with no triangulation from other reporters such as teacher or child. This may be particularly problematic for internalising symptoms in view of the evidence for higher levels of agreement between parent and self reports on the SDQ for externalising than for internalising disorders (Van der Meer, Dixon, & Rose, 2008). An additional source of bias in our study could be introduced by using information stemming from different sources since cognitive ability was based on observational tests while internalising and externalising symptoms were both measured using parental reports. Finally, cognitive ability was not measured with the same tasks at each assessment, hence we could

not test for measurement invariance across time-points. Nonetheless, we used a validated approach to derive a general cognitive ability score at each assessment (Johnson et al., 2004), which has already been successfully implemented in previous studies (Basatemur et al., 2013; Flouri, Midouhas, & Joshi, 2015). For the same reason, we could not run the growth curve models on the raw cognitive ability scores, which is the recommended practice. However, we believe that to the extent that the latent growth parameters capture within-person variation, the estimated values obey the rank order of raw scores across assessment points and, hence, the estimates of the cognitive ability trajectories presented are reliable.

Conclusion

This study explores for the first time the joint development of internalising symptoms, externalising symptoms and cognitive ability across childhood and adolescence in the general population. The results suggest that, within children, cognitive ability and, particularly, externalising symptoms are inversely correlated in early childhood and develop in parallel over time. Internalising symptoms in childhood are also associated with both externalising symptoms and cognitive ability, but the growth trajectory of internalising symptoms unfolds in parallel only to the trajectory of externalising symptoms. Overall, our findings suggest that children on an increasing trajectory of externalising symptoms are an important group to target for intervention, as they are also likely to both increase in internalising symptoms and decrease in cognitive skills. Investigating the causes of the co-development of externalising symptoms with both cognitive and emotional difficulties in children should be an important priority for future research.

Another important finding is that a significant amount of the between-child variation in the growth trajectories of the three domains is accounted for by modifiable risk factors, such as low maternal education, socioeconomic disadvantage, not being breastfed, low

birthweight and maternal psychological distress. In general, the risk factors we examined explained more variance in the intercepts, rather than the slopes, of internalising and externalising symptom trajectories. This suggests that interventions targeting these time-varying and time-invariant risk factors have the potential to reduce the initial level of mental health symptomatology in childhood and, thus, alleviate some of the burden associated with it. Similar to the above, the effects of these risk factors appeared to be lowering mainly the residual correlations between the intercepts of the three domains, while their effects on the residual correlations between the slopes were less prominent. Accordingly, early prevention strategies or interventions to reduce these risk factors might not only reduce the initial level of children's mental health symptomatology directly, but could also disrupt the mutually reinforcing relationships between mental health and cognitive ability early in life. The potential of such interventions becomes clear with the quantification of risk that we attempted: setting the risk factors at their minimum and maximum values predicted internalising, externalising and cognitive ability scores more than 1.5 standard deviations away from the average estimated trajectories with risk factors set at their default values. Hence, it appears that the potential of such prevention and intervention programmes is immense.

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Table 1. Unweighted estimates of problem behaviour, cognitive ability and covariates across sexes			
	Males (N=8,860; 51%)	**Females (N=8,458; 49%)**	**p-value**
Internalising symptoms (M±SE)			
Age 3	3.03 ± 0.03	2.82 ± 0.03	<0.001
Age 5	2.63 ± 0.03	2.51 ± 0.03	0.005
Age 7	2.85 ± 0.04	2.69 ± 0.03	0.001
Age 11	3.26 ± 0.04	3.22 ± 0.04	0.47
Age 14	3.59 ± 0.05	3.99 ± 0.05	<0.001
Externalising symptoms (M±SE)			
Age 3	7.16 ± 0.05	6.32 ± 0.04	<0.001
Age 5	5.33 ± 0.04	4.33 ± 0.04	<0.001
Age 7	5.37 ± 0.05	4.15 ± 0.04	<0.001
Age 11	5.11 ± 0.05	3.91 ± 0.04	<0.001
Age 14	4.93 ± 0.05	3.88 ± 0.04	<0.001
Cognitive ability (M±SE)			
Age 3	98.05 ± 0.18	101.94 ± 0.18	<0.001
Age 5	98.82 ± 0.18	101.23 ± 0.17	<0.001
Age 7	99.47 ± 0.19	100.54 ± 0.18	<0.001
Age 11	100.52 ± 0.19	99.47 ± 0.18	<0.001
Age 14	99.78 ± 0.21	100.22 ± 0.20	0.13
Ethnicity			
White	7,288 (82%)	6,953 (82%)	
Mixed	262 (3%)	263 (3%)	
Indian	233 (3%)	212 (3%)	
Pakistani / Bangladeshi	599 (7%)	603 (7%)	0.76
Black	343 (4%)	301 (4%)	
Other	131 (1%)	122 (1%)	
Mother is university-educated	1,358 (16%)	1,358 (17%)	0.22
Low birth weight (<2.5 kg)	547 (6%)	633 (8%)	0.001
Maternal age at birth (M±SE)	28.40 ± 0.06	28.49 ± 0.07	0.34
Not breastfed	2,660 (31%)	2,635 (32%)	0.14
Socioeconomic disadvantage (M±SE)			
Age 3	0.88 ± 0.01	0.90 ± 0.01	0.36
Age 5	0.90 ± 0.01	0.93 ± 0.01	0.19
Age 7	0.82 ± 0.01	0.85 ± 0.01	0.12
Age 11	0.78 ± 0.01	0.77 ± 0.01	0.95
Maternal psychological distress (M±SE)			
Age 3	3.33 ± 0.05	3.22 ± 0.05	0.10
Age 5	3.24 ± 0.05	3.16 ± 0.05	0.19
Age 7	3.22 ± 0.05	3.07 ± 0.05	0.03
Age 11	4.08 ± 0.06	4.07 ± 0.06	0.96
Age 14	4.30 ± 0.06	4.37 ± 0.06	0.40
Values presented as N(%) unless otherwise specified.			

Table 2. Mean growth parameter estimates (intra-individual development) of parallel-process latent growth curves of internalising symptoms, externalising symptoms and cognitive ability adjusted for time-invariant and time-varying covariates

Males			
		Estimate (SE)	**p-value**
Intercept	Internalising symptoms	2.96 (0.04)	<0.001
	Externalising symptoms	6.57 (0.07)	<0.001
	Cognitive ability	96.30 (0.33)	<0.001
Slope	Internalising symptoms	0.14 (0.01)	<0.001
	Externalising symptoms	-0.25 (0.02)	<0.001
	Cognitive ability	0.47 (0.07)	<0.001
Intercept-slope correlations	I_{int}-I_{ext}	0.65 (0.02)	<0.001
	I_{int}-I_{CA}	-0.40 (0.02)	<0.001
	I_{ext}-I_{CA}	-0.44 (0.02)	<0.001
	S_{int}-S_{ext}	0.76 (0.03)	<0.001
	S_{int}-S_{CA}	-0.06 (0.06)	0.35
	S_{ext}-S_{CA}	-0.32 (0.06)	<0.001
Females			
		Estimate (SE)	**p-value**
Intercept	Internalising symptoms	2.70 (0.05)	<0.001
	Externalising symptoms	5.44 (0.07)	<0.001
	Cognitive ability	100.17 (0.36)	<0.001
Slope	Internalising symptoms	0.21 (0.01)	<0.001
	Externalising symptoms	-0.28 (0.01)	<0.001
	Cognitive ability	-0.42 (0.07)	<0.001
Intercept-slope correlations	I_{int}-I_{ext}	0.61 (0.02)	<0.001
	I_{int}-I_{CA}	-0.37 (0.02)	<0.001
	I_{ext}-I_{CA}	-0.42 (0.02)	<0.001
	S_{int}-S_{ext}	0.78 (0.05)	<0.001
	S_{int}-S_{CA}	-0.07 (0.04)	0.14
	S_{ext}-S_{CA}	-0.23 (0.06)	<0.001

Table 3. Crude and adjusted residual variance estimates (inter-individual differences) of parallel-process latent growth curves of internalising symptoms, externalising symptoms and cognitive ability

Males

		Model A[a]		Model B[b]		Model C[c]	
		Estimate (SE)	**p-value**	**Estimate (SE)**	**p-value**	**Estimate (SE)**	**p-value**
Standardised residual variance of intercept	Internalising symptoms	1.00	NA	0.89 (0.01)	<0.001	0.63 (0.02)	<0.001
	Externalising symptoms	1.00	NA	0.90 (0.01)	<0.001	0.72 (0.02)	<0.001
	Cognitive ability	1.00	NA	0.76 (0.02)	<0.001	0.69 (0.02)	<0.001
Standardised residual variance of slope	Internalising symptoms	1.00	NA	0.98 (0.01)	<0.001	0.80 (0.04)	<0.001
	Externalising symptoms	1.00	NA	0.99 (0.01)	<0.001	0.91 (0.03)	<0.001
	Cognitive ability	1.00	NA	0.71 (0.06)	<0.001	0.59 (0.08)	<0.001
Standardised intercept-slope residual correlations	I_{int}-I_{ext}	0.66 (0.02)	<0.001	0.64 (0.02)	<0.001	0.52 (0.03)	<0.001
	I_{int}-I_{CA}	-0.41 (0.02)	<0.001	-0.31 (0.02)	<0.001	-0.24 (0.02)	<0.001
	I_{ext}-I_{CA}	-0.45 (0.02)	<0.001	-0.39 (0.02)	<0.001	-0.34 (0.02)	<0.001
	S_{int}-S_{ext}	0.75 (0.03)	<0.001	0.75 (0.03)	<0.001	0.74 (0.04)	<0.001
	S_{int}-S_{CA}	-0.04 (0.06)	0.56	0.01 (0.07)	0.88	0.06 (0.08)	0.45
	S_{ext}-S_{CA}	-0.31 (0.06)	<0.001	-0.32 (0.07)	<0.001	-0.34 (0.08)	<0.001
R-square	I_{int}	---	---	0.11 (0.01)	<0.001	0.37 (0.02)	<0.001
	S_{int}	---	---	0.02 (0.01)	0.003	0.20 (0.04)	<0.001
	I_{ext}	---	---	0.10 (0.01)	<0.001	0.28 (0.02)	<0.001
	S_{ext}	---	---	0.01 (0.01)	0.04	0.09 (0.03)	0.002
	I_{CA}	---	---	0.24 (0.02)	<0.001	0.32 (0.02)	<0.001
	S_{CA}	---	---	0.29 (0.06)	<0.001	0.41 (0.08)	<0.001

Females

		Model A[a]		Model B[b]		Model C[c]	
		Coefficient (SE)	**p-value**	**Coefficient (SE)**	**p-value**	**Coefficient (SE)**	**p-value**
Standardised residual variance of intercept	Internalising symptoms	1.00	NA	0.88 (0.02)	<0.001	0.65 (0.02)	<0.001
	Externalising symptoms	1.00	NA	0.96 (0.01)	<0.001	0.73 (0.02)	<0.001
	Cognitive ability	1.00	NA	0.87 (0.01)	<0.001	0.65 (0.02)	<0.001
Standardised residual variance of slope	Internalising symptoms	1.00	NA	0.98 (0.01)	<0.001	0.77 (0.04)	<0.001
	Externalising symptoms	1.00	NA	0.74 (0.02)	<0.001	0.79 (0.04)	<0.001
	Cognitive ability	1.00	NA	0.65 (0.05)	<0.001	0.61 (0.06)	<0.001
Standardised intercept-slope residual correlations	I_{int}-I_{ext}	0.63 (0.02)	<0.001	0.60 (0.02)	<0.001	0.49 (0.03)	<0.001
	I_{int}-I_{CA}	-0.38 (0.02)	<0.001	-0.28 (0.02)	<0.001	-0.19 (0.03)	<0.001
	I_{ext}-I_{CA}	-0.44 (0.02)	<0.001	-0.38 (0.02)	<0.001	-0.30 (0.02)	<0.001
	S_{int}-S_{ext}	0.78 (0.05)	<0.001	0.79 (0.05)	<0.001	0.74 (0.06)	<0.001
	S_{int}-S_{CA}	-0.05 (0.05)	0.34	0.05 (0.06)	0.33	0.05 (0.06)	0.42
	S_{ext}-S_{CA}	-0.25 (0.06)	<0.001	-0.23 (0.07)	0.01	-0.25 (0.08)	0.001
R-square	I_{int}	---	---	0.12 (0.02)	<0.001	0.35 (0.02)	<0.001
	S_{int}	---	---	0.04 (0.01)	<0.001	0.23 (0.04)	<0.001
	I_{ext}	---	---	0.13 (0.01)	<0.001	0.27 (0.02)	<0.001
	S_{ext}	---	---	0.02 (0.01)	0.03	0.21 (0.04)	<0.001
	I_{CA}	---	---	0.26 (0.02)	<0.001	0.35 (0.02)	<0.001
	S_{CA}	---	---	0.35 (0.05)	<0.001	0.39 (0.06)	<0.001

[a]Crude Model.

[b]Model adjusted for the time-invariant covariates of ethnicity, maternal education, birth weight, maternal age at birth and breastfeeding status.

[c]Model adjusted for the time-invariant covariates as in Model B, and also the time-varying covariates of socioeconomic disadvantage and maternal psychological distress.

Table 4. Unstandardised regression coefficients (SE) of covariates on the growth parameters of the fully-adjusted parallel-process latent growth curve model of internalising symptoms, externalising symptoms and cognitive ability

	Intercept, Internalising symptoms		**Slope, Internalising symptoms**		**Intercept, Externalising symptoms**		**Slope, Externalising symptoms**		**Intercept, Cognitive ability**		**Slope, Cognitive ability**	
	Coefficient (SE)	p-value	Coefficient (SE)	p-value	Coefficient (SE)	p-value	Coefficient (SE)	p-value	Coefficient (SE)	p-value	Coefficient (SE)	p-value
Males												
Ethnicity												
White	Ref	NA	Ref	NA	Ref	NA	Ref	NA	Ref	NA	Ref	NA
Mixed	-0.09 (0.21)	0.67	-0.02 (0.06)	0.76	0.12 (0.29)	0.68	-0.14 (0.07)	0.06	-0.77 (1.08)	0.48	0.69 (0.25)	0.01
Indian	0.61 (0.22)	0.01	-0.07 (0.21)	0.76	-0.18 (0.34)	0.60	-0.04 (0.18)	0.82	-6.20 (1.59)	<0.001	1.43 (0.39)	<0.001
Pakistani / Bangladeshi	1.26 (0.14)	<0.001	-0.25 (0.04)	<0.001	-0.12 (0.18)	0.52	-0.17 (0.05)	0.002	-13.97 (1.01)	<0.001	2.60 (0.24)	<0.001
Black	0.07 (0.21)	0.75	0.07 (0.08)	0.37	-0.61 (0.36)	0.09	0.01 (0.08)	0.89	-8.48 (1.42)	<0.001	2.19 (0.38)	<0.001
Other	1.03 (0.40)	0.01	-0.09 (0.13)	0.46	-0.35 (0.45)	0.44	-0.08 (0.12)	0.51	-7.14 (2.49)	0.004	1.20 (0.48)	0.01
Mother is university-educated	-0.24 (0.07)	0.001	-0.04 (0.03)	0.21	-1.04 (0.11)	<0.001	0.09 (0.03)	0.001	6.13 (0.54)	<0.001	0.07 (0.14)	0.59
Low birth weight	0.40 (0.16)	0.01	0.05 (0.05)	0.38	0.27 (0.16)	0.08	0.06 (0.06)	0.31	-2.88 (0.88)	0.001	0.25 (0.19)	0.20
Maternal age at birth	-0.02 (0.01)	0.001	0.00 (0.00)	0.74	-0.06 (0.01)	<0.001	-0.00 (0.00)	0.70	0.03 (0.04)	0.38	-0.00 (0.01)	0.71
Not breastfed	0.29 (0.08)	<0.001	-0.06 (0.03)	0.05	0.42 (0.12)	0.001	0.00 (0.04)	0.97	-3.36 (0.44)	<0.001	0.22 (0.13)	0.09
Maternal psychological distress[a]												
Intercept	0.31 (0.01)	<0.001	---	---	0.38 (0.02)	<0.001	---	---	-0.24 (0.07)	0.001	---	---
Slope	---	---	0.47 (0.08)	<0.001	---	---	0.34 (0.06)	<0.001	---	---	-0.23 (0.20)	0.25
Socioeconomic disadvantage[a]												
Intercept	0.21 (0.04)	<0.001	---	---	0.38 (0.06)	<0.001	---	---	-3.03 (0.24)	<0.001	---	---
Slope	---	---	-0.24 (0.22)	0.26	---	---	-0.13 (0.21)	0.54	---	---	-3.63 (1.30)	0.01
Females												
Ethnicity												
White	Ref	NA	Ref	NA	Ref	NA	Ref	NA	Ref	NA	Ref	NA
Mixed	0.34 (0.19)	0.07	-0.05 (0.07)	0.41	0.17 (0.28)	0.56	-0.03 (0.08)	0.71	-1.60 (1.09)	0.14	0.57 (0.29)	0.05
Indian	0.67 (0.23)	0.004	-0.25 (0.06)	<0.001	0.12 (0.33)	0.72	-0.14 (0.08)	0.07	-7.27 (1.38)	<0.001	2.16 (0.25)	<0.001
Pakistani / Bangladeshi	1.48 (0.22)	<0.001	-0.20 (0.06)	<0.001	0.06 (0.27)	0.84	-0.09 (0.08)	0.26	-14.28 (0.83)	<0.001	2.99 (0.19)	<0.001
Black	0.35 (0.23)	0.12	-0.16 (0.06)	0.01	-0.78 (0.36)	0.03	0.02 (0.07)	0.82	-7.64 (0.97)	<0.001	2.27 (0.30)	<0.001
Other	1.00 (0.31)	0.001	-0.36 (0.10)	<0.001	-0.56 (0.33)	0.09	-0.08 (0.11)	0.45	-7.78 (1.69)	<0.001	2.97 (0.39)	<0.001
Mother is university-educated	-0.20 (0.08)	0.01	-0.04 (0.03)	0.17	-0.70 (0.10)	<0.001	0.01 (0.02)	0.56	5.45 (0.47)	<0.001	0.04 (0.12)	0.74
Low birth weight	0.11 (0.13)	0.43	0.04 (0.04)	0.28	0.62 (0.18)	0.001	-0.03 (0.05)	0.51	-3.17 (0.68)	<0.001	0.62 (0.16)	<0.001
Maternal age at birth	-0.03 (0.01)	<0.001	0.00 (0.00)	0.14	-0.07 (0.01)	<0.001	0.01 (0.00)	0.003	0.00 (0.04)	0.90	0.01 (0.01)	0.32
Not breastfed	0.33 (0.08)	<0.001	-0.11 (0.03)	<0.001	0.59 (0.11)	<0.001	-0.11 (0.03)	<0.001	-2.64 (0.39)	<0.001	0.29 (0.11)	0.01

Maternal psychological distress[a]												
Intercept	0.30 (0.02)	<0.001	---	---	0.30 (0.02)	<0.001	---	---	-0.22 (0.06)	<0.001	---	---
Slope	---	---	0.40 (0.05)	<0.001	---	---	0.35 (0.04)	<0.001	---	---	-0.06 (0.14)	0.70
Socioeconomic disadvantage[a]												
Intercept	0.15 (0.04)	<0.001	---	---	0.36 (0.05)	<0.001	---	---	-3.13 (0.21)	<0.001	---	---
Slope	---	---	-0.36 (0.18)	0.04	---	---	-0.08 (0.16)	0.61	---	---	-2.02 (0.78)	0.01
[a]Time-varying covariates												

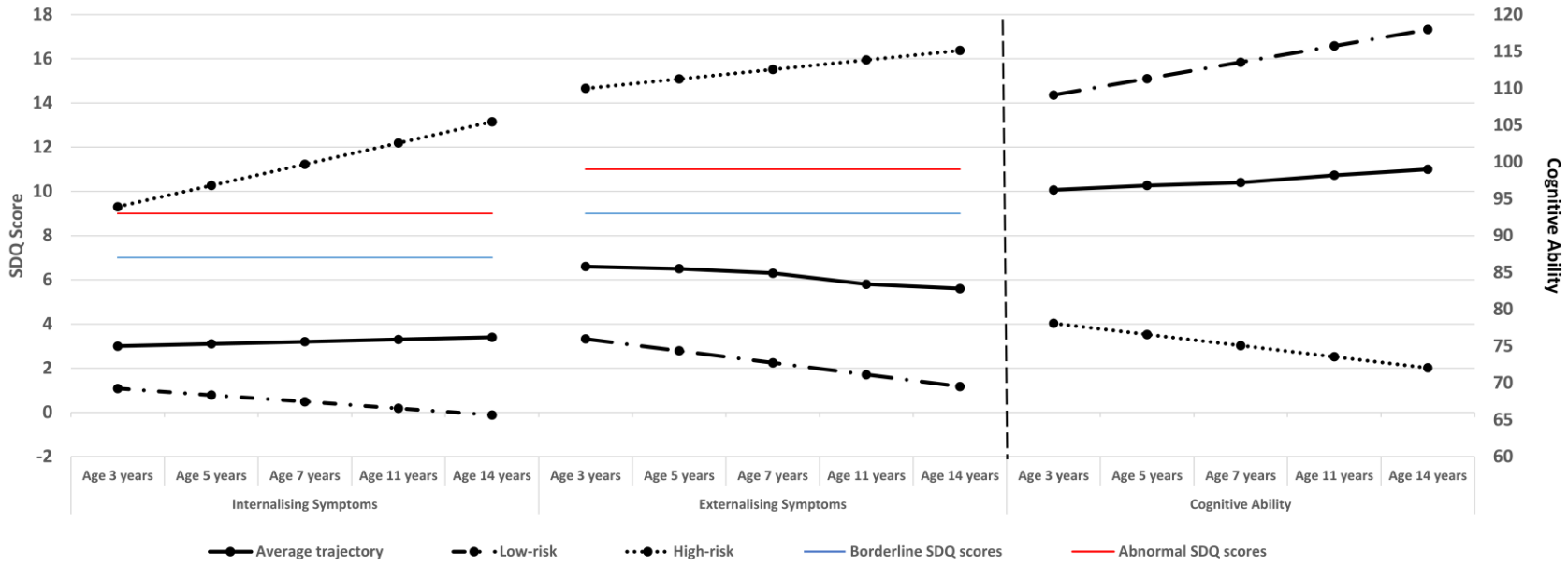

Figure 1. Predicted latent growth curve trajectories of internalising symptom scores, externalising symptom scores and cognitive ability scores in males with time-varying and time-invariant covariates held to their mean (average) and to their extremes (low-risk and high-risk) values.

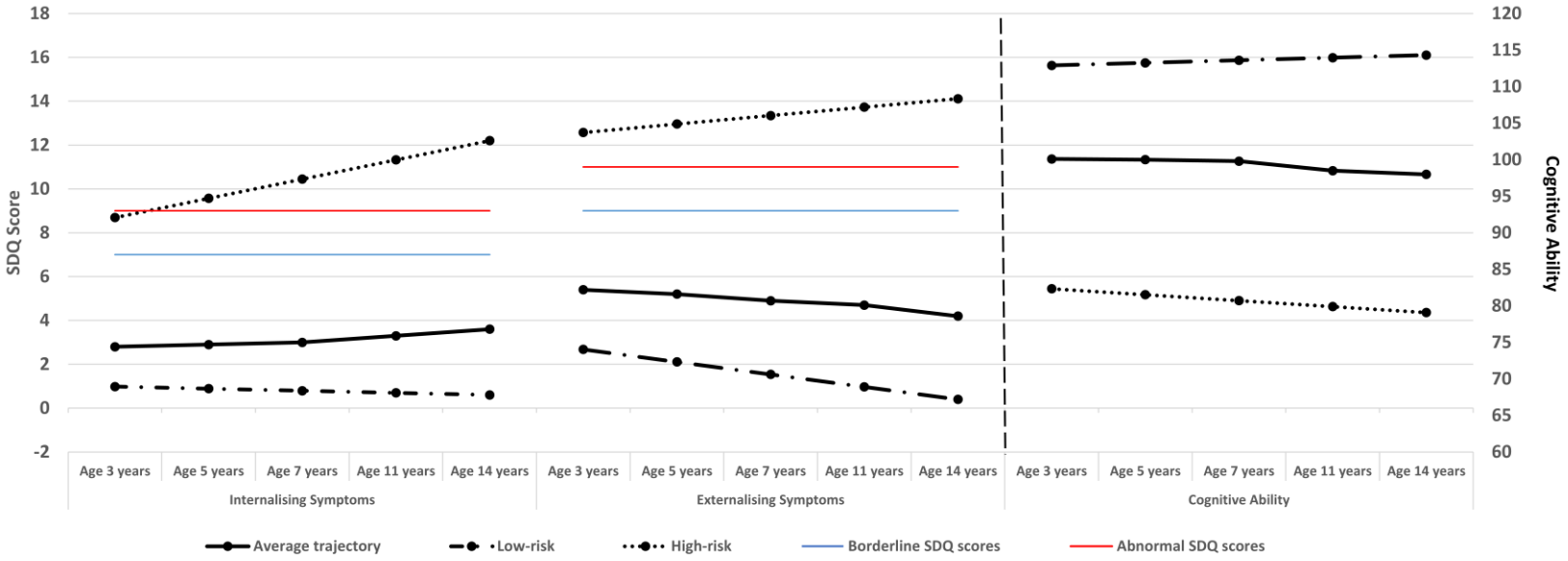

Figure 2. Predicted latent growth curve trajectories of internalising symptom scores, externalising symptom scores and cognitive ability scores in females with time-varying and time-invariant covariates held to their mean (average) and to their extremes (low-risk and high-risk) values.

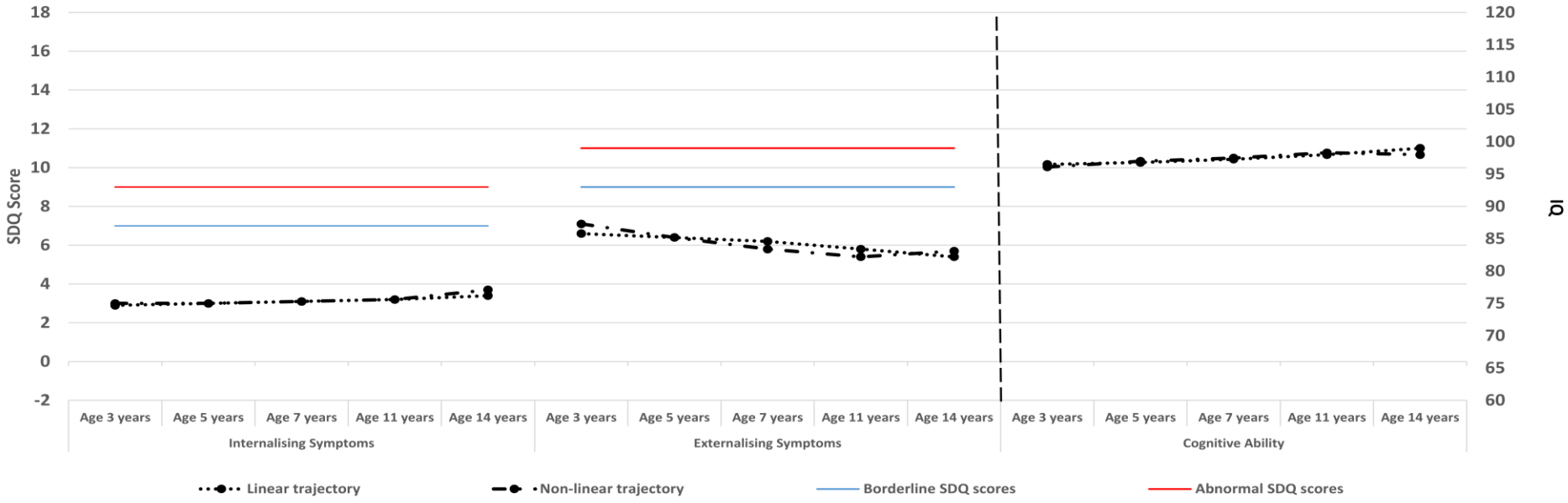

Figure 3. Linear and non-linear trajectories of internalising symptom scores, externalising symptom scores and cognitive ability scores in males.

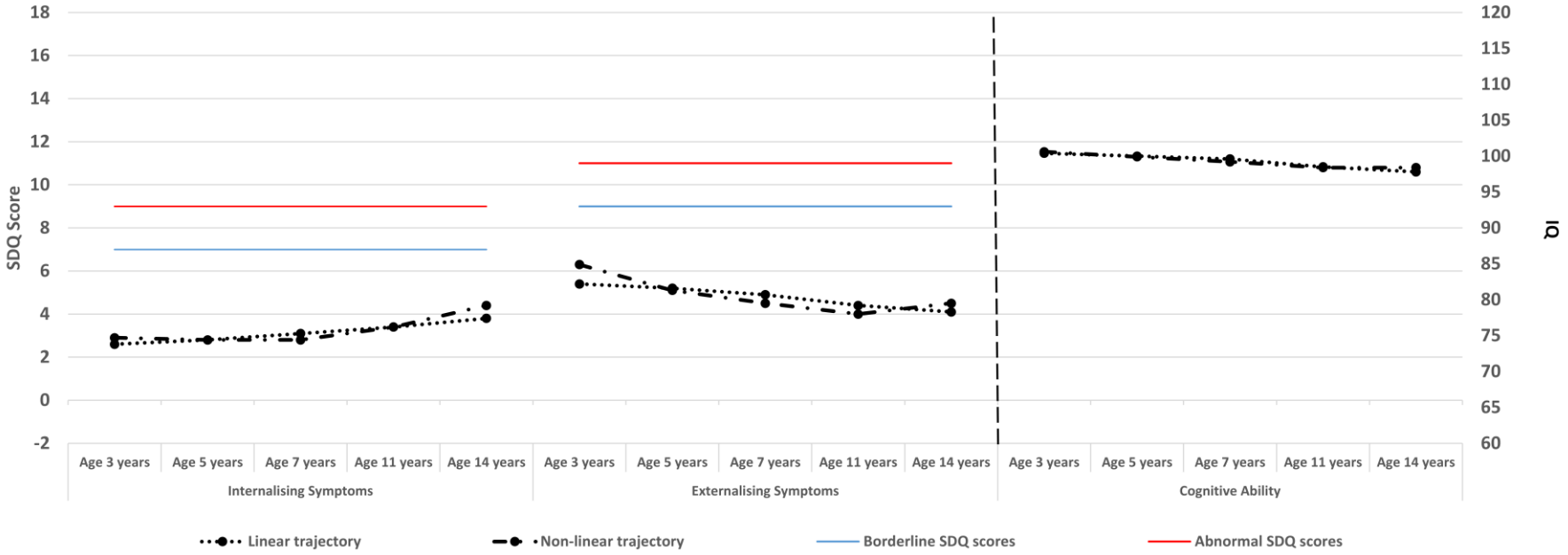

Figure 4. Linear and non-linear trajectories of internalising symptom scores, externalising symptom scores and cognitive ability scores in females.