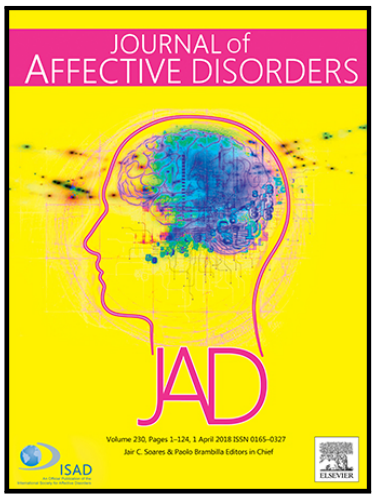

Distinct developmental trajectories of internalising and externalising symptoms in childhood: Links with mental health and risky behaviours in early adolescence

Efstathios Papachristou Ph.D. , Eirini Flouri Ph.D.

PII: S0165-0327(20)32583-0
DOI: https://doi.org/10.1016/j.jad.2020.07.130
Reference: JAD 12300

To appear in: *Journal of Affective Disorders*

Received date: 28 February 2020
Revised date: 11 July 2020
Accepted date: 28 July 2020

Please cite this article as: Efstathios Papachristou Ph.D. , Eirini Flouri Ph.D. , Distinct developmental trajectories of internalising and externalising symptoms in childhood: Links with mental health and risky behaviours in early adolescence, *Journal of Affective Disorders* (2020), doi: https://doi.org/10.1016/j.jad.2020.07.130

Highlights

- Increasing 'pure' internalising symptom trajectories in childhood are associated with self-harm, depression and drug use in early adolescence.
- 'Pure' externalising problem trajectories appear to have poorer predictive value.
- Compared to asymptomatic children those with high yet decreasing levels of externalising symptoms are less likely to be in trouble with the police in early adolescence.

Research paper

Distinct developmental trajectories of internalising and externalising symptoms in childhood: Links with mental health and risky behaviours in early adolescence

Efstathios Papachristou, Ph.D. [a]

Eirini Flouri, Ph.D.[a]

[a]UCL Institute of Education, University College London

Corresponding author: Efstathios Papachristou, Department of Psychology and Human Development, UCL Institute of Education, University College London, 25 Woburn Square, London WC1H 0AA, UK. Email: efstathios.papachristou@ucl.ac.uk. Tel: +442076126706.

Abstract

Background:High or increasing trajectories of internalising and externalising symptoms in childhood are mutually re-enforcing and associated with poor emotional and behavioural outcomes in adolescence. This study sought to identify classes of children with similar trajectories of internalising and externalising symptoms after adjusting one domain's symptoms for the other's at the classification stage, and to relate them to emotional and behavioural outcomes in mid-adolescence.

Methods:We used growth mixture modelling to classify 16,844 members of the Millennium Cohort Study (baseline N=19,244) into distinct classes based on their trajectories, across ages 3,5,7 and 11 years, of internalising and externalising symptoms adjusted for one

another. We examined the predictive ability of these classes for depression, self-harm, trouble with police and drug use among 11,134 children with available data at age 14.

Results:We identified four classes of children following distinct trajectories of 'pure' internalising and externalising symptoms. After adjustments for confounding, those with increasing or initially high yet decreasing levels of internalising symptomatology, and those with persistently high or increasing levels of externalising problems were at increased risk of depression in early adolescence. Having initially low yet increasing levels of internalising symptomatology was additionally associated with an increased risk of self-harm and drug use in early adolescence.

Limitations: We cannot ascertain whether our longitudinal typology of internalising and externalising symptoms holds for outcomes later in adolescence or adulthood.

Conclusions:Interventions aiming to prevent depression, drug use or self-harm in mid-adolescence may be more successful if they target children showing increasing internalising symptoms in the primary school years.

Keywords: Depression; drug use; externalising symptoms; internalising symptoms; self-harm; trouble with police.

Internalising (emotional) and externalising (behavioural) problems or symptoms in childhood are associated with a range of later adverse outcomes (Loth et al., 2014, Reef et al., 2010, Von Stumm et al., 2011). However, longitudinal evidence has now established that there is significant heterogeneity in how they develop during the childhood years in the general population (Hofstra et al., 2002, Hofstra and Verhulst, 2000). As a result, a growing number of studies are using trajectory-based approaches to identify subpopulations of children and adolescents within general population samples who follow distinct developmental courses of internalising and externalising symptoms (Flouri et al., 2018, Nivard et al., 2017, Parkes et al., 2016, Patalay et al., 2015, Patalay et al., 2016, Yoon, 2017). While the number and type of developmental patterns extracted in these studies vary according to the measurement instrument used, sample size, number of follow-ups or the age of the sample, there is agreement that a significant proportion of children – ranging

from approximately 11% to 30% - follow developmental trajectories characterised by persistently high or increasing levels of emotional and behavioural problems. Children on such trajectories tend to show poorer decision-making skills and academic attainment, and to have lower self-esteem and a higher probability of engaging in antisocial behaviours (Flouri et al., 2018, Patalay et al., 2016).

A common limitation of studies aiming to identify groups with distinct developmental trajectories of symptoms in each of these two domains in childhood is that they model them separately despite evidence suggesting that internalising and externalising symptoms are highly comorbid and mutually reinforcing (Boylan et al., 2010, Flouri et al., 2019, Morin et al., 2017, Wiesner, 2003). To the best of our knowledge, two studies to date have modelled both types of symptom dimensions concurrently, albeit using different methodological approaches (Flouri et al*.,* 2018; Nivard et al*.,* 2017). Flouri et al. (2018) identified subpopulations with joint developmental trajectories of internalising and externalising symptoms using growth mixture modelling. Nivard et al. (2017) considered the two domains separately and used regression models to examine similarities and overlap of the extracted developmental trajectories. However, mutual adjustments for the two symptom dimensions were not considered in either of the two studies at the classification stage. It is thus likely that estimates for the association between developmental trajectories of symptoms in one domain and later 'cross-domain' outcomes are inflated. Without considering both types of problems, one also ignores the now strong evidence for heterotypic continuity of childhood problems (particularly for girls) (Costello et al., 2003). For example, externalising problems such as noxious behaviours and lack of social skills can alienate peers which increases vulnerability to internalising symptoms (Gooren et al., 2011), in turn associated with future psychiatric illness including affective disorders (Dingle et al., 2011, Qualter et al., 2010). The rates of homotypic continuity of internalising and externalising symptoms for 'same-domain' outcomes might also be affected. Homotypic continuity is generally more prominent for ADHD and behavioural disorders (Ford et al*.,* 2017, Wichstrøm et al*.,* 2017), but holds for affective disorders too. For example, results from the British Child and Adolescent Mental Health Survey suggest that homotypic persistence of psychiatric disorders characterised by emotional (for example depression)

and behavioural (for example ADHD) problems are approximately 19% and 44%, respectively (Ford et al*.,* 2017).

With this study we attempted to extract 'pure' symptom trajectories across childhood and estimate their independent predictive ability for adverse emotional (depression and self-harm) and behavioural (drug use and trouble with the police) outcomes in early adolescence. We expected that elevated levels of externalising symptoms in childhood would increase the risk for adolescent problem behaviours in the same broad domain, such as trouble with the police and drug use. We also expected that elevated levels of internalising symptoms would predict adolescents' risk of depression, self-harm and drug use, albeit we expected the predictive ability of internalising symptoms for the outcomes to be slightly weaker in line with the evidence for homotypic continuity mainly for ADHD and behavioural disorders (Ford et al*.,* 2017, Wichstrøm et al., 2017).

Methods

Sample

We used data from the Millennium Cohort Study (MCS), a population-based cohort of children born in the UK over 12 months from September 2000 (Connelly and Platt, 2014, Joshi and Fitzsimons, 2016). The children were around 9 months old at Sweep 1, and 3, 5, 7, 11 and 14 years old at Sweeps 2-6, respectively. In total, 19,244 families participated in MCS. Ethical approval for the MCS was gained from NHS Multi-Centre Ethics Committees, and parents (and children after age 11 years) gave informed consent before interviews took place. Our sample included children (singletons and first-born twins or triplets) with valid data on internalising and externalising symptoms in at least one of Sweeps 2 (when they were first measured in MCS) to 5 (90% of total sample size; N=16,844; 51% male). As will be explained in more detail below, our analytic sample was restricted for further analyses to 11,134 (58% of total sample size; 50% male) to include those with available data on at least one of the outcomes considered at age 14 (Sweep 6). Figure 1 illustrates the flow chart of the study.

Measures

Internalising and externalising symptoms

These were assessed with the parent-reported Strengths and Difficulties Questionnaire (SDQ) at cohort member's ages 3, 5, 7 and 11 years. The SDQ is a short and psychometrically-valid behavioural screening tool (Goodman, 1997). It includes four scales of difficulties: emotional symptoms, conduct problems, hyperactivity, and peer problems. In line with recommended practice for community samples (Goodman et al., 2010), the internalising scale comprised the 10 SDQ items from the emotional and peer problems scales, and the externalising scale the 10 items from the hyperactivity and conduct problems scales. Scores on these two scales can range 0-20 with higher scores indicating more serious problems or symptoms. In the analytic sample, the internal consistency (Cronbach's alpha) of the scales was satisfactory across assessments, ranging from 0.61 (internalising symptoms at age 3) to 0.81 (externalising symptoms at age 11).

Outcomes

All emotional, social and behavioural outcomes considered (depression, self-harm, trouble with the police and drug use) were assessed using self-reported information at age 14 years (Sweep 6). *Depression* was measured with the Mood and Feelings Questionnaire – short version (SMFQ). The SMFQ is a psychometrically sound instrument validated for use in children aged 6 years or older comprising 13 items on affective symptoms occurring in the last 2 weeks (Angold et al., 1995). The internal consistency of the SMFQ in the analytic sample was excellent (Cronbach's alpha=0.93). We dichotomised the outcome at scores >=8 because this cut-off provides good diagnostic accuracy for major depression (sensitivity 60%, specificity 85%) as diagnosed with the Diagnostic Interview Schedule for Children (DISC-C) (Angold et al., 1995). The unweighted prevalence of depression at age 14 years in our analytic sample was 28% (N=3,068). A dichotomous question asking whether cohort members had self-harmed in the past year was used to measure *self-harm* [15% (unweighted) (N=1,634) of our analytic sample responded positively to the question]. *Drug use* was assessed using two dichotomous items asking whether cohort members had ever tried "cannabis (also known as weed, marijuana, dope, hash or skunk)" or "any other illegal drug (such as ecstasy, cocaine, speed)". Anyone with a positive answer on either of the two questions was classified as having used drugs (5% (unweighted); N=509). Cohort members were also asked to report whether they had a) "ever been given a formal warning or caution from police"; b) "been stopped or questioned by police"; and c) "ever been arrested". Those

responding positively to at least one of these items were classified as having been in *trouble with the police* (16% (unweighted); N=1,794).

Covariates

We adjusted for time-invariant and time-varying confounders which have been demonstrated to relate to internalising and externalising symptoms as well as to the outcomes we considered, including *sex, ethnicity, socioeconomic disadvantage, cognitive ability, maternal education* and *maternal psychological distress* (Choe et al*.,* 2013, Flouri et al*.,* 2018, Huisman et al*.,* 2010, Hackman and Farah, 2009, Koenen et al*.,* 2009, Linver et al*.,* 2002, Karestan et al*.,* 2009, Maxwell et al*.,* 2018, Papachristou and Flouri, 2019, Weinstock, 2005).

We considered as time-invariant covariates *sex*, *age* in years at baseline, *ethnicity* (white vs. other), and *maternal education* (university degree or not). The time-varying covariates assessed at cohort member's ages 3, 5, 7 and 11 years included *maternal psychological distress* [measured using the Kessler K6 (Kessler et al., 2002)], *socioeconomic disadvantage* and *cognitive ability*. Socioeconomic disadvantage 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 and Flouri, 2011). Cognitive ability was calculated for each age by using the age-adjusted ability assessments that were available in MCS.

Statistical analysis

In all analyses the MCS stratum was controlled for to account for the disproportionately stratified design of the study. Attrition and non-response were taken into account by using study-specific weights. Analyses were performed in Stata SE 15.1 (StataCorp, 2011) and Mplus 7.4 (Muthén and Muthén, 2009).

We grouped cohort members in latent classes with similar developmental trajectories of internalising and externalising symptoms, using growth mixture modelling (GMM) (Wickrama et al., 2016). GMM is an extension of latent class analysis whereby the latent class indicators are the growth parameters, i.e. the intercept (scores at baseline) and

the slope (rates of change), of a prospectively assessed outcome. The assignment of individuals to classes is based on "posterior probabilities" which are similar to factor loadings and each person is classified into the class to which she has the highest posterior probability of belonging. In order to obtain classes of one domain's symptoms adjusted for the other's we regressed externalising symptoms on internalising symptoms across waves when extracting classes of externalising symptoms, and vice versa. Compared to other trajectory-based approaches, GMM relaxes the assumption of homogeneity of variance of the growth parameters across classes, which can lead to incorrect conclusions regarding the number and nature of the trajectories, and can take into consideration non-normal distributions of the data (Infurna and Grimm, 2018). In addition, GMM, as implemented in Mplus, allows observed variables to be regressed on the repeatedly assessed outcome variable, thus allowing for the extraction of 'pure' (since we adjusted for the 'opposite' symptom domain at the classification stage) symptom trajectories.

We estimated models with 1 to 7 classes and compared model fit with five goodness of fit indices: a) the Bayesian Information Criterion (BIC; lower values indicate better model fit); b) the Sample-Size Adjusted BIC (SSA-BIC; lower values indicate better model fit); c) the Akaike Information Criterion (AIC; lower values indicate better model fit); d) the entropy of each model (values >=0.80 indicate little ambiguity in class allocation); and e) the Lo–Mendell–Rubin (LMR) likelihood ratio test which compares fit of two adjacent models with K and K-1 number classes (p-values<0.05 indicate a significantly better fit of the model with K number of classes) (Jung and Wickrama, 2008). Models with small classes (<3% of the sample) were deemed substantively inconsequential and thus disregarded (Berlin et al., 2014). To avoid model convergence to local maxima we increased the number of starts (500 random sets of starting values and 50 optimisations in the final stage). The models were run under the assumption that the variances and covariances of growth parameters are equal across classes (the Mplus default). In well-fitting models this restriction does not substantively alter the results and aids convergence. Our models were carried out using the maximum likelihood with robust standard errors estimator, which takes into account skewed distribution in the data. Full information maximum likelihood (FIML) was used to accommodate missing data on internalising and externalising symptoms. FIML estimates

parameters using all available information that is contained in the analytic model and is considered to be superior to other methods traditionally used to treat missing data.

Next, we used the manual 3-step procedure for incorporating distal outcomes to GMM to assess the predictive ability of class membership for the outcomes (Asparouhov and Muthén, 2014, Wickrama et al., 2016). Having identified the best-fitting model for both internalising and externalising symptom trajectories (Step 1) we used the variables identifying the class to which each cohort member most likely belongs from the latent class posterior distributions to specify a latent class indicator variable which takes into account misclassification error rates by fixing the means of each class using the logit values obtained from Step 1 (Step 2). In the final step (Step 3) we included each outcome successively to calculate crude odds ratios (ORs) and 95% confidence intervals (CI) of class membership for the four distal outcomes (Model A). We also regressed each of the outcomes on the covariates to obtain adjusted ORs (95% CI) of class membership (Model B). Because FIML cannot be used at this step to accommodate missing data on covariates, these analyses (Model B) were run on 25 imputed datasets generated using sequential regression models (Asparouhov and Muthén, 2010). We removed from the analysis covariates with variance inflation factor (VIF) estimates >4 to avoid obtaining biased standard errors due to multicollinearity. Figure S1 in the Supplementary Material illustrates the analytic model of the study.

Results

Model selection

The GMMs were run on a sample of 16,844 children (51% male) with available data on the SDQ at any of the assessments at ages 3, 5 7 or 11 years. The mean internalising symptom scores in the analytic sample ranged from 2.55 (SD=2.54) at age 5 to 3.22 (SD=3.16) at age 11 while the mean externalising symptom scores from 4.75 (SD=3.60) at age 7 to 6.73 (SD=3.82) at age 3.

In the GMM for internalising symptom scores we identified a 4-class solution as optimal compared to other solutions based on the entropy of the model and the LMR p-value (=0.02) (Table S1 in the Supplementary Material). The 5-class solution had lower BIC, AIC and SSA-BIC values, but also lower entropy and a non-significant LMR p-value (p=0.64)

indicating worse fit. The average latent class probabilities for most likely class membership in the selected 4-class solution ranged between 79% and 92% indicating minimal ambiguity in class assignment. Figure 2 illustrates the internalising symptom trajectories of the four classes. We identified a class characterised by persistently low scores of internalising symptoms throughout the study period (N=13,230; 79% of the sample; intercept=2.12, SE=0.04, p<0.001; slope=-0.06, SE=0.01, p<0.001), a class with low levels of internalising symptoms at baseline (age 3) which increased at a significant rate across subsequent assessments (N=1,804, 11% of the sample; intercept=2.70, SE=0.11, p<0.001; slope=1.15, SE=0.07, p<0.001), a class characterised by high levels of internalising symptoms at baseline which decreased at a significant rate across subsequent assessments (N=1,332, 8% of the sample; intercept=7.59, SE=0.24, p<0.001; slope=-0.77, SE=0.06, p<0.001), and a smaller class characterised by high levels of internalising symptoms at baseline which increased even further at a significant rate during the study period (N=478, 3% of the sample; intercept=5.36, SE=0.47, p<0.001; slope=1.86, SE=0.13, p<0.001).

Results of the GMM for externalising symptom scores also suggested that a 4-class solution is optimal. Similar to the results of the GMM for internalising symptoms, the 5-class solution had lower BIC, AIC and SSA-BIC values, but also lower entropy and a non-significant LMR p-value (p=0.09) indicating worse fit (Table S2 in the Supplementary Material). Moreover, two of the extracted classes of the 5-class solution were very similar, and hence redundant in terms of interpretability. For the 4-class solution the average latent class probabilities for most likely class membership ranged between 79% and 93% indicating minimal ambiguity in class assignment. Figure 3 illustrates the externalising symptom trajectories of the four classes. Children allocated in the first class were characterised by persistently low levels of externalising symptoms (N=14,101, 84% of the sample; intercept=5.47, SE=0.06, p<0.001; slope=-0.40, SE=0.02, p<0.001). Those in the second class had persistently high levels of externalising problems (N=817, 5% of the sample; intercept=9.18, SE=0.31, p<0.001; slope=-0.33, SE=0.12, p=0.01); despite the significant negative slope observed for externalising symptom trajectories in this class the average score of externalising problems at age 11 was higher compared to those of the three remaining classes, hence we termed this class “persistently high”. Those in the third class had high levels of externalising symptoms at baseline which increased at a significant rate

across assessments (N=760, 5% of the sample; intercept=7.17, SE=0.34, p<0.001; slope=0.34, SE=0.13, p=0.001), and finally, those in the fourth class had high levels of externalising symptoms at baseline which decreased at a significant rate throughout the study period (N=1,166, 7% of the sample; intercept=8.84, SE=0.28, p<0.001; slope=-0.63, SE=0.08, p<0.001).

A cross tabulation examining the relationship between the variables measuring class membership for the two symptom domains showed that, by and large, the trajectories of internalising and externalising symptoms develop similarly (p<0.01). The results are summarised in Table S3 in the Supplementary Material. The majority of those with persistently low levels of internalising symptoms (97%) also had persistently low levels of externalising symptoms. Of those with low yet increasing levels of internalising symptoms the majority (75%) had persistently low (44%) or high and increasing (31%) levels of externalising symptoms. Those with high yet decreasing levels of internalising symptoms had predominantly (54%) high and decreasing levels of externalising symptoms. Finally, of those with high and increasing levels of internalising symptoms, 40% also had high and increasing levels of externalising symptoms.

Predictive value of class membership for outcomes

Of the 16,844 children included in the GMM, 11,134 had available data on at least one of the four outcomes considered. Those lost due to attrition (N=5,710) were more likely to be male (54%, p<0.001), and had higher levels of internalising and externalising symptoms and poorer cognitive ability at all assessments (all p-values<0.001). Moreover, their mothers were less likely to be university-educated (10% with university degree, p<0.001) and had higher levels of psychological distress (p-values<0.001 across all assessments). In terms of their internalising symptom profiles, those lost due to attrition were more likely to have had high and increasing levels of internalising symptoms (41%), followed by high (40%), low (34%) and low and increasing (29%) levels (p<0.01). In terms of their externalising symptom profiles, those lost due to attrition were more likely to have had persistently high levels (43%), followed by persistently low (43%), high and decreasing (39%) and high and increasing (27%) levels (p<0.01).

Table 1 summarises the characteristics of the sample in terms of the covariates considered in the regression analyses by class membership. Overall, children with persistently low levels of internalising and externalising problems were more likely to be female, white and to have more educated mothers. In addition, they were of higher socioeconomic status, had less psychologically distressed mothers, and higher cognitive ability at all four assessments compared to their counterparts allocated in the remaining classes (all p-values <0.05).

Table 2 summarises the ORs (95% CI) for the relationships between class membership and outcomes at age 14 years. Unadjusted regression models suggest that compared to children with “persistently low” levels of internalising symptoms between ages 3 and 11 years, those with “high and increasing” and “low and increasing” levels of internalising symptoms were more likely to score >=8 on the SMFQ, to self-harm, to use drugs and to be in trouble with the police at age 14. Moreover, those with “high and decreasing” levels of internalising symptoms were more likely to use drugs and be in trouble with the police at age 14. Similarly, compared to children with “persistently low” levels of externalising symptoms, those with “persistently high” and those with “high and increasing” levels of externalising symptoms were more likely to score >=8 on the SMFQ, to report that they were self-harming and to report trouble with the police at age 14. Only those with “persistently high” levels of externalising symptoms were at a significantly increased risk for drug use. Next, we ran these models adjusting for sex, ethnicity, maternal education and time-varying socioeconomic disadvantage (at ages 3, 7 and 11 years), maternal psychological distress (at ages 3, 5, 7, and 11 years) and cognitive ability (at ages 3, 5, 7, and 11 years). We removed socioeconomic disadvantage assessed at age 5 from the list of covariates because it was highly collinear with the remaining assessments of socioeconomic disadvantage. The results of these models showed that compared to children with “persistently low” levels of internalising symptoms at ages 3 to 11 years, those allocated in the remaining three classes were more likely to score >=8 on the SMFQ at age 14. In addition, those with “low and increasing” levels of internalising symptoms were at an increased risk of self-harming and using drugs at age 14. Children with “persistently high” and those with “high and increasing” levels of externalising symptoms were more likely to

score >=8 on the SMFQ at age 14 compared to their counterparts allocated in the class with "persistently low" levels of externalising symptoms.

Discussion

In this study we sought to identify groups of children who follow similar developmental trajectories of internalising and externalising symptoms, adjusted for one another, from the preschool years to the end of primary school, and to explore group differences in risks for adverse emotional and behavioural outcomes (depression, self-harm, drug use and trouble with the police) in early adolescence. The results suggested that children in the general populations follow 4 distinct trajectories of 'pure' internalising and externalising symptoms between ages 3 and 11 years. Moreover, we found that after adjustments for confounding, those with increasing or high at baseline yet decreasing levels of internalising symptoms, as well as those with persistently high or high at age 3 and further increasing levels of externalising problems are at a 1.4 to 2.3-fold increased risk of depression at age 14 years. Having initially low yet increasing levels of internalising symptomatology was additionally associated with an increased risk of self-harm and drug use in early adolescence. In contrast to our hypothesis, the strongest predictor of the adverse outcomes considered was high or increasing levels of internalising symptoms in childhood which suggests that interventions aiming to prevent these outcomes in adolescence may be more successful if they target children showing emotional problems levels in the primary school years.

The classes extracted for both symptom domains resemble those identified in previous studies (Flouri et al., 2018, Nivard et al., 2017, Parkes et al., 2016, Patalay et al., 2015, Patalay et al., 2016, Yoon, 2017). In our sample, 5% (N=817) had persistently elevated levels of externalising symptoms throughout childhood and 3% (N=473) had high levels of internalising symptoms at age 3 which increased even further during the study period. For a long period of time these two groups had, respectively, externalising and internalising symptom scores above our measure's cut-off for 'borderline' scores (>=9 on the externalising symptoms scale and >=7 on the internalising symptoms scale) according to the widely-used normative banding of SDQ scores for UK samples (Goodman, 1997). Based on evidence that approximately 85% of children and adolescents referred to community child and adolescent mental health services score above 'borderline' cut-off (Mathai et al., 2002)

it is likely that these children in our sample represent a high-risk group for future psychopathology.

Turning to the outcomes considered in this study, it appears that compared to the majority of children in the general population who, as expected, remain symptom-free during childhood, those with high or increasing levels of internalising or externalising symptoms are by and large at a significantly elevated risk for depression, drug use, trouble with the police and self-harm in early adolescence. After adjustments for sex, ethnicity, socioeconomic disadvantage, maternal education, maternal psychological distress and cognitive ability those with increasing or initially high yet decreasing levels of internalising symptomatology and those with persistently high or high at baseline and further increasing levels of externalising problems were at a significantly increased risk for future depression, while those with low at baseline yet increasing levels of internalising symptomatology were additionally at increased risk of self-harming and using drugs in early adolescence. Together, these findings highlight the important predictive value of chronically high or rising levels of internalising symptoms in childhood for a range of related but distinct mental health problems in early adolescence. Future research could build on these results to additionally examine possible distinct biological or genetic features of children following trajectories characterised by high or increasing levels of emotional problems to inform risk assessments in clinical care and shed light in potentially causal mechanisms linking internalising symptoms with future depression, drug use and suicide risk. For example, dysregulation of the Hypothalamic-Pituitary-Adrenal (HPA) axis or the immune system could mediate some of the associations, particularly with depression or suicide risk (Pompili et al., 2012). Additionally, we found an association between decreasing externalising symptoms and trouble with the police in early adolescence: compared to children with persistently low externalising symptoms, those with high externalising symptoms at baseline which decreased thereafter were 30% less likely to be in trouble with the police in early adolescence, even after adjustments for confounding. This finding suggests that externalising problems in childhood could be targeted by therapeutic and preventative efforts aiming to reduce antisocial behaviours in adolescence.

One of the unexpected findings of our study was that after adjustments for confounders, compared to their (internalising) symptom-free counterparts, children with

low at baseline yet increasing levels – but not those with high at baseline and increasing levels - of internalising symptoms were at a higher risk of both using drugs and self-harming at age 14 years. A potential explanation is that the latter group was the smallest of the ones extracted comprising only 3% of the sample, thus resulting in wide confidence intervals for the associated regression coefficient in the predictive models. However it should also be noted that the children in this group had mothers with the highest levels of psychological distress, and thus at high risk of depression themselves (Beardslee et al., 2011), as we also showed. In contrast, children with low at baseline yet increasing levels of internalising problems, a less cognitively and socially disadvantaged group as can be seen by their baseline characteristics, could exhibit different internalising symptoms, such as anxiety symptoms and worsening socio-emotional and coping skills, in turn associated with both drug use (Siennick et al., 2016, Taylor et al., 2017) and self-harm in adolescence (O'connor et al., 2009, Stanford et al., 2018).

Nonetheless, the outcomes for this study were measured at age 14 years and we cannot ascertain whether our longitudinal typology of childhood emotional and behavioural symptoms could still be relevant for outcomes later in adolescence or in adulthood. For example, adult-onset depression and adolescent-onset depression are likely aetiologically different (Thapar et al., 2012). In addition, we cannot exclude the possibility that some of the outcomes considered at age 14 had not already occurred at an earlier age. In this case the predictive ability of our typology for our outcomes would represent an artefact of cross-sectional associations. It is also possible that the outcomes are intertwined, too. For example, we treated self-harm and depression as two distinct outcomes, albeit the two are highly comorbid and mutually re-enforcing (Pompili et al., 2013). Future studies could aim to estimate the predictive ability of internalising and externalising symptom trajectories for emotional and behavioural multimorbidity to identify points of intervention for multiple comorbid adverse outcomes. Another critical point regarding the results presented in this study pertains to the use of composite internalising and externalising symptom scores, rather than the 4 individual SDQ subscales scores. Studies using the SDQ have shown that at the within-domain, SDQ subscales are differentially associated with some of the outcomes we considered (Muris et al., 2003). It is, thus, likely that the predictive value of the individual SDQ subscales for the outcomes we considered could yield somewhat different results. It

should also be noted that subject attrition may have led to a biased sample at the regression modelling stage. The sample was more likely to be male, white, and with more educated and less distressed mothers. Attrition was also more likely for some of the classes extracted compared to others, as we showed. Since the analytic sample is not representative of the baseline sample the generalisability of the regression estimates might be somewhat compromised. Nonetheless, attrition and non-response were largely accounted for by using study-specific weights in our analyses. In addition, it is likely that the children lost due to attrition during the latest follow-up would have been more likely to report emotional and behavioural problems at age 14 years since they were from more deprived backgrounds. Therefore it is likely that some of the observed associations between class membership and the outcomes would be stronger if attrition rates for the more disadvantaged children had been lower. Moreover, it is likely that unmeasured confounders (Wichstrøm et al*.,* 2017) could explain the relationships that we found to be significant between class membership and the outcomes. It is also possible that combining internalising and externalising symptom trajectories might be more predictive of certain emotional, social and behavioural outcomes. This hypothesis is plausible in light of the evidence suggesting strong mutually re-enforcing relationships between internalising and externalising symptoms during childhood and adolescence (Flouri et al*.,* 2019). Nonetheless, our study was designed to answer a different research question, the predictive ability of children's 'pure' internalising and externalising symptom trajectories for their outcomes in adolescence. Finally, the distinct trajectories of internalising and externalising symptoms identified in our study cover only the larger part of the first decade of life and thus we cannot make distinctions between early-onset persistent, childhood-limited or adolescent-onset symptoms, important distinctions for both conduct (Barker and Maughan, 2009, Barker et al., 2010, Bongers et al., 2004, Moffitt et al., 1996) and internalising problems (Nivard et al., 2017). Future research could aim to test patterns of trajectories over longer follow-up periods.

Overall, it appears that increasing levels of 'pure' internalising symptoms between ages 3 to 11 years are associated with self-harm, depression and drug use in early adolescence. Developmental trajectories characterised by increased levels of 'pure' externalising problems appear to have poorer predictive value in comparison. Perhaps the most important message of this study for clinicians, teachers, parents and those guiding

intervention planning is that preventing emotional problems in the primary school years may help prevent depression, drug use and self-harm in early adolescence.

Declaration of Competing Interest

None.

Acknowledgments: This work was supported by the Economic and Social Research Council [grant number ES/N007921/1]. The funding source had no involvement in the study design; in the collection, analysis and interpretation of data; in the writing of the report; or in the decision to submit the article for publication.

Contributors: E Papachristou performed the analyses and wrote the first draft of the manuscript. E Flouri reviewed the first draft and finalised the article. Both authors contributed to and have approved the final manuscript.

References

Angold, A., Costello, E. J., Messer, S. C., Pickles, A., 1995. Development of a short questionnaire for use in epidemiological studies of depression in children and adolescents. Int. J. Meth. Psych. Res. 5, 237-249.

Asparouhov, T., Muthén, B., 2010. Multiple imputation with Mplus. MPlus Web Notes.

Asparouhov, T., Muthén, B., 2014. Auxiliary variables in mixture modeling: Three-step approaches using M plus. Structural Equation Modeling: A Multidisciplinary Journal 21, 329-341.

Barker, E. D., Maughan, B., 2009. Differentiating early-onset persistent versus childhood-limited conduct problem youth. Am. J. Psychiat. 166, 900-908.

Barker, E. D., Oliver, B. R., Maughan, B., 2010. Co-occurring problems of early onset persistent, childhood limited, and adolescent onset conduct problem youth. J. Child Psycho. Psyc. 51, 1217-1226.

Beardslee, W. R., Gladstone, T. R., O'Connor, E. E., 2011. Transmission and prevention of mood disorders among children of affectively ill parents: A review. J. Am. Acad. Child Psy. 50, 1098-1109.

Berlin, K. S., Williams, N. A., Parra, G. R., 2014. An introduction to latent variable mixture modeling (part 1): Overview and cross-sectional latent class and latent profile analyses. J. Pediatr. Psychol. 39, 174-187.

Bongers, I. L., Koot, H. M., Van Der Ende, J., Verhulst, F. C., 2004. Developmental trajectories of externalizing behaviors in childhood and adolescence. Child Dev. 75, 1523-1537.

Boylan, K., Georgiades, K., Szatmari, P., 2010. The longitudinal association between oppositional and depressive symptoms across childhood. J. Am. Acad. Child Psy. 49, 152-161.

Choe, D. E., Olson, S. L., Sameroff, A. J., 2013. Effects of early maternal distress and parenting on the development of children's self-regulation and externalizing behavior. Dev. Psychopathol. 25, 437-453.

Connelly, R., Platt, L., 2014. Cohort profile: UK millennium Cohort study (MCS). Int. J. Epidemiol. 43, 1719-1725.

Costello, E. J., Mustillo, S., Erkanli, A., Keeler, G., Angold, A., 2003. Prevalence and development of psychiatric disorders in childhood and adolescence. Arch. Gen. Psychiatry 60, 837-844.

Dingle, K., Clavarino, A., Williams, G. M., Bor, W., Najman, J. M., Alati, R., 2011. Predicting depressive and anxiety disorders with the YASR internalising scales (empirical and DSM-oriented). Soc. Psychiatry Psychiatr. Epidemiol. 46, 1313-1324.

Flouri, E., Papachristou, E., Midouhas, E., Joshi, H., Ploubidis, G. B., Lewis, G., 2018. Early adolescent outcomes of joint developmental trajectories of problem behavior and IQ in childhood. Eur. Child Adolesc. Psychiatry 27, 1595–1605.

Flouri, E., Papachristou, E., Midouhas, E., Ploubidis, G. B., Lewis, G., Joshi, H., 2019. Developmental cascades of internalising symptoms, externalising problems and cognitive ability from early childhood to middle adolescence. Eur. Psychiatry 57, 61-69.

Ford, T., Macdiarmid, F., Russell, A.E., Racey, D., Goodman, R., 2017. The predictors of persistent DSM-IV disorders in 3-year follow-ups of the British Child and Adolescent Mental Health Surveys 1999 and 2004. Psychol. Med. 47, 1126-1137.

Goodman, A., Lamping, D. L., Ploubidis, G. B., 2010. When to use broader internalising and externalising subscales instead of the hypothesised five subscales on the Strengths and Difficulties Questionnaire (SDQ): data from British parents, teachers and children. J. Abnorm. Child Psychol. 38, 1179-1191.

Goodman, R., 1997. The Strengths and Difficulties Questionnaire: a research note. J. Child Psycho. Psyc. 38, 581-586.

Gooren, E. M., van Lier, P. A., Stegge, H., Terwogt, M. M., Koot, H. M., 2011. The development of conduct problems and depressive symptoms in early elementary school children: The role of peer rejection. J. Clin. Child Adolesc. Psychol. 40, 245-253.

Hackman, D. A., Farah, M. J., 2009. Socioeconomic status and the developing brain. Trends Cogn. Sci. 13, 65-73.

Hawton, K., Saunders, K. E., O'Connor, R. C., 2012. Self-harm and suicide in adolescents. Lancet 379, 2373-2382.

Hofstra, M. B., Van Der Ende, J., Verhulst, F. C., 2002. Child and adolescent problems predict DSM-IV disorders in adulthood: a 14-year follow-up of a Dutch epidemiological sample. J. Am. Acad. Child Psy. 41, 182-189.

Hofstra, M. B., Verhulst, F. C., 2000. Continuity and change of psychopathology from childhood into adulthood: a 14-year follow-up study. J. Am. Acad. Child Psy. 39, 850-858.

Huisman, M., Araya, R., Lawlor, D. A., Ormel, J., Verhulst, F. C., Oldehinkel, A. J., 2010. Cognitive ability, parental socioeconomic position and internalising and externalising problems in adolescence: findings from two European cohort studies. Eur. J. Epidemiol. 25, 569-580.

Infurna, F. J., Grimm, K. J., 2018. The use of growth mixture modeling for studying resilience to major life stressors in adulthood and old age: Lessons for class size and identification and model selection. J. Gerontol. B-Psychol. 73, 148-159.

Joshi, H., Fitzsimons, E., 2016. The Millennium Cohort Study: the making of a multi-purpose resource for social science and policy. Longitudinal and Life Course Studies 7, 409-430.

Jung, T., Wickrama, K., 2008. An introduction to latent class growth analysis and growth mixture modeling. Soc. Personal. Psychol. Compass 2, 302-317.

Kessler, R. C., Andrews, G., Colpe, L. J., Hiripi, E., Mroczek, D. K., Normand, S.-L., Walters, E. E., Zaslavsky, A. M., 2002. Short screening scales to monitor population prevalences and trends in non-specific psychological distress. Psychol. Med. 32, 959-976.

Koenen, K.C., Moffitt, T.E., Roberts, A.L., Martin, L.T., Kubzansky, L., Harrington, H., Poulton, R., Caspi, A., 2009. Childhood IQ and adult mental disorders: a test of the cognitive reserve hypothesis. Am. J. Psychiat. 166, 50-57.

Linver, M. R., Brooks-Gunn, J., Kohen, D. E., 2002. Family processes as pathways from income to young children's development. Dev. Psychol. 38, 719–734.

Loth, A. K., Drabick, D. A., Leibenluft, E., Hulvershorn, L. A., 2014. Do childhood externalizing disorders predict adult depression? A meta-analysis. J. Abnorm. Child Psychol. 42, 1103-1113.

Malmberg, L. E., Flouri, E., 2011. The comparison and interdependence of maternal and paternal influences on young children's behavior and resilience. J. Clin. Child Adolesc. Psychol. 40, 434-44.

Mathai, J., Anderson, P., Bourne, A., 2002. The Strengths and Difficulties Questionnaire (SDQ) as a screening measure prior to admission to a Child and Adolescent Mental Health Service (CAMHS). Australian e-journal for the advancement of Mental Health 1, 235-246.

Maxwell, S. D., Fineberg, A. M., Drabick, D. A., Murphy, S. K., Ellman, L. M., 2018. Maternal prenatal stress and other developmental risk factors for adolescent depression: Spotlight on sex differences. J. Abnorm. Child Psychol. 46, 381-397.

Moffitt, T. E., Caspi, A., Dickson, N., Silva, P., Stanton, W., 1996. Childhood-onset versus adolescent-onset antisocial conduct problems in males: Natural history from ages 3 to 18 years. Dev. Psychopathol. 8, 399-424.

Morin, A. J., Arens, A. K., Maïano, C., Ciarrochi, J., Tracey, D., Parker, P. D., Craven, R. G., 2017. Reciprocal relationships between teacher ratings of internalizing and externalizing behaviors in adolescents with different levels of cognitive abilities. J. Youth Adolesc. 46, 801-825.

Muris, P., Meesters, C., van den Berg, F., 2003. The strengths and difficulties questionnaire (SDQ): Further evidence for its reliability and validity in a community sample of Dutch children and adolescents. Eur. Child Adolesc. Psychiatry 12, 1-8.

Muthén, L. K., Muthén, B. O. (2009). Mplus. Wiley: Hoboken.

Nivard, M. G., Lubke, G. H., Dolan, C. V., Evans, D. M., Pourcain, B. S., Munafò, M. R., Middeldorp, C. M., 2017. Joint developmental trajectories of internalizing and externalizing disorders between childhood and adolescence. Dev. Psychopathol. 29, 919-928.

O'connor, R. C., Rasmussen, S., Hawton, K., 2009. Predicting deliberate self-harm in adolescents: a six month prospective study. Suicide Life Threat. Behav. 39, 364-375.

Papachristou, E., Flouri, E. (2019). The codevelopment of internalizing symptoms, externalizing symptoms, and cognitive ability across childhood and adolescence. Dev. Psychopathol. 1-15.

Parkes, A., Sweeting, H., Wight, D., 2016. Early childhood precursors and school age correlates of different internalising problem trajectories among young children. J. Abnorm. Child Psychol. 44, 1333-1346.

Patalay, P., Deighton, J., Fonagy, P., Wolpert, M., 2015. The relationship between internalising symptom development and academic attainment in early adolescence. PloS one 10, e0116821.

Patalay, P., Fink, E., Fonagy, P., Deighton, J., 2016. Unpacking the associations between heterogeneous externalising symptom development and academic attainment in middle childhood. Eur. Child Adolesc. Psychiatry 25, 493-500.

Pompili, M., Gibiino, S., Innamorati, M., Serafini, G., Del Casale, A., De Risio, L., . . . Sher, L. 2012. Prolactin and thyroid hormone levels are associated with suicide attempts in psychiatric patients. Psychiatry Res. 200, 389-394.

Pompili, M., Shrivastava, A., Serafini, G., Innamorati, M., Milelli, M., Erbuto, D., . . . Lester, D., 2013. Bereavement after the suicide of a significant other. Indian J. Psychiatry 55, 256-263.

Qualter, P., Brown, S. L., Munn, P., Rotenberg, K. J., 2010. Childhood loneliness as a predictor of adolescent depressive symptoms: an 8-year longitudinal study. Eur. Child Adolesc. Psychiatry 19, 493-501.

Reef, J., van Meurs, I., Verhulst, F. C., van der Ende, J., 2010. Children's problems predict adults' DSM-IV disorders across 24 years. J. Am. Acad. Child Psy. 49, 1117-1124.

Siennick, S. E., Widdowson, A. O., Woessner, M., Feinberg, M. E., 2016. Internalizing symptoms, peer substance use, and substance use initiation. J. Res. Adolesc. 26, 645-657.

Stanford, S., Jones, M. P., Hudson, J. L., 2018. Appreciating complexity in adolescent self-harm risk factors: psychological profiling in a longitudinal community sample. J. Youth Adolesc. 47, 916-931.

StataCorp, L. P. (2011). College Station. TX, USA.

Taylor, R. D., Oberle, E., Durlak, J. A., Weissberg, R. P., 2017. Promoting positive youth development through school-based social and emotional learning interventions: A meta-analysis of follow-up effects. Child Dev. 88, 1156-1171.

Thapar, A., Collishaw, S., Pine, D. S., Thapar, A. K., 2012. Depression in adolescence. Lancet 379, 1056-1067.

Von Stumm, S., Deary, I. J., Kivimäki, M., Jokela, M., Clark, H., Batty, G. D. 2011. Childhood behavior problems and health at midlife: 35-year follow-up of a Scottish birth cohort. J. Child Psycho. Psyc. 52, 992-1001.

Weinstock, M., 2005. The potential influence of maternal stress hormones on development and mental health of the offspring. Brain Behav. Immun. 19, 296–308.

Wichstrøm, L., Belsky, J., Steinsbekk, S., 2017. Homotypic and heterotypic continuity of symptoms of psychiatric disorders from age 4 to 10 years: a dynamic panel model. J. Child Psycho. Psyc. 58, 1239-1247.

Wickrama, K. K., Lee, T. K., O'Neal, C. W., Lorenz, F. O., 2016. Higher-order growth curves and mixture modeling with Mplus: a practical guide. Routledge: New York and London.

Wiesner, M., 2003. A longitudinal latent variable analysis of reciprocal relations between depressive symptoms and delinquency during adolescence. J. Abnorm. Psychol. 112, 633-645.

Yoon, S., 2017. Child maltreatment characteristics as predictors of heterogeneity in internalizing symptom trajectories among children in the child welfare system. Child Abuse Negl. 72, 247-257.

FIGURE CAPTIONS

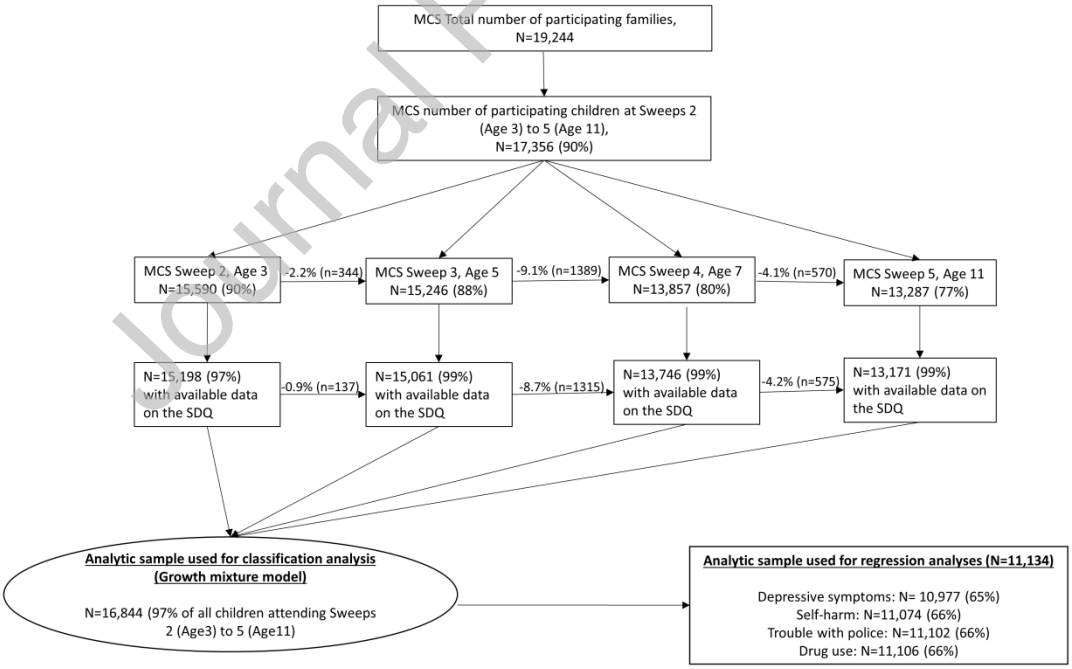

Figure 1. Flow chart of the study

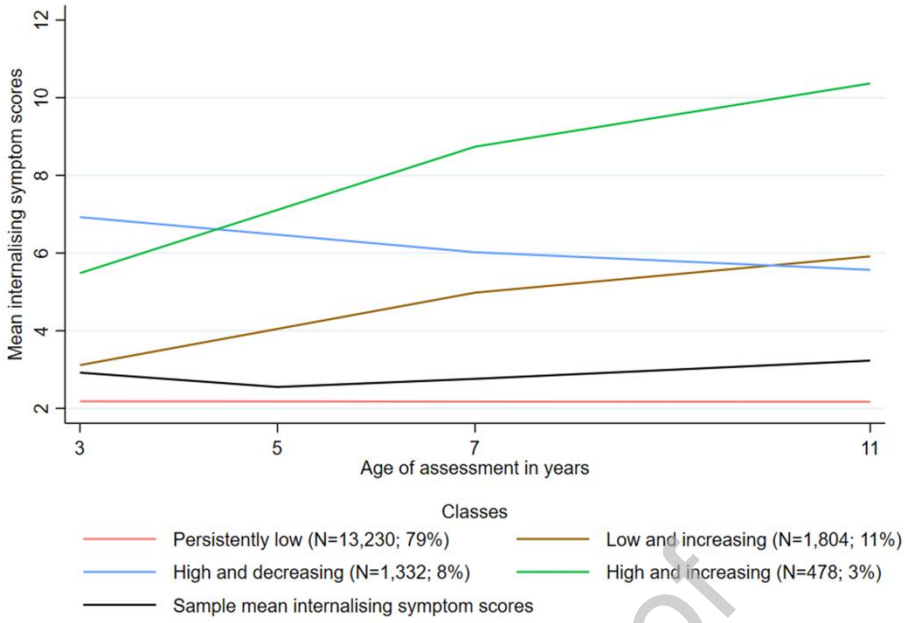

Figure 2. Predicted internalising symptom trajectories by class membership

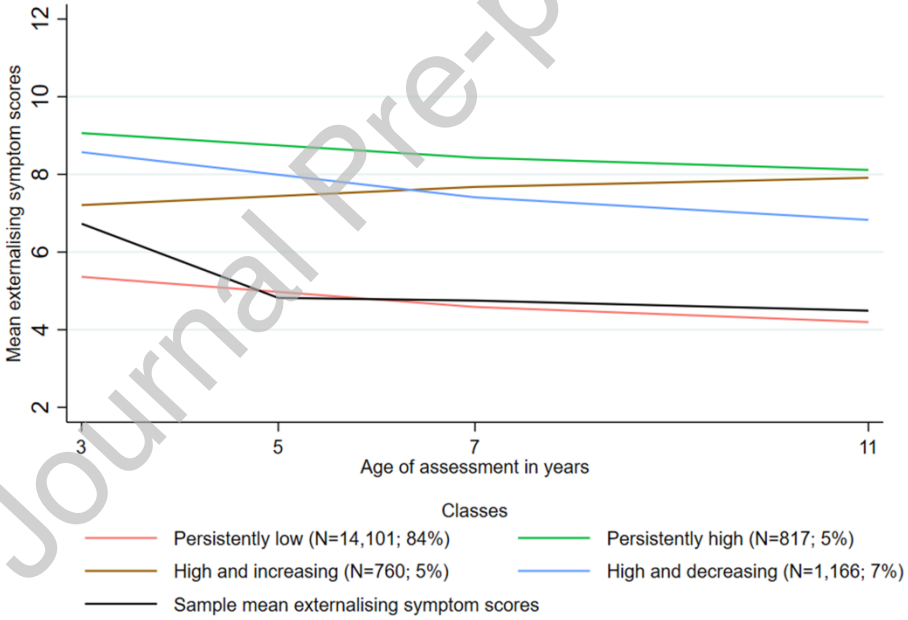

Figure 3. Predicted externalising symptom trajectories by class membership

Table 1. Descriptive statistics of internalising symptoms, externalising symptoms and covariates by class membership

	Internalising symptom trajectories					Externalising symptom trajectories				
	Persistently low (C1)	**Low and increasing (C2)**	**High and decreasing (C3)**	**High and increasing (C4)**	**p-value**	**Persistently low (C1)**	**Persistently high (C2)**	**High and increasing (C3)**	**High and decreasing (C4)**	**p-value**
Categorical Variables										
Sex, *N (% male)*	4,286 *(49)*	674 *(53)*	415 *(52)*	147 *(52)*	0.05	4,597 *(49)*	305 *(56)*	255 *(46)*	365 *(52)*	0.002
Maternal education, *N (% with a university degree)*	1,891 *(22)*	147 *(12)*	72 *(10)*	20 *(7)*	<0.001	1,926 *(21)*	49 *(10)*	73 *(14)*	82 *(13)*	<0.001
Ethnicity, *N (% white)*	7,376 *(84)*	1,052 *(82)*	508 *(63)*	235 *(83)*	<0.001	7,811 *(84)*	417 *(77)*	474 *(85)*	469 *(66)*	<0.001
Continuous Variables										
Internalising symptoms (M±SD)										
Age 3	2.28 (1.90)	3.04 (1.98)	7.90 (2.52)	5.49 (2.75)	<0.001	2.46 (2.13)	4.38 (2.91)	3.77 (2.74)	6.11 (3.14)	<0.001
Age 5	1.78 (1.70)	3.44 (2.21)	7.00 (2.81)	7.29 (3.38)	<0.001	1.83 (1.64)	4.62 (2.51)	3.13 (2.22)	8.66 (2.10)	<0.001
Age 7	1.81 (1.79)	4.89 (2.71)	6.39 (2.91)	9.47 (3.15)	<0.001	1.91 (1.77)	9.65 (2.21)	4.64 (2.65)	5.64 (2.87)	<0.001
Age 11	1.98 (1.73)	7.88 (1.95)	4.64 (2.31)	12.69 (2.23)	<0.001	2.31 (2.07)	6.04 (3.35)	10.81 (2.27)	5.50 (3.24)	<0.001
Externalising symptoms (M±SD)										
Age 3	5.90 (3.38)	8.20 (3.73)	9.99 (4.11)	10.29 (3.94)	<0.001	6.11 (3.52)	9.36 (4.02)	8.04 (3.67)	9.14 (4.21)	<0.001
Age 5	3.94 (2.84)	6.43 (3.56)	7.66 (3.69)	9.36 (3.92)	<0.001	4.14 (2.99)	7.68 (3.88)	6.30 (3.71)	7.24 (3.88)	<0.001
Age 7	3.78 (2.91)	7.16 (3.90)	7.09 (3.67)	10.07 (4.18)	<0.001	4.01 (3.10)	8.33 (4.05)	7.05 (4.29)	6.73 (3.78)	<0.001
Age 11	3.50 (2.79)	7.56 (3.77)	5.77 (3.47)	10.80 (4.11)	<0.001	3.80 (3.06)	7.36 (4.24)	7.89 (4.28)	6.01 (3.75)	<0.001
Socioeconomic disadvantage (M±SD)										
Age 3	0.65 (1.02)	1.10 (1.23)	1.42 (1.32)	1.54 (1.25)	<0.001	0.69 (1.05)	1.26 (1.26)	1.07 (1.22)	1.32 (1.30)	<0.001
Age 5	0.66 (1.01)	1.13 (1.20)	1.44 (1.27)	1.57 (1.22)	<0.001	0.70 (1.03)	1.41 (1.25)	1.09 (1.21)	1.34 (1.26)	<0.001
Age 7	0.61 (0.98)	1.10 (1.18)	1.34 (1.22)	1.48 (1.23)	<0.001	0.65 (1.00)	1.30 (1.21)	1.03 (1.16)	1.28 (1.22)	<0.001
Age 11	0.58 (0.94)	1.02 (1.16)	1.24 (1.21)	1.29 (1.16)	<0.001	0.61 (0.96)	1.13 (1.18)	0.99 (1.15)	1.20 (1.21)	<0.001
Maternal psychologi										

cal distress (M±SD)	2.70	4.28	5.98	6.64	<0.0	2.82	5.47	4.79	5.25	<0.0
Age 3	(3.15)	(4.20)	(4.87)	(5.42)	01	(3.27)	(4.95)	(4.61)	(4.65)	01
Age 5	2.58	4.30	5.81	7.07	<0.0	2.68	5.57	4.45	5.82	<0.0
Age 7	(3.19)	(4.31)	(5.04)	(5.62)	01	(3.30)	(4.90)	(4.41)	(5.13)	01
Age 11	2.52	4.61	5.25	7.38	<0.0	2.63	6.33	4.94	4.81	<0.0
	(3.22)	(4.59)	(4.95)	(5.77)	01	(3.34)	(5.50)	(4.71)	(4.70)	01
	3.22	6.30	5.36	9.17	<0.0	3.43	6.46	7.19	6.16	<0.0
	(3.65)	(5.15)	(5.15)	(6.20)	01	(3.84)	(5.29)	(5.54)	(5.48)	01
Cognitive ability										
(M±SD)	102.71	97.47	92.37	93.71	<0.0	102.32	95.79	97.21	92.97	<0.0
Age 3	(14.48)	(15.71)	(15.24)	(15.18)	01	(14.63)	(15.58)	(14.86)	(16.17)	01
Age 5	102.73	97.19	95.38	94.15	<0.0	102.35	95.94	97.42	95.59	<0.0
Age 7	(14.01)	(15.47)	(15.56)	(17.05)	01	(14.17)	(16.37)	(15.75)	(15.40)	01
Age 11	103.02	95.85	95.52	90.67	<0.0	102.53	95.05	94.68	96.03	<0.0
	(13.93)	(15.38)	(15.40)	(15.80)	01	(14.10)	(15.89)	(15.34)	(16.08)	01
	102.21	97.38	96.46	92.39	<0.0	101.83	97.30	96.02	96.63	<0.0
	(13.80)	(16.17)	(15.64)	(16.35)	01	(13.97)	(16.16)	(17.10)	(15.93)	01

Table 2. Predictive ability [OR (95% CI)] of trajectory class membership for outcomes

	Depression (SMFQ score>=8)		**Self-harm**		**Trouble with police**		**Drug use**	
	Model A[a]	Model B[b]	Model A[a]	Model B[b]	Model A[a]	Model B[b]	Model A[a]	Model B[b]
Class membership based on internalising symptom trajectories at ages 3-11 years								
Persistently low	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Low and increasing	**2.33 (1.85-2.93)**	**2.28 (1.64-2.92)**	**1.90 (1.45-2.50)**	**1.60 (1.12-2.08)**	**1.66 (1.26-2.19)**	0.94 (0.64-1.24)	**1.66 (1.04-2.64)**	**1.71 (1.04-2.31)**
High and decreasing	1.07 (0.78-1.47)	**1.43 (1.03-1.83)**	0.85 (0.58-1.25)	1.27 (0.81-1.73)	**1.58 (1.15-2.18)**	0.85 (0.57-1.13)	**1.89 (1.08-3.32)**	0.81 (0.19-1.43)
High and increasing	**2.28 (1.51-3.44)**	**2.04 (1.02-3.06)**	**2.78 (1.80-4.29)**	1.37 (0.59-2.15)	**2.10 (1.38-3.19)**	0.71 (0.27-1.15)	**2.33 (1.22-4.47)**	0.90 (0.02-1.78)
Class membership based on externalising symptom trajectories at ages 3-11 years								
Persistently low	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Persistently	**1.99**	**2.02**	**1.60**	1.56	**1.97**	1.17	**1.99**	2.07

high	**(1.44-2.75)**	**(1.32-2.72)**	**(1.14-2.24)**	(0.96-2.16)	**(1.33-2.91)**	(0.73-1.61)	**(1.19-3.32)**	(0.91-3.23)
High and increasing	**2.32 (1.71-3.14)**	**2.30 (1.54-3.06)**	**2.21 (1.55-3.14)**	1.42 (0.86-1.98)	**1.42 (1.03-1.96)**	0.74 (0.44-1.04)	1.54 (0.85-2.77)	1.20 (0.32-2.08)
High and decreasing	1.21 (0.91-1.62)	1.26 (0.88-1.64)	1.00 (0.71-1.39)	1.11 (0.67-1.55)	**1.45 (1.03-2.04)**	**0.69 (0.41-0.97)**	1.36 (0.73-2.54)	0.70 (0.10-1.30)

Estimates in boldface are statistically significant (p-values<0.05)

[a]*Unadjusted logistic regression model*

[b]*Logistic regression model adjusted for sex, ethnicity, socioeconomic disadvantage (at ages 3, 7 and 11 years), maternal education, maternal psychological distress (at ages 3, 5, 7, and 11 years) and cognitive ability (at ages 3, 5, 7, and 11 years)*