European Child & Adolescent Psychiatry
https://doi.org/10.1007/s00787-026-03221-1

RESEARCH

Early predictors of the childhood dysregulation profile from ages 5 to 8: evidence from the UK household study

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Received: 5 March 2026 / Accepted: 9 October 2026
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Abstract
Childhood dysregulation profile (DP) involves co-occurring difficulties regulating emotions, behaviour and cognitions, and is associated with adverse outcomes. However, its early predictors are not well understood. We examined child- and family-related predictors of borderline and clinical DP symptoms at ages 5 and 8 years. Participants were 3,467 children from the UK Household Longitudinal Study. Parents reported on early child-related factors (infant regulatory problems, age 3 temperament and screen media use), prenatal exposures (smoking, alcohol) and postnatal family factors (parental psychological distress, stimulating home environment), and DP symptoms at ages 5 and 8 years. Hierarchical logistic regression analyses were conducted to examine predictors of borderline and clinical DP symptoms and their persistence over time. Negative affect at age 3 predicted borderline and clinical DP symptoms at ages 5 and 8 years, as well as symptom persistence. Other child-related predictors included impulsivity, impatience and irregularity, whereas family-related factors were not consistent predictors. These findings highlight the role of child temperament, particularly negative affect, in the development and persistence of dysregulation symptoms. Early identification of temperament-based risk factors may support targeted interventions to strengthen children's self-regulation skills before dysregulation difficulties become more persistent and difficult to modify.

Keywords Childhood dysregulation profile · Childhood psychopathology · Early predictors

Childhood dysregulation profile (DP) is a pattern of co-occurring difficulties in regulating emotions, behaviour and cognition [1], observable from preschool age [2]. It reflects broad difficulties in self-regulation that cut across internalising and externalising domains [3]. DP is typically operationalized using composite scores derived from the emotional problems, conduct problems, and hyperactivity/inattention subscales of widely used instruments such as the Child Behaviour Checklist (CBCL) and the Strengths and Difficulties Questionnaire (SDQ) [4]. Although the SDQ-DP is shorter, it demonstrates clinical associations comparable to those of the CBCL-DP [5]. Conceptualised dimensionally, higher levels of DP are associated with greater impairment and increased psychiatric risk. Accordingly, DP is increasingly regarded as an early transdiagnostic risk marker for persistent self-regulation difficulties and later severe and persistent psychopathology [6, 7]. However, limited evidence exists regarding which early child and environmental factors predict borderline and clinical levels of DP, and which contribute to the persistence of these symptoms across early childhood.

Early life predictors of childhood DP: existing knowledge and gaps in the literature

Two systematic reviews have examined predictors of childhood DP. The first [8] reported associations with genetic heritability, differences in brain functioning, parental substance use, and parenting behaviours. However, its conclusions were constrained by limited search terms, the inclusion of cross-sectional and longitudinal designs, and the small number of studies specifically focused on childhood DP, thereby limiting the generalizability of its findings.

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Published online: 22 October 2026

A second systematic review [9] provided a more comprehensive synthesis of prospective longitudinal studies conducted in non-clinical populations, aiming to identify early emerging risk factors prior to the onset of clinical symptoms. This review highlighted several family-related predictors, including poorer parental mental health, lower parental education, prenatal substance exposure, and broader social adversity. Child-related factors such as male sex, difficult temperament, and early regulatory problems (infant excessive crying, sleeping or feeding problems), were also associated with increased risk of DP.

Despite these findings, important gaps remain. Most studies have examined family and child-related factors in isolation, limiting understanding of their contributions. Much empirical literature is based on cross-sectional or clinically referred samples, limiting insights into the developmental emergence and persistence of childhood DP within the general population. No population-based studies to date have yet investigated early predictors of childhood DP while distinguishing between borderline and clinical levels of severity. Identifying such early risk factors is essential for clarifying developmental pathways and informing early detection and intervention efforts.

Current study

From a developmental psychopathology perspective, early child characteristics such as temperament, regulatory capacity, and impulsivity may represent proximal vulnerabilities for the emergence of DP, whereas family-related factors reflect broader contextual influences that may shape risk trajectories over time [10]. To examine their contributions to the severity and persistence of DP symptoms across early childhood, the current study uses a stepwise modelling approach. First, covariates and child-related factors are examined to evaluate their associations with borderline and clinical levels of DP at ages 5 and 8. Second, family-related factors are added to test whether they provide incremental explanatory value beyond child characteristics. Predictors of DP at a single time point may differ from those underlying persistence, making it important to distinguish factors linked to emergence and continuity of DP at different severity levels.

Because persistent DP is more strongly associated with later psychopathology than transient symptoms [1], the primary focus of the study was to identify predictors of stable borderline and clinical DP symptoms across early childhood. Transitions between borderline and clinical levels were examined in exploratory analyses to provide preliminary insights into factors associated with changes in symptom severity over time.

The aims of the current study were:

1. To identify child- and family-related predictors of borderline and clinical DP symptoms at ages 5 and 8 years.
2. To identify child- and family-related predictors of persistent borderline and clinical DP symptoms from ages 5 to 8 years, and to examine whether these predictors differ from those associated with borderline and clinical level DP symptoms at single time point.

Methods

Participants

The data are from Understanding Society, the UK Household Longitudinal Study (UKHLS) (University of Essex [11]), a stratified clustered sample of approximately 30,000 UK households in 2009, with additional ethnic minority boost samples. UKHLS annually collects extensive information on social, economic, health and behavioural information of all household members. Adult participants (16 or older) are interviewed face-to-face or through a self-completion online survey, while for children under the age of 10 their parents or responsible adults provide information about them [12].

Participants were drawn from the Pregnancy and Early Childhood (PEACH) dataset, a cross-wave dataset derived from Understanding Society that combines parent-reported pregnancy, infancy, child development and family information collected between 2009 and 2024 (University of Essex [11]). Because the PEACH combines information collected across multiple survey waves and developmental stages, data availability varied across measures. Some children did not have data for all variables because they had not yet reached the target age for a particular assessment, were not eligible for specific modules, or because the relevant information was unavailable within a given survey wave. Of the 4,635 children with available PEACH data, 3,467 (74.8%) had outcome data available at both ages 5 and 8 years and were therefore included in the present study. Consequently, exclusions primarily reflected data availability within the longitudinal survey design rather than active withdrawal from the study.

The sample included equal proportions of males (51%) and females (49%), which is representative of the UK population (Office for National Statistics [13]). The mean birth weight of children was 3.41 kg (SD=0.65) and 18.9% were born preterm. Most mothers and fathers were of white ethnicity (81% and 83%) and attained a university degree (63% and 55%). More fathers were employed (81%) than mothers (34%).

Measures

Childhood dysregulation profile (DP)

Mothers reported on their children's emotional and behavioural difficulties at ages 5 and 8 using the SDQ [14]. The SDQ contains 25 items across five subscales: emotional symptoms, conduct problems, hyperactivity/ inattention, peer problems and prosocial behaviour. Responses are rated on a 3-point Likert scale (0 = not true, 1 = somewhat true, 2 = certainly true); higher scores indicating higher difficulties. In line with the previous studies [3, 4], emotional symptoms, conduct problems, and hyperactivity/inattention subscales were summed to create a total childhood DP symptom score at each time point. Consistent with previous research [15], children were classified as showing borderline (> 10 and < 14, 80th percentile) and clinical symptoms (= > 14, 90th percentile) at ages 5 and 8.

Early predictors

A range of child- and family-related early predictors were included (See Table 1 for a brief overview; Supplementary File 1 for detailed explanation)

Covariates

The following covariates were included: child sex, parental ethnicity (non-white British = 0; white British = 1) and educational attainment (no university degree = 0; university degree or other higher degree = 1), preterm birth (born on time = 0; born before expected due date = 1) [18, 19].

Statistical analysis

Weights and missing data strategy

8-year pooled cross-sectional weights were applied to the analyses which incorporates the sampling strata and primary sampling units specified in the survey design, allowing to estimate population-level parameters that are generalizable to the UK population. To account for missing data, multiple imputation (20 times) using chained equations were conducted using the "mi" command in Stata (StataCorp [20]).

Main analyses

Descriptive statistics were used to summarize the characteristics of the sample. Bivariate Pearson correlations were conducted to examine the relationships between dysregulation symptoms at ages 5 and 8, including total dysregulation scores and the three subscales (emotional symptoms, hyperactivity/ inattention, and conduct problems) at each time point.

To address the main hypotheses, first, a multinomial logistic regression analysis was conducted to examine the predictors of borderline and clinical level childhood DP symptoms at ages 5 and 8 following the procedure outlined by Aiken and West [21]. This involved: (a) entering covariates (female sex, parental ethnicity and education level, preterm birth); (b) including child-related predictors (regulatory problems, negative affect, risk-taking, impulsivity, impatience, regularity, and screen media exposure; Model 1); and (c) including family-related predictors (maternal and paternal psychological distress, and maternal substance use during pregnancy; Model 2). The analyses were conducted separately for 5- and 8-years outcomes.

Second, to investigate the predictors of the persistence of DP symptoms from ages 5 to 8, the following groups were created: (1) persistent borderline level DP (0 = no DP symptoms at 5 and 8 years, 1 = borderline level DP symptoms at both 5 and 8 years), and (2) persistent clinical levels (0 = no DP symptoms at 5 and 8 years, 1 = clinical level DP symptoms at both 5 and 8 years). The main analyses were repeated using these variables as outcome measures, following the same analytical procedure.

In line with previous research which has used multiple pairwise comparisons [22], a Bonferroni correction was applied to the four primary outcomes examined (borderline and clinical DP symptoms at ages 5 and 8), resulting in an adjusted significance threshold of $p = .0125$ (= 0.05/4). The same threshold was also applied to the persistence analyses as a conservative criterion for statistical significance.

Exploratory analyses

To explore the predictors of changes in DP symptom severity, exploratory secondary analyses were conducted focusing on children whose DP symptom levels transitioned between ages 5 and 8 from borderline at 5 years to clinical at 8 years, and from clinical at 5 years to borderline at 8 years, Multinomial logistic regression models were estimated using transition status (i.e., borderline to clinical, and clinical to borderline) as outcome variables, following the same analytical procedure as the primary analyses, with covariates entered in the first step, child-related predictors in the second step, and family-related predictors in the final step. These analyses were intended to provide preliminary insights into factors associated with changes in DP symptom severity over time.

Table 1 Description of included predictors, measurement, and assessment time points

Predictors	Measurement	Assessment Time Point
Child-related predictors		
Regulatory Problems	Sum score of two items per regulatory problem [16]. *Excessive crying* Difficult to soothe (0 = very easy to about average; 1 = somewhat difficult and very difficult) Cries a lot or most of the time (0 = some of the time to rarely if at all; 1 = a lot of the time and most of the time) *Sleeping problems* Long settling duration (0 = 30 min or less; 1 = more than 30 min) Night waking frequency (based on 90th percentile, 0 = 3 times or less; 1 = 4 times or more). *Feeding problems* Food refusal (0 = not true; 1 = somewhat true and certainly true) No appetite (0 = not true; 1 = somewhat true and certainly true).	Within one year after birth
Temperament: negative affect	Sum score of three items: Usually unhappy, irritated, and difficult to comfort (1 = agree completely; 4 = disagree completely).	3 years
Risk aversion	Unwillingness to take risks (1 = completely unwilling to take risks; 7 = completely willing to take risks).	3 years
Impatience	Generally impatient (1 = very impatient; 7 = very patient).	3 years
Impulsivity	Generally impulsive (1 = not at all impulsive; 7 = very impulsive).	3 years
Regularity	Sum score of two items: Regularity of sleeping and eating times (No, never or almost never; yes, sometimes; yes, usually; yes, always).	3 years
Screen media exposure	Duration of screen media exposure (hours) on a school day and weekend.	3 years
Family-related predictors		
Psychological distress	General Health Questionnaire (GHQ) [17]	Within one year after birth
Stimulating home environment	Parent reads to child (not at all; less often; once or twice a month; once or twice a week; several times a week; everyday).	3 years
Substance use during pregnancy		
Smoking during pregnancy	Smoking at all during pregnancy (Yes/no).	Within one year after birth
Alcohol use during pregnancy	Frequency of alcohol use (every day, 5–6 times per week, 3–4 times per week, 1–2 times per week, 1–2 times per month, less than once a month, never).	Within one year after birth

Interpretation

Results are presented as odds ratios (OR) and 95% confidence intervals (CI) for membership in each category relative to the reference category. An OR indicates the change in the odds of belonging to a specific childhood DP symptom category associated with a one-unit increase in each predictor, compared with the reference category. An OR greater than 1 indicates higher odds of membership in that category, whereas an OR less than 1 indicates lower odds. The 95% CI represents the range within which the true OR is expected lie within 95% confidence; associations are considered statistically significant when CI does not include 1. Given the likelihood of finding significant *P* values for small effects in large sample sizes, results are interpreted with emphasis on both effect size and the corresponding 95% CIs [23].

Results

Supplementary Table 1 shows bivariate correlations among childhood dysregulation profile symptoms at 5 and 8 years. At both time points, similar proportions of children exhibited borderline level DP symptoms (10.8% at age 5 and 13.8% at age 8) and clinical level DP symptoms (9.8% at age 5 and 10.2% at age 8) (Fig. 1).

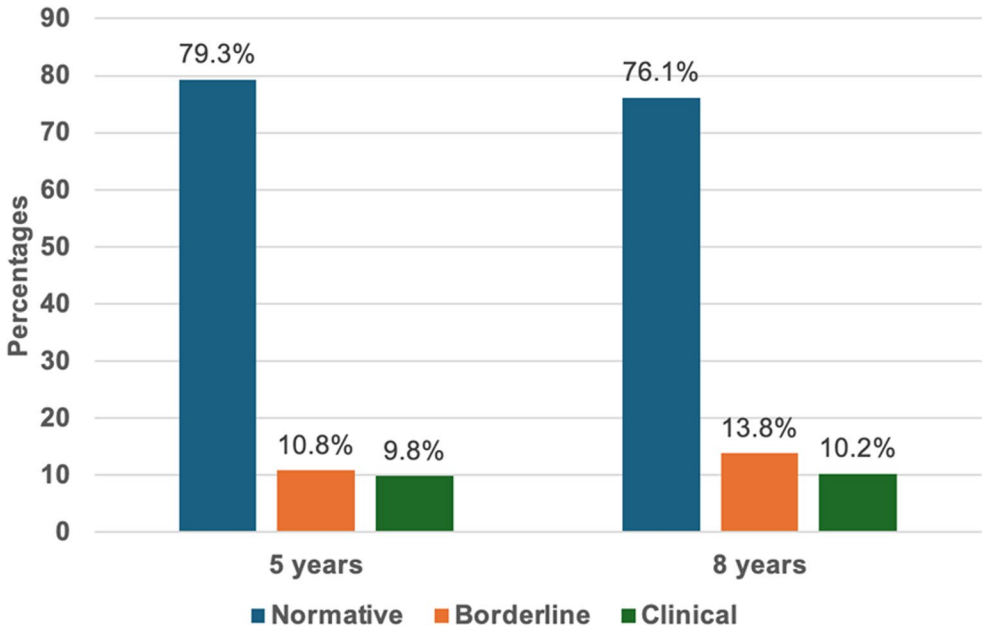


Fig. 1 Percentages of normative, borderline and clinical DP symptoms at 5 and 8 years

Most children (84.1%) showed no DP symptoms at either age, 3.8% had persistent borderline and 5.8% had persistent clinical level DP symptoms. Similar percentages of children transitioned between severity levels over time: 3.1% moved from borderline symptoms at age 5 to clinical symptoms at age 8, 3.2% moved from clinical symptoms at age 5 to borderline symptoms at age 8.

Associations between early child and family-related predictors and childhood dysregulation symptoms at 5 and 8 years

Borderline DP symptoms at 5 years

In Model 1, feeding problems (OR = 1.76, 95% CI = 1.03; 3.01, p=.03), negative affect at age 3 (OR = 1.21, 95% CI = 1.11; 1.31, p<.001), impulsivity (OR = 1.26, 95% CI = 1.09; 1.46, p<.001) and impatience (OR = 1.18, 95% CI = 1.05; 1.33, p<.001) at age 3 were associated with an increased the likelihood of borderline level DP symptoms at age 5 (Supplementary Table 2; Fig. 2). Following Bonferroni correction, only negative affect, impulsivity and impatience remained significant.

After adjusting for family-related variables in Model 2, negative affect (OR = 1.24, 95% CI = 1.14; 1.36, p<.001), impulsivity (OR = 1.26, 95% CI = 1.09; 1.46, p=.001), and impatience at age 3 (OR = 1.21, 95% CI = 1.07; 1.37, p=.003) remained significant predictors. Only negative affect and impulsivity remained significant after correction for multiple comparisons. Father's postnatal psychological distress was also associated with an increased likelihood of borderline DP symptoms at age 5 (OR = 1.04, 95% CI = 1.00; 1.09, p=.04). However, this association did not remain significant after correction for multiple comparisons.

Clinical DP symptoms at 5 years

In Model 1, sleeping problems during the first year (OR = 1.67, 95% CI = 1.09; 2.56, p=.01), negative affect (OR = 1.32, 95% CI = 1.20; 1.46, p<.001), impulsivity (OR = 1.49, 95% CI = 1.27; 1.76, p<.001), impatience at age 3 (OR = 1.49, 95% CI = 1.29; 1.72, p<.001) were associated with an increased likelihood of clinical DP symptoms at age 5. Regularity at age 3 was associated with a decreased likelihood of clinical level DP symptoms at age 5 (OR=0.79, 95% CI=0.66; 0.93, p=.006) (Supplementary Table 2; Fig. 2). All survived Bonferroni corrections.

After adjusting for family-related variables in Model 2, negative affect (OR = 1.28, 95% CI = 1.17; 1.41, p<.001), impulsivity (OR = 1.57, 95% CI = 1.33; 1.84, p<.001), and impatience (OR = 1.44, 95% CI = 1.24; 1.68, p<.003) at age 3 remained significant predictors. These associations were robust to correction for multiple comparisons.

Additional predictors of clinical DP symptoms at 5 years included excessive crying during the first year (OR = 1.81, 95% CI = 1.02; 3.22, p=.04). A stimulating home environment at age 3 (OR=0.76, 95% CI=0.59; 0.98, p=.03) decreased the likelihood of clinical DP predictors.

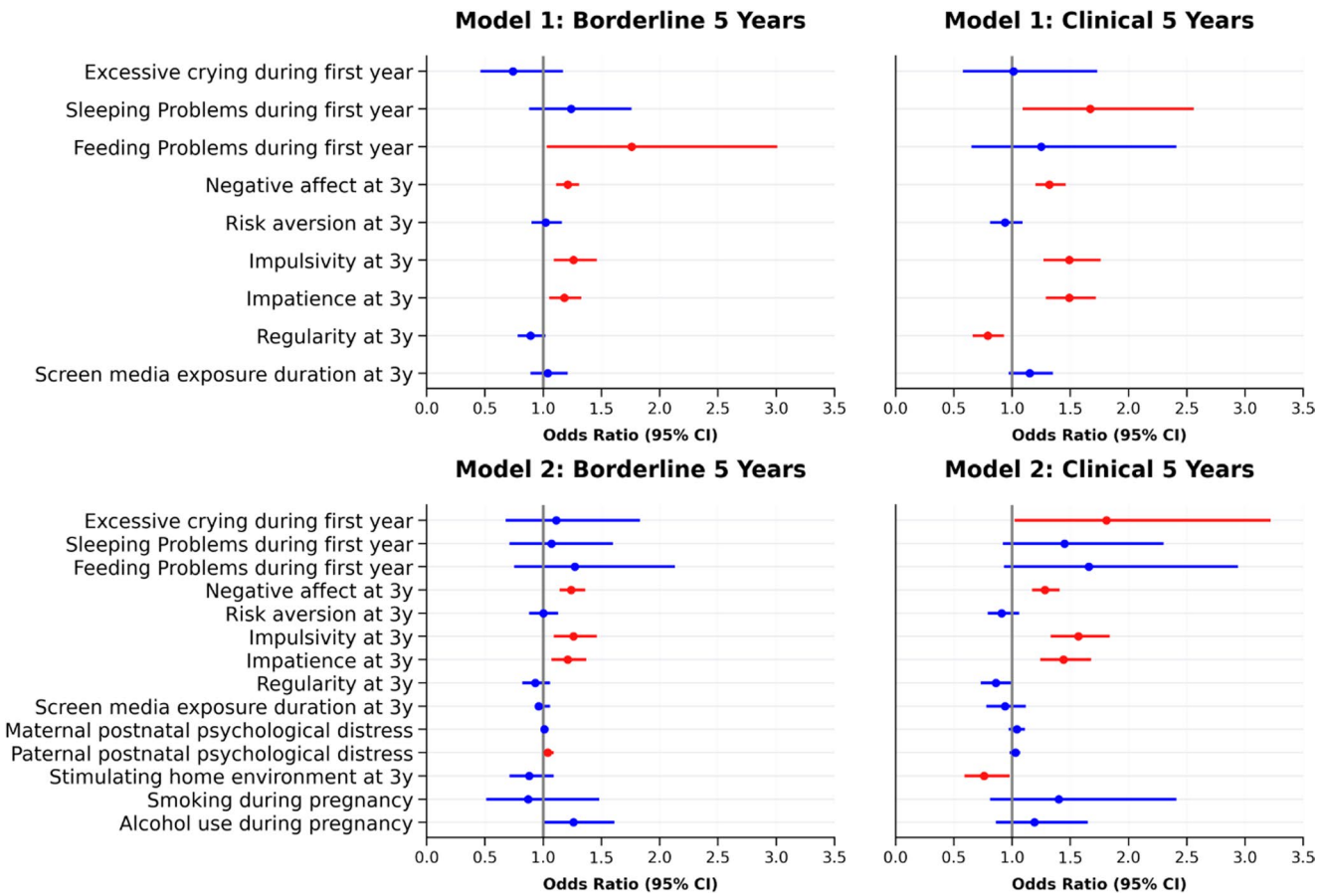


Fig. 2 Associations between early child and family-related predictors and borderline and clinical DP symptoms at 5 years. Odds ratios (ORs) and 95% confidence intervals (CIs) are shown for each predictor. Red markers indicate statistically significant associations ($p < .05$), and blue markers indicate non-significant associations. Asterisks (*) denote associations that remained statistically significant following Bonferroni correction for multiple comparisons

However, these associations did not remain significant after correction for multiple comparisons.

Borderline DP symptoms at 8 years

In Model 1, negative affect at age 3 (OR = 1.13, 95% CI = 1.06; 1.21, p<.001) was associated with borderline-level DP symptoms. Regularity (OR=0.90, 95% CI=0.82; 0.99, p=.04) was associated with a decrease in borderline-level DP symptoms. Following Bonferroni correction, only the association with negative affect remained significant. After adjusting for family-related in Model 2, both negative affect (OR = 1.19, 95% CI = 1.06; 1.33, p=.003) and regularity (OR=0.82, 95% CI=0.68; 0.99, p=.04) at age 3 remained significant predictors. However, after Bonferroni correction, only negative remained significant. No other significant associations were observed (Fig. 3).

Clinical DP symptoms at 8 years

In Model 1, excessive crying during the first year (OR = 1.71, 95% CI = 1.13; 2.59, p=.01), negative affect at age 3 (OR = 1.12, 95% CI = 1.05; 1.21, p=.001), impulsivity (OR = 1.33, 95% CI = 1.19; 1.48, p<.001) and impatience (OR = 1.16, 95% CI = 1.06; 1.28, p=.001) were associated with an increased likelihood of clinical-level DP symptoms at age 3 (Fig. 3). All associations remained significant following Bonferroni corrections.

After adjusting for family-related variables in Model 2, only negative affect (OR = 1.21, 95% CI = 1.07; 1.38, p=.002) and impulsivity at age 3 (OR = 1.50, 95% CI = 1.22; 1.86, p<.001) remained significant predictors. These associations were robust to correction for multiple comparisons.

Additionally, maternal postnatal psychological distress was associated with an increased likelihood of clinical DP symptoms at 8 years (OR = 1.08, 95% CI = 1.01; 1.16,

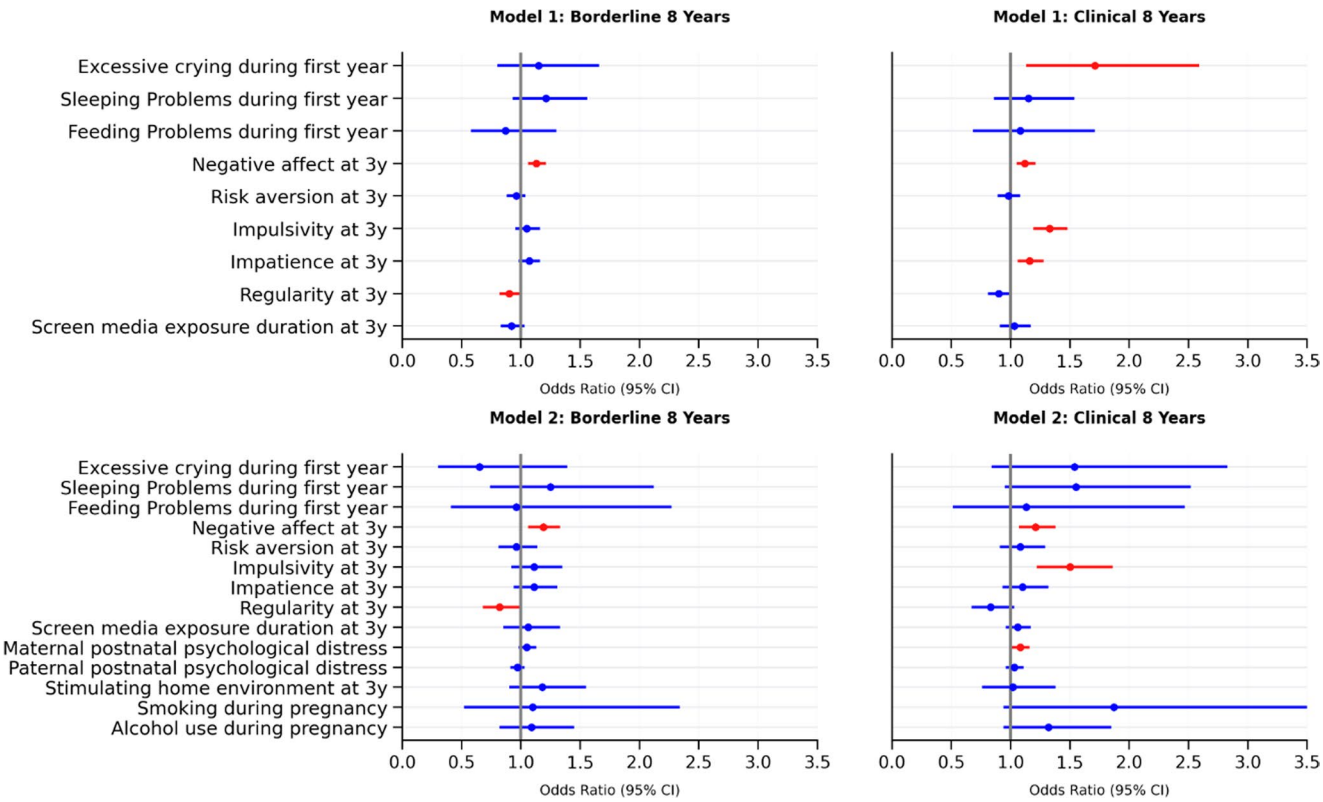


Fig. 3 Associations between early child and family-related predictors and borderline andclinical DP symptoms at 8 years. Odds ratios (ORs) and 95% confidence intervals (CIs) are shown for each predictor. Red markers indicate statistically significant associations (p < .05), and blue markers indicate non-significant associations. Asterisks (*) denote associations that remained statistically significant following Bonferroni correction for multiple comparisons

p=.03). However, this association did not remain significant after correction for multiple comparisons.

Associations between early child and family-related predictors and the course of childhood dysregulation symptoms from 5 to 8 years

Persistent borderline DP symptoms

In Model 1, significant predictors of persistent borderline level DP symptoms were feeding problems during the first year (OR = 2.99, 95% CI = 1.22; 7.35, p=.01) and negative affect at 3 years (OR = 1.34, 95% CI = 1.16; 1.55, p<.001). Following Bonferroni correction, feeding problems and negative affect remained significant. After adjusted for family-related variables in Model 2, both feeding problems (OR = 2.88, 95% CI = 1.17; 7.11, p=.02) and negative affect at 3 years (OR = 1.34, 95% CI = 1.16; 1.54, p<.001) remained significant predictors. In contrast, excessive crying during the first year was associated with reduced odds of persistent borderline-level DP symptoms between ages 5 and 8 (OR= 0.39, 95% CI= 0.15; 0.99, p=.047) (Supplementary Table 3; Fig. 4). However, only negative affect at 3 years remained significant after Bonferroni correction. No other significant associations were observed

Persistent clinical DP symptoms

In Model 1, negative affect at age 3 (OR = 1.35, 95% CI = 1.18; 1.54, *p*<.001), impulsivity (OR = 1.60, 95% CI = 1.27; 2.02, *p*<.001) and impatience (OR = 1.35, 95% CI = 1.09; 1.67, *p*=.005) were associated with an increased likelihood of persistent clinical-level DP symptoms. In contrast, regularity at age 3 was associated with a decreased likelihood (OR=0.66, 95% CI=0.52; 0.84, *p*=.001). All four associations remained significant following Bonferroni corrections.

After adjusting for family-related variables in Model 2, the effects of negative affect (OR = 1.31, 95% CI = 1.14; 1.51, *p*<.001), impulsivity (OR = 1.57, 95% CI = 1.23; 1.99, *p*<.001), impatience (OR = 1.36, 95% CI = 1.10; 1.68, *p*=.004) and regularity (OR=0.68, 95% CI=0.54; 0.86, *p*=.002) at age 3 remained significant (Supplementary Table 3; Fig. 4). All associations were robust to correction for multiple comparisons.

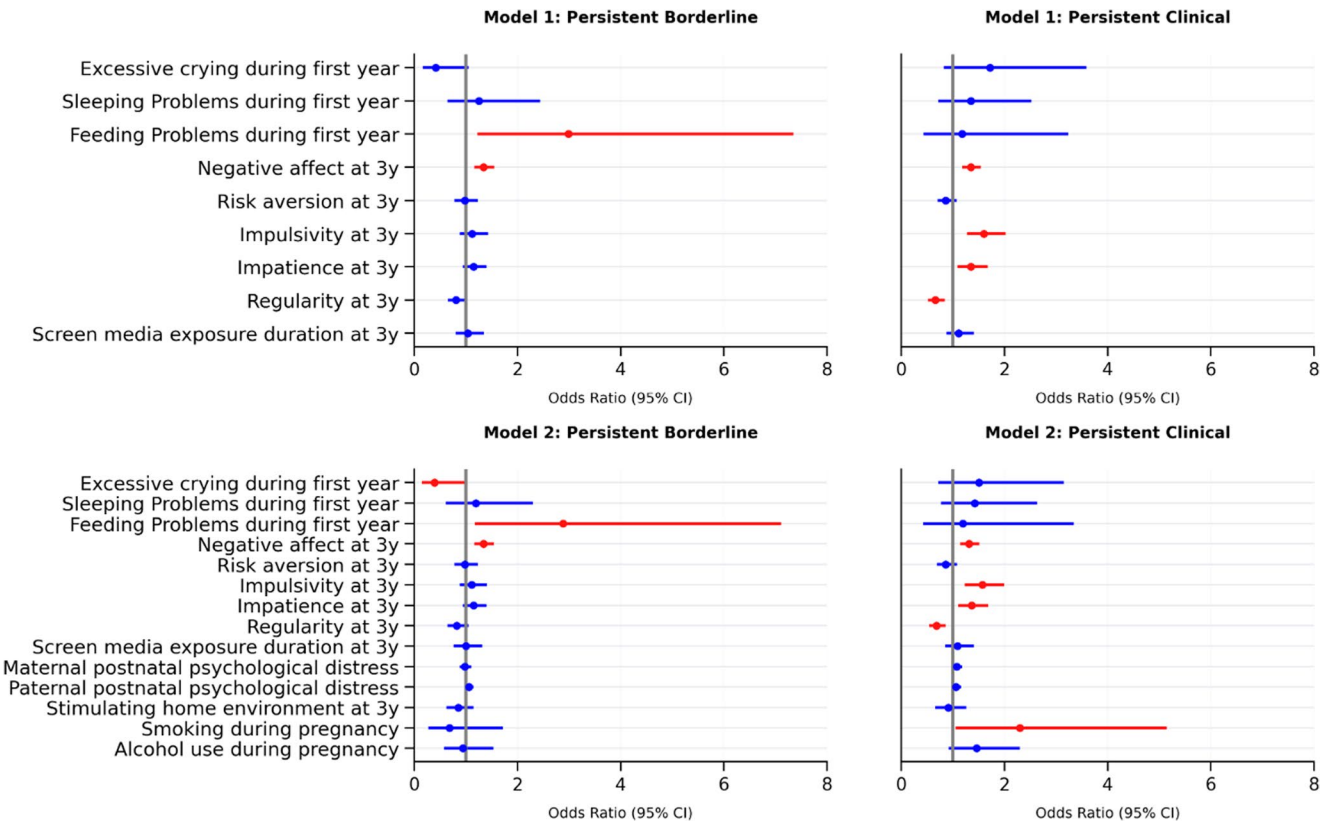


Fig. 4 Associations between early child and family-related predictors and the course of persistent borderline and clinical DP symptoms from 5 to 8 years. Odds ratios (ORs) and 95% confidence intervals (CIs) are shown for each predictor. Red markers indicate statistically significant associations ($p < .05$), and blue markers indicate non-significant associations. Asterisks (*) denote associations that remained statistically significant following Bonferroni correction for multiple comparisons

Maternal smoking during pregnancy was also associated with an increased likelihood of persistent clinical-level DP symptoms (OR = 2.30, 95% CI = 1.05; 5.14, p = .036). However, this association did not remain significant after correction for multiple comparisons.

Exploratory Analyses

Infant sleeping problems and negative affect, impulsivity and impatience at 3 years were associated with transitions between borderline and clinical level DP symptoms from ages 5 to 8, in both directions (all $p <= 0.03$) (Supplementary Table 4).

Discussion

This study examined early child- and family-related predictors of borderline and clinical DP symptoms at ages 5 and 8 years, as well as predictors of symptom persistence across early childhood. Several child-related characteristics emerged as consistent predictors of childhood DP. In particular, negative affect at age 3 was the strongest and most robust predictor of both borderline and clinical DP symptoms at ages 5 and 8 and of persistent DP symptoms over time, even after adjusting for family-related factors and correction for multiple comparisons. Impulsivity, impatience, and low regularity also showed consistent associations with DP, particularly at the clinical level and for symptom persistence. In contrast, family-related factors showed limited and comparatively weak associations with DP symptoms and did not consistently predict their persistence.

Clinically significant DP symptoms typically emerged by age 5 and showed substantial stability across early childhood, with relatively few children developing new clinical-level symptoms by age 8. This pattern indicates that childhood DP is largely an early-emerging and persistent condition [24, 25]. This suggests that the preschool period may represent a critical window for detection and intervention, as difficulties present by age 5 are unlikely to resolve spontaneously for many children.

Negative affect at age 3 appears to reflect a temperamental vulnerability that contributes to the emergence of dysregulation, which in turn may increase risk for later psychopathology. This finding aligns with prior research identifying negative affect as a key risk factor for childhood DP [19, 26,

27], and with broader evidence linking negative affect to a range of later mental health difficulties [28]. Longitudinal studies spanning from early childhood through adolescence and adulthood are needed to clarify whether DP mediates the pathway from early negative affect to later mental health outcomes.

Alternatively, the association between negative affect and DP may reflect shared genetic and environmental vulnerabilities. Negative affect, internalising and externalising, and cognitive regulation difficulties have been shown to share genetic underpinnings as well as environmental influences such as low parental warmth and stress [29, 30]. Increasingly, research suggests that genetic factors confer susceptibility rather than direct causation, with their effects unfolding through interactions with environmental contexts [29, 31]. Thus, early negative affect may signal a biologically rooted sensitivity that increases the likelihood of DP symptoms in the presence of certain environmental factors.

In addition to negative affect, impulsivity, impatience and regularity at 3 years were consistent predictors of DP symptoms. Impulsivity predicted clinical DP at 5 and 8 years as well as the persistence of symptoms, suggesting that early difficulties with behavioural control may play a central role in more severe and enduring dysregulation. This finding is consistent with research documenting comorbidity between DP and ADHD [7, 32]. It raises the possibility that early impulsivity may mark a shared developmental pathway linking DP and later neurodevelopmental difficulties, though further research is needed to disentangle these trajectories.

The results also indicate that dysregulation problems in childhood may be detectable from infancy, with excessive crying, feeding problems and sleeping difficulties emerging early indicators. Excessive crying in the first year predicted clinical DP at age 5 after adjusting for family-related factors, although its association at age 8 weakened when family factors were considered. Feeding problems were associated with persistent borderline-level DP symptoms. In addition, excessive crying during the first year was also associated with a lower likelihood of persistent borderline-level DP symptoms between ages 5 and 8 years. This finding was unexpected [33] and should be interpreted cautiously, given its marginal significance and failure to survive correction for multiple comparisons. One possible explanation is that crying was assessed broadly across the first year of life, potentially capturing heterogenous developmental patterns rather than persistent regulatory difficulties. Sleeping problems were related to changes in levels of DP symptoms. These findings suggest that, early regulatory difficulties may represent some of the earliest manifestations of later dysregulation [23]. This is consistent with meta-analytic evidence identifying early irritability as a transdiagnostic risk factor for later psychopathology [34]. However, their longer-term impact may depend partly on subsequent developmental and environmental factors.

Family-related factors showed limited independent associations with DP symptoms after accounting for child temperament and infant regulatory problems. Although paternal psychological distress predicted borderline DP at age 5 and maternal distress predicted clinical DP at age 8, these effects were small, and did not predict persistence symptoms, and did not remain significant after correction for multiple comparisons. This was unexpected given extensive prior evidence highlighting the role of family-related factors in childhood DP [19, 25, 35].

The limited family effects observed here may reflect methodological differences, statistical adjustment for child temperament, particularly negative affect, or bidirectional parent-child processes. Parenting a child who is often distressed, irritable or difficult to soothe may increase parental stress and psychological difficulties, and may shape parental perceptions of the child [36]. Such reciprocal influences are consistent with transactional models of development, suggesting child temperament and family environment likely influence one another over time [16, 37, 38].

One explanation for the limited direct effects of family-related factors comes from the exploratory analyses examining transitions in DP symptom severity over time. An interesting finding from these analyses was that sleeping problems, negative affect, impulsivity and impatience consistently predicted transitions in childhood DP symptom severity over time. Specifically, these characteristics were associated with both an increase in symptom severity, from borderline-level DP symptoms at age 5 to clinical-level symptoms at age 8, and a decrease in severity, from clinical-level symptoms at age 5 to borderline-level symptoms at age 8. At first glance, these bidirectional associations may appear contradictory. However, they suggest that these child characteristics may not function as direct determinants of developmental trajectories but rather as markers of underlying vulnerability to dysregulation. Whether this vulnerability becomes amplified or attenuated over time may depend on broader family and environmental context in which development unfolds. This interpretation is consistent with transactional models of development and the circular feedback processes described by Caro-Cañizares et al., [8], whereby child characteristics and environmental influences continuously shape one another over time. For example, a child with high levels of negative affect or poor self-regulation may show reductions in DP symptoms when these difficulties are met with sensitive parenting, emotional support and adaptive family functioning [24, 39]. In contrast, the same child characteristics may contribute to escalating symptom severity when combined with adverse experiences such as

harsh parenting, family stress or poor parental mental health [40–42]. Thus, the same early temperamental vulnerabilities may be associated with both improvement and deterioration in DP symptoms because their developmental impact is contingent on the environmental context.

Notably, postnatal maternal distress did not follow the same pattern as the child-related predictors. Whereas sleeping problems, negative affect, impulsivity and impatience were associated with both worsening and improving symptom trajectories, maternal distress was only associated with worsening outcomes. This finding suggests that parental psychological distress may act less as a marker of underlying vulnerability and more as a contextual risk factor that increases the likelihood that early dysregulatory tendencies become entrenched over time. However, given the observational nature of the study, bidirectional influences between child difficulties and parental distress cannot be ruled out.

Clinical implications

The present findings provide several practical implications for clinicians working with young children and families. First, clinicians can incorporate early assessment of temperament, particularly negative affect and emotional reactivity, into routine developmental surveillance. Brief parent-report measures or structured observations of how a child responds to frustration or distress may help identify those at risk for persistent dysregulation. Impulsivity and impatience at age 3 emerged as a robust predictor of both borderline and clinical DP symptoms at ages 5 and 8, as well as their persistence over time [43]. Children who are frequently irritable, intensely reactive, and slow to calm may warrant closer monitoring, even in the absence of marked family adversity.

Second, clinicians can use this information to guide the timing of intervention. Because clinically significant DP symptoms tend to emerge early and remain stable, waiting for difficulties to resolve with age may not be appropriate for children showing persistent emotional and behavioural dysregulation in preschool years. Instead, early referral to preventive or early intervention services may be justified when high negative affect co-occurs with emerging regulation difficulties [44, 45].

Third, intervention planning can prioritize strengthening emotion regulation capacities [46]. Clinicians may focus on helping children build skills in identifying emotions, tolerating frustration, and using adaptive calming strategies. Parent-focused components can emphasize scaffolding, co-regulation, consistent responses to emotional outbursts, and structured support for behavioural control. Even when family-related predictors are not independently robust, involving caregivers remains essential, as they are central to shaping children's regulatory environment.

Fourth, clinicians can use information about early temperament and sleep difficulties to inform psychoeducation. Explaining to parents that high negative affect may reflect a temperamental vulnerability, rather than intentional misbehaviour, can help reduce blame and increase engagement with supportive strategies [47]. Clinicians can also help parents understand that sleep difficulties and self-regulatory challenges often co-occur in early childhood and may reinforce one another over time. Promoting healthy sleep routines may therefore represent one pathway for supporting children's emerging self-regulatory capacities [48]. This framing may encourage parents to adopt proactive regulation-building approaches, rather than relying primarily on reactive disciplinary responses.

Finally, screening systems in primary care, health visiting, and early education settings could integrate questions about persistent irritability, impulsivity, and difficulties with calming by age 3. Identifying at-risk children before school entry creates an opportunity to intervene during a developmentally sensitive period, when regulatory systems are still maturing and more amenable to change. Clinicians can use early temperamental markers not only to identify risk, but to act preventively and potentially alter longer-term developmental trajectories.

Strengths and Limitations

Strengths of this study include the use of a large longitudinal UK sample and inclusion of multiple child and family factors. Several limitations should be considered. First, reliance on parent reports may have introduced reporter bias and social desirability effects. Second, although exclusions primarily reflected age-specific data availability rather than active participant attrition, analyses were restricted to children with outcome data available at both ages 5 and 8 years. Because formal comparisons between included and excluded participants were not conducted, it remains unclear whether systematic differences existed between these groups. Consequently, the representativeness of the final analytical sample and the potential for selection bias cannot be fully evaluated. Third, because UKHLS data are collected in waves rather than at fixed developmental ages, some measures were obtained at slightly different ages across participants. For example, infant crying was assessed at varying points during the first year of life, despite rapid developmental changes in crying across infancy [49]. Similarly, prenatal substance use was assessed as a broad measure of exposure during pregnancy and did not capture the timing of exposure across pregnancy trimesters, which may be important given evidence of timing specific effects on development [50, 51]. Additionally, several predictors yielded relatively wide confidence intervals, particularly in analyses examining

persistent and changing DP symptom trajectories, where subgroup sizes were smaller. Wide confidence intervals indicate greater uncertainty and reduced precision around effect estimates, suggesting that some associations should be interpreted cautiously. This was especially evident for less common exposures and outcomes, where the number of children within specific trajectory groups was limited. Therefore, although the direction of effects was generally consistent, the magnitude of some associations may be less stable and should be replicated in independent samples. Finally, some constructs were assessed using single-item measures, which may have reduced reliability.

Recommendations for future research

First, future studies should examine bidirectional relationships between child and family factors to better understand the direction of effects. For example, it remains unclear whether sleep difficulties contribute to the development of DP symptoms or whether dysregulation itself disrupts sleep. Longitudinal designs with repeated assessments would help clarify these transactional processes. Second, additional assessments of DP symptoms from earlier childhood and beyond age 8 years would allow researchers to better characterise developmental trajectories and identify factors associated with the onset, persistence, remission, and escalation of dysregulation symptoms across childhood and adolescence. Third, future research should further investigate the role of sleep in the development of childhood DP, including examining potential mediating and moderating mechanisms linking early temperament, environmental influences, and later dysregulation symptoms. Finally, a wider range of family and environmental factors should be examined. Although family-related variables showed limited effects in the current study, this may reflect the measures available within the dataset rather than the absence of family influences. Future research should consider parenting behaviours, parent-child relationship quality, family conflict, parental emotional regulation, parental neurodiversity, exposure to trauma and broader contextual stressors, all of which may interact with child temperament to shape developmental trajectories of DP symptoms. Such factors may help explain why similar child characteristics were associated with both improving and worsening symptom trajectories in the present study. In particular, future studies should also investigate whether family and environmental factors moderate the associations between early temperamental vulnerabilities and later DP symptoms, thereby identifying the conditions under which risk is either exacerbated or buffered over time.

Conclusion

The present findings suggest that early temperamental characteristics, particularly negative affect, are among the most robust predictors of borderline and clinical DP symptoms and their persistence across early childhood. Clinically significant DP symptoms appear to emerge early and remain relatively stable, highlighting the preschool years as a potentially important window for identification and intervention. Although, family-related factors showed limited independent effects after accounting for child characteristics, the findings are consistent with transactional models in which child vulnerabilities and environmental influences shape developmental trajectories over time, Together, these results underscore the importance of early identification of temperament-based vulnerabilities and support efforts to strengthen self-regulation before dysregulation difficulties become more persistent and entrenched.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s00787-026-03221-1.

Author contributions Ms Vitkauskaite carried out the analyses, drafted and revised the manuscript. Dr Bilgin conceptualized and designed the study, carried out the analyses, supervised the study, drafted and revised the manuscript. Both authors approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

Funding The authors received no funding related to this study.

Data availability No datasets were generated or analysed during the current study.

Declarations

Competing interests The authors declare no competing interests.

Short Title Predictors of Childhood Dysregulation Profile.

Financial Disclosure The authors have no financial relationships relevant to this article to disclose.

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