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ORIGINAL CONTRIBUTION

A prospective longitudinal investigation of the (dis)continuity of mental health difficulties between mid- to late-childhood and the predictive role of familial factors

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Abstract Understanding individual variation in the continuity of youth mental health difficulties is critical for identifying the factors that promote recovery or chronicity. This study establishes the proportion of children showing psychopathology at 9 years, whose pathology had either remitted or persisted at 13. It describes the socio-demographic and clinical profiles of these groups, and examines the factors in 9-year-olds' familial environments that predict longitudinal remission vs. persistence of psychopathology. The study utilised data from a prospective longitudinal study of 8568 Irish children. Child psychopathology was assessed using the Strengths and Difficulties Questionnaire (SDQ). Analysis established the rates of continuity of SDQ classifications between 9 and 13 years. Analysis also investigated the familial factors that predicted the remission vs. persistence of psychopathological symptoms, controlling for socio-demographic and child factors. Average SDQ scores improved between the ages of 9 and 13, $F(1, 7292) = 276.52$, $p < 0.001$, $\eta_p^2 = 0.04$. Of children classified Abnormal aged 9, 41.1% remained so classified at 13, 21.4% were reclassified Borderline, and 37.6% Normal. Demographic and child risk factors for persistence of pathology were maleness ($\beta = -1.00$, $p = 0.001$, CI = 0.20–0.67), one-carer households ($\beta = -0.71$, $p = 0.04$, CI = 0.25–0.97), poor physical health ($\beta = -0.64$, $p = 0.03$, CI = 0.30–0.92), and low cognitive ability ($\beta = 0.61$, $p = 0.002$, CI = 1.26–2.70). Controlling for these factors, the only familial variable at 9 years that predicted subsequent pathological persistence was caregiver depression ($\beta = -0.07$, $p = 0.03$, CI = 0.87–0.99). The analysis highlights substantial rates of psychopathological discontinuity in a community sample and identifies the children most at risk of chronic mental health problems. These results will inform the targeting of early interventions and distribution of clinical resources.

Keywords Child psychopathology · Persistence · Remission · Strengths and Difficulties Questionnaire · Longitudinal · Parental depression

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Introduction

Longitudinal studies demonstrate that mental health difficulties in childhood are a major predictor of later psychopathology. For instance, the Christchurch birth cohort study found that attentional difficulties aged 8 predicted a range of negative psychosocial outcomes at 18 [1]; early anxiety/withdrawal increased the risk of later anxiety and depression [2, 3]; and children exhibiting disruptive behaviour at 7–9 were 16 times more likely to develop conduct disorder in adolescence [4]. The temporal continuity of mental health difficulties is such that in the empirical literature, prior pathology is the strongest known predictor of later pathology [5–7].

However, the high average correlations between mental health measures across time belie a considerable amount of individual variability. One longitudinal study reported that

almost 60% of those with a DSM disorder aged 11 did not show pathology aged 15, while over 80% of those diagnosable at 15 did not have a history of disorder at 11 [8]. Another study found up to half of children who received a diagnosis between the ages of 2–5 failed to meet criteria for any psychiatric disorder 4 years later [9]. Levels of stability vary across diagnostic categories, apparently lowest for conduct and anxiety disorders and highest for autistic spectrum disorder (ASD) and attention deficit hyperactivity disorder (ADHD) [10]. However, even these most reliable classifications show considerable temporal variability. Kleinman et al. report that one-fifth of children diagnosed with ASD at initial consultation moved off the spectrum within 2–4 years [11]. Recent research indicates that just 21.9% of children who met diagnostic criteria for childhood ADHD continued to meet these criteria by 18 years: correspondingly the majority (67.5%) of those who qualified for ADHD diagnosis in adulthood had not shown ADHD symptoms in childhood [12]. Similar data from a four-decade cohort study shows almost no overlap between those who qualified for an ADHD diagnosis in childhood and adulthood [13]. However, the precise level of (dis)continuity of youth psychiatric disorder remains unclear since different methodological factors (e.g. diagnostic criteria used, parent/clinician/self-reported symptoms) affect the estimates generated [14].

Research using specific disorder diagnoses may underestimate the functional persistence of pathology due to developmental changes in symptomatology or because symptom criteria for common childhood disorders may not apply to adolescents or adults (e.g. ADHD checklists querying behaviour at play or in school) [14, 15]. As such, general mental health scales, rather than formal diagnostic criteria, may facilitate more sensitive although less specific estimates of pathological continuity. Yet studies using general scales show similar patterns to those using specific diagnoses. Anselmi et al. report that of Brazilian children presenting pathological child behaviour checklist (CBCL) scores aged 4, only 31% remained deviant at 12 [16]. A German study assessing pathology using the parent-rated Strengths and Difficulties Questionnaire (SDQ), with data collected at four time-points over 6 years, suggests approximately half of children whose initial assessment showed abnormal hyperactivity scores had moved out of the abnormal range by the final testing period [17]. Even more dramatically, a longitudinal study in Brazil found just 15.3% of 11-year-olds with abnormal hyperactivity SDQ scores continued to be so classified at 18, while 86.6% of 18-year-olds with abnormal scores had not shown problematic hyperactivity in childhood [18].

Understanding individual variation in the continuity of mental health difficulties is critically important for identifying the factors that promote recovery or chronicity. In the literature to date, the factors associated with the (dis)continuity of mental health problems remain ambiguous [7]. Macro-social factors such as socio-economic status are associated with mental health outcomes across the lifecourse, and indeed seem to affect the likelihood of remission vs. persistence of early pathology [17]. However, while research on macro-social factors is crucial for informing health and social policy, such factors are generally not viable targets for direct clinical intervention.

Some research investigating the continuity of psychopathology has focused on the characteristics of the individual child. For instance, a longitudinal study of mental health problems from kindergarten to primary school found continued psychopathology was higher among boys than girls [19]. Some studies suggest this gender effect deviates across diagnostic categories, presenting evidence that externalising disorders show greater continuity for boys and internalising disorders for girls [8, 17]. It is unclear whether this effect reflects genuine gender differences in disorder trajectories or the under- or mis-diagnosis of girls with attentional and conduct difficulties [20]. Other child factors such as temperament are clearly related to mental health overall, but show limited utility distinguishing between children whose difficulties are persistent vs. transient [21].

Feng et al. suggest that while dispositional factors may predispose children to psychopathology, family factors may explain changes in symptoms over time [21]. The longitudinal literature does contain evidence linking familial variables to the persistence or remission of mental health problems. For instance, temporal increases in anxiety are associated with less secure parent–child attachment [3, 22]. Persistence of child anxiety is also related to greater maternal anxiety and experience of negative life events [22]. In children with ADHD, low maternal warmth, low maternal involvement and household chaos predict poorer trajectories [23]. These variables are often associated with maternal depression, which also correlates with longitudinal persistence of mental health problems [24, 25]. Studies confirm maternal depression is one factor differentiating children with persistent vs. remittent ADHD [12]. Some research indicates the statistical effects of maternal depression disappear when other related variables, such as economic deprivation, early parenthood and maternal substance use, are controlled [24, 25]. However, the relationship of these wider familial factors with pathological persistence is inconsistent: Beyer et al. indicate that the persistence of mental health problems is related to recent breakdowns of parental relationships, but not single-family status or maternal age or educational level [19]. Thus, the literature strongly suggests family contexts affect the (dis)continuity of child psychopathology, but the specific family variables that exert unique effects remain unclear.

Most studies exploring continuity of child psychopathology have utilised cross-sectional or retrospective longitudinal designs. Cross-sectional studies are not equipped to track individual-level changes, while retrospective studies typically rely on fallible parental recall of their child's earlier life. Prospective longitudinal designs are critical for

systematically mapping psychopathological trajectories and understanding variability in outcomes [26]. The longitudinal research that exists tends to recruit samples from clinical settings, where pathology has already been recognised by referral, diagnosis and/or treatment [7]. Given the high underdetection and underdiagnosis of mental health problems in children, research with community samples is necessary to attain a comprehensive picture of typical trajectories.

The current research utilises data from Growing Up in Ireland—the National Longitudinal Study of Children (GUI) to explore psychopathological trajectories in a large community sample of children. Previous analysis of GUI data has established that child psychopathology is statistically related to economic vulnerability, young parenthood, one-parent families, unmarried or separated parents, and low maternal education [27]. However, no GUI research has investigated the factors that predict the longitudinal remission of psychopathology. Moreover, GUI includes a range of variables on familial dynamics, such as caregiver depression, child-reported parenting style and parent-reported child–parent relationship, whose links with child mental health have not been examined in the published GUI literature. The current study has two main aims:

1. To establish the proportion of children with psychopathology at 9 years who had either improved or remained pathological by age 13, and to describe the socio-demographic and clinical profiles of these two groups.
2. To identify the factors in the familial environment at age 9 that predict pathological persistence vs. remission at age 13, controlling for other child and socio-demographic factors.

Method

GUI study

GUI is a longitudinal study that collects extensive health, psychological, social and economic data from a nationally representative sample of Irish children. The study involves an Infant Cohort who were recruited into the study at 9 months (N = 11,134), and a Child Cohort recruited at 9 years (N = 8568). The current analysis utilises the latter sample, within which two waves of data are available, collected when the children were aged 9 (2007–08) and 13 (2011–12). Using a stratified sampling strategy based on the primary school system, the original household response rate was 57% and represented approximately one in seven of all 9-year-olds resident in Ireland at that time. The retention rate for Wave 2 was 87.8% (N = 7525). Study instruments and design were developed through an extensive expert consultation process and trialled in numerous pilot stages [28]. Questionnaires were administered to children, their caregivers and teachers by trained interviewers using both self-complete and computer-assisted personal interviewing techniques. The study received ethical approval from the Irish Health Research Board's Research Ethics Committee. Further information on the study's methodological and ethical procedures is available at http://www.growingup.ie/.

Measures

The primary measure of psychopathology used in the GUI study was the Strengths and Difficulties Questionnaire (SDQ), a 25-item scale assessing five dimensions of a child's behaviour: emotional symptoms, hyperactivity/inattention, conduct problems, peer relationship problems, and prosocial behaviour. The SDQ has good psychometric properties, with satisfactory reliability, validity, sensitivity and specificity demonstrated in large community and clinical samples [29, 30]. The first four subscales can be combined to create a Total Difficulties score, which operates as an index of global psychopathology [31, 32]. Most previous similar research using the SDQ to measure pathological continuity has proceeded by classifying participants' SDQ scores into categories of psychosocial well-being ('Normal', 'Borderline' and 'Abnormal') using cut-off points derived from community norms [17, 18]. These cut-offs are deemed appropriate for young people aged between 4 and 17 [29]. While some detail is undoubtedly lost through this approach, the advantage for the current study is that longitudinal categorical shifts from Abnormal to Normal can be more definitively identified as cases with strong evidence of remission. By analysing improvement as a categorical rather than continuous variable, and excluding the Borderline cases, one can concentrate on the most distinct cases of remission vs. persistence and discard the 'noise' incurred by changes in the region of 1–3 SDQ points, which may reflect measurement error and/or have limited clinical significance.

Table 1 describes the variables included in the analysis and stipulates the GUI wave from which they were extracted. To provide appropriate levels of statistical control, the socio-demographic and child variables were extracted from Wave 2. The GUI data includes a range of variables that tap aspects of a child's familial environment (Table 1); to assess their longitudinal predictive power, these variables derived from Wave 1. All measures were completed by the child or the child's Primary Caregiver (PCG). In 97.8% of cases, the person who self-identified as the PCG was female (i.e. the child's mother).

Data analysis

GUI advises use of a statistical weight to ensure the data are representative of the population. The weighting system

Table 1 Measures included in the analysis

Category of variable	Variable	Who completed	Wave	Description
Psychopathology	Strengths and Difficulties Questionnaire (SDQ)	PCG	1 and 2	30-item scale assessing five dimensions of child's behaviour: emotional symptoms, hyperactivity/inattention, conduct problems, peer relationship problems, and prosocial behaviour. First four subscales combined to create a Total Difficulties score, which operates as an index of child psychopathology [31, 32]
Socio-demographics	Gender	PCG	2	Child's gender
	PCG citizenship	PCG	2	PCG an Irish citizen
	Household equivalised income	PCG	2	Calculated as disposable household income divided by equivalised household size. This allows for meaningful comparisons by accounting for differences in household composition. Households' equivalised income was categorised into quintiles and deciles
	Two-carer household	PCG	2	Whether PCG's spouse/partner lived in the household
Child	General health	PCG	2	Child's health in the past year rated on a 4-point scale from 'very healthy, no problems' to 'almost always unwell'
	Cognitive ability	Child	2	Standardised psychometric tests, the Drumcondra Reasoning Tests (Verbal Reasoning and Numerical Ability). Averages of verbal and numeric reasoning scores calculated and converted into logit scores that adjusted the percentage correct score using two parameters (difficulty and discrimination) for each item
Parent	PCG education	PCG	2	Whether PCG held a university degree
	PCG health	PCG	1	PCG's health rated on a 5-point scale from 'Excellent' to 'Poor'
	PCG depression	PCG	1	8-item short version of the Center for Epidemiological Studies Depression Scale (CES-D) [36], a self-report screening instrument for depression in the general population
	Pianta child–parent relationship Scale [37]	PCG	1	30 item scale measuring the positive and negative aspects of the child–parent relationship. Comprises three subscales measuring the child's dependence, closeness, and conflict with the PCG
	Parenting style inventory [38]	Child	1	15 items assessing mother's parenting style along the dimensions of responsiveness and demandingness. Using conventional cut-offs [39], parenting styles were classified as authoritative (high demandingness and high responsiveness), authoritarian (high demandingness and low responsiveness), permissive (low demandingness and high responsiveness) and neglectful (low demandingness and low responsiveness)

used (GROSS) is based on a standard iterative procedure for adjusting the completed sample to known population totals, using census data on socio-economic status, social class and family structure [28]. In this analysis, the weights for Wave 2 were applied throughout to account for differential attrition rates.

Analysis was conducted using IBM SPSS 22. Across the whole sample, differences in SDQ Total Difficulties scores

between ages 9 and 13 and gender differences were examined using a two-way repeated measures ANOVA. Total Difficulties scores were assigned into categories of psychosocial well-being using cut-off points derived from community norms [29], where 'Normal' = 0–13, 'Borderline' = 14–16, and 'Abnormal' = 17–40. Cross-tabulations quantified the frequency with which cases of Abnormal SDQ scores at 9 years shifted into the Normal and Borderline ranges at 13 years. Descriptive statistics established the demographic and clinical characteristics of those whose early pathology remitted and persisted.

With the objective of predicting longitudinal remission or persistence of psychopathology, further analysis was restricted to children who scored in the Abnormal SDQ range at 9 years ($N = 543$, 7.3%). A new dichotomous variable was created to indicate whether these children recorded a Normal or Abnormal SDQ score at age 13. The factors predicting remission vs. persistence were assessed through hierarchical logistic regression. To avoid ambiguity resulting from mere 1- or 2-point vacillations, cases classified as Borderline at age 13 were excluded from the regression analysis. As such, Normal or Abnormal 13-year-old SDQ scores comprised the dichotomous dependent variable (Abnormal coded as 0, Normal as 1). Predictors were introduced in four blocks, with the order determined according to their theoretical and/or empirical relatedness to psychopathological outcomes in previous literature. The first block contained the child's continuous SDQ Total Difficulties score at age 9, to control for the extent of initial pathology. The second and third blocks included demographic and child variables at age 13. The fourth and final block added a range of familial variables collected when the child was aged 9. Missing data were excluded pairwise, leaving an active sample for the regression analysis of $N = 249$.

Results

Do SDQ scores differ across age and gender?

A two-way mixed ANOVA on the whole GUI sample detected a significant main effect of age on Total SDQ scores, $F(1, 7292) = 276.52$, $p < 0.001$, $\eta_p^2 = 0.04$. Descriptive statistics showed mean scores were lower at 13 years ($M = 7.09$, SD = 5.4) than 9 years ($M = 7.98$, SD = 5.34). This pattern was consistent across SDQ subscales (Table 2). There was also a significant main effect of gender with boys scoring higher than girls at both time-points, $F(1, 7292) = 15.74$, $p < 0.001$, $\eta_p^2 = 0.002$. There was no significant interaction between gender and age ($p = 0.26$).

Table 3 Cross-tabulation of rates of pathology at age 9 and 13 ($N = 7488$)

		Age 13			Total
		Normal	Borderline	Abnormal	
Age 9					
Normal	*N*	5982	237	158	6377
	%	93.8	3.7	2.5	100
Borderline	*N*	380	84	104	568
	%	66.9	14.8	18.3	100
Abnormal	*N*	204	116	223	543
	%	37.6	21.4	41.1	100
Total	*N*	6566	437	485	7488
	%	87.7	5.8	6.5	100

What is the overall prevalence of pathology and how does it change over time?

Based on Total SDQ scores at 9, 85.2% ($n = 6377$) of the weighted sample was classified as Normal, 7.6% ($n = 568$) Borderline and 7.3% ($n = 543$) Abnormal. At age 13, the proportion classified as Normal grew to 87.7% ($n = 6566$), while the proportion categorised as Borderline and Abnormal fell to 5.8% ($n = 437$) and 6.5% ($n = 485$), respectively. This longitudinal change in overall SDQ profiles was statistically significant, χ^2 (4, $n = 7488$) = 1845.55, $p < 0.001$, $V = 0.35$.

Table 3 displays how the various case classifications at 9 were redistributed at 13. It shows that of those classified Abnormal at 9, 41.1% ($n = 223$) remained in this category,

Table 2 Mean SDQ subscale scores across gender and study wave

	Age 9			Age 13		
	Total sample mean (SD)	Female mean (SD)	Male mean (SD)	Total sample mean (SD)	Female mean (SD)	Male mean (SD)
Emotional	2.14 (2.06)	2.27 (2.10)	2.02 (2.01)	1.90 (2.01)	2.10 (2.06)	1.72 (1.95)
Conduct	1.36 (1.51)	1.28 (1.47)	1.44 (1.55)	1.23 (1.48)	1.21 (1.44)	1.25 (1.51)
Hyperactivity	3.23 (2.50)	2.90 (2.37)	3.54 (2.57)	2.82 (2.48)	2.47 (2.35)	3.15 (2.55)
Peer	1.26 (1.5)	1.26 (1.45)	1.26 (1.55)	1.14 (1.50)	1.11 (1.45)	1.18 (1.53)
Total difficulties	7.98 (5.34)	7.70 (5.23)	8.26 (5.43)	7.09 (5.40)	6.88 (5.35)	7.29 (5.44)

21.4% ($n = 116$) moved into the Borderline range, and 37.6% ($n = 204$) met the cut-off point for Normal. The overall continuity across the sample (i.e. the proportion who retained the same classification in both waves) was 84% ($n = 6289$).

What are the characteristics of those with remittent vs. persistent pathology?

Further analysis was restricted to cases classified as Abnormal at age 9 ($n = 543$). Descriptive statistics were computed to characterise the groups whose early pathology had remitted (i.e. shifted into Normal range, $n = 204$) or remained persistent (Abnormal range, $n = 223$) at 13. The mean age 13 Total Difficulties score of the remittent group was 8.85 (SD = 2.93) and the persistent group 21.77 (SD = 4.23), $t(425) = 36.35$, $p < 0.001$, $d = 3.55$.

Table 4 displays the socio-demographic characteristics of the two groups at 13. Overall, the groups were relatively similar, although the profile of those who had improved was characterised by higher social class and more married two-parent households.

GUI also collected limited data on participants' clinical histories. Table 5 displays the relevant clinical information that was recorded in both study waves. It shows that children whose pathology remained persistent had greater prevalence of parent-identified chronic illness, speech and language difficulty, autism spectrum disorder and emotional or behavioural disorder (e.g. ADHD). Almost one quarter of cases of persistent pathology reported an emotional or behavioural disorder, as opposed to approximately 3% of those who improved. Approximately 15% of children with persistent pathology had been prescribed medication for an emotional or behavioural disorder (e.g. Ritalin), while no child with improved pathology had such a prescription. While it is unsurprising that medication was concentrated among the most extreme cases, the absence of medication in the improved group suggests that remission of difficulties was not due to pharmaceutical intervention. Inspecting the individual SDQ subscales, the dimensions that most differentiate 9-year-olds who subsequently improved vs. remained pathological were Hyperactivity and Peer Problems scores.

Table 4 Socio-demographic profiles of 13-year-olds whose prior pathology had either remitted or persisted ($N = 427$)

	Remittent pathology at 13 ($N = 204$)		Persistent pathology at 13 ($N = 223$)	
	N	%	*N*	%
Gender				
Male	109	53.4	129	57.8
Female	95	46.6	94	42.2
Household structure				
1 parent, 1–2 children	30	14.7	42	18.8
1 parent, ≥3 children	22	10.8	38	17.0
2 parents, 1–2 children	79	38.7	85	38.1
2 parents, ≥3 children	73	35.8	59	26.5
PCG marital status				
Married and cohabiting	126	61.8	117	52.5
Married and separated	11	5.4	27	12.1
Divorced/widowed	20	9.8	31	13.9
Never married	40	19.6	47	21.1
Household equivalised income quintile				
1st (lowest)	70	34.3	70	31.4
2nd	38	18.6	48	21.5
3rd	22	10.8	31	13.9
4th	38	18.6	47	21.1
5th (highest)	26	12.7	15	6.7
Family occupational class				
Professional workers	13	6.4	6	2.7
Managerial and technical workers	50	24.5	34	15.2
Non-manual workers	25	12.3	35	15.7
Skilled manual workers	47	23.0	52	23.3
Semi-skilled workers	31	15.2	21	9.4
Unskilled workers	3	1.5	13	5.8
Never worked—no social class	34	16.7	57	25.6
PCG educational attainment				
None/primary	14	6.9	29	13.0
Secondary/vocational	140	68.7	146	65.5
Diploma/certificate	28	13.7	31	13.9
Primary degree	15	7.4	14	6.3
Postgraduate degree	7	3.4	3	1.4
PCG Irish citizen				
Yes	192	94.1	214	96.0
No	12	5.9	7	3.1

What variables predict remission vs. persistence of pathology?

A hierarchical logistic regression was performed to establish the various predictors of remitted (vs. persistent) overall pathology. Table 6 displays the results. Unsurprisingly, initial severity of symptoms, as indicated by the SDQ Total Difficulties score at 9, was strongly related to the likelihood of improvement by 13, i.e. children with initially lower scores were more likely to remit. Controlling for initial severity, several other factors showed independent relationships with remission.

Gender was highly significant in all models: persistent pathology was more likely in male than female children (Block 4 $\beta = -1.00$, $p = 0.001$, CI = 0.20–0.67). Persistence of pathology was also significantly more likely in single-carer households ($\beta = -0.71$, $p = 0.04$,

Table 5 Clinical profile of children whose pathology either remitted or persisted ($N = 427$)

	Data collected aged 9				Data collected aged 13			
	Remitted pathology at 13 ($N = 204$)		Persistent pathology at 13 ($N = 223$)		Remitted pathology at 13 ($N = 204$)		Persistent pathology at 13 ($N = 223$)	
	N	%	*N*	%	*N*	%	*N*	%
Abnormal score on SDQ scales								
Total (≥17)	204	100.0	223	100.0	0	0.0	223	100.0
Emotional (≥5)	141	69.1	149	66.8	26	12.7	147	65.9
Conduct (≥4)	124	60.8	142	63.7	10	4.9	141	63.2
Hyperactivity (≥7)	97	47.5	159	71.3	14	6.9	170	76.2
Peer problems (≥4)	85	41.7	141	63.2	19	9.3	108	48.4
Prosocial (≤4)	13	6.4	15	6.7	5	2.5	28	12.6
Current chronic illness/disability								
Yes	43	21.1	94	42.0	48	23.5	91	40.8
No	161	78.9	130	58.3	156	76.5	132	59.2
Number of GP contacts in last 12 months								
≤3	188	92.2	204	91.5	183	89.7	206	92.4
≥4	16	7.8	20	9.0	21	10.3	17	7.6
Number of other health professional contacts in last 12 months								
≤2	197	96.6	186	83.4	194	95.1	172	77.1
≥3	7	3.4	35	15.7	10	4.9	51	22.9
PCG-identified disorder								
Speech and language difficulty	4	2.0	29	13.0	3	1.5	13	5.8
Autism/Asperger's	2	1.0	24	10.8	2	1.0	26	11.7
Emotional or behavioural disorders, e.g. ADHD	7	3.4	51	22.9	5	2.5	54	24.2
Treatment								
Prescribed medication for emotional or behavioural disorder[a]	–	–	–	–	0	0.0	34	15.2

[a] Not assessed at 9 years

CI = 0.25–0.97). Income and PCG citizenship did not show a significant relation with outcomes in any iteration of the regression.

The child's physical health was significantly related to psychopathological outcomes: improvement was more likely in children rated healthier by their parents ($\beta = -0.64$, $p = 0.03$, CI = 0.30–0.92). Greater cognitive ability also increased the likelihood of remission of symptoms ($\beta = 0.61$, $p = 0.002$, CI = 1.26–2.70).

The final regression model revealed no significant effects of parenting style or child–parent relationship measures recorded when the child was 9. PCG education and health also emerged as non-significant. However, PCG depression did show a significant effect: the higher the caregiver's depression score at 9, the less likely their child had improved 4 years later ($\beta = -0.07$, $p = 0.03$, CI = 0.87–0.99).

Discussion

The current analysis accords with previous research indicating that the rate of chronic mental health problems in childhood is approximately 2–3% [17]. For clinicians and policy makers, it is critical to identify the children with emerging psychological disturbance who are likely to comprise that 2–3%, so that early interventions can be instigated and clinical resources distributed in an optimally effectual manner. The current research contributes to the development of such predictive tools by clarifying the socio-demographic, child and familial variables that do (and do not) differentiate children with persistent vs. transient pathology. The results suggest mental health policy and practice should direct particularly intense clinical resources towards children who are male, live in one-parent households and have poor physical health and cognitive ability. In counterpoint to previous

Table 6 Results of the hierarchical logistic regression analysis predicting remission (vs. persistence) of pathology

	Block 1						Block 2						Block 3						Block 4					
	B	SE B	*p*	e^B	CI (L)	CI (U)	*B*	SE B	*p*	E^B	CI (L)	CI (U)	*B*	SE B	*p*	e^B	CI (L)	CI (U)	*B*	SE B	*p*	e^B	CI (L)	CI (U)
Total SDQ score at 9	−0.403	0.058	0.000***	0.669	0.597	0.748	−0.455	0.066	0.000***	0.634	0.557	0.722	−0.430	0.066	0.000***	0.651	0.571	0.741	−0.400	0.070	0.000***	0.670	0.585	0.769
Equivalised income deciles							−0.013	0.050	0.801	0.987	0.895	1.089	−0.044	0.054	0.411	0.957	0.861	1.063	−0.041	0.057	0.469	0.960	0.858	1.073
PCG Irish citizen							0.864	0.598	0.148	2.373	0.735	7.657	0.816	0.616	0.186	2.261	0.676	7.563	0.750	0.684	0.273	2.117	0.554	8.095
Child gender							−0.848	0.277	0.002**	0.428	0.249	0.737	−1.068	0.295	0.000***	0.344	0.193	0.613	−1.007	0.311	0.001**	0.365	0.198	0.672
Two-carer household							−0.750	0.311	0.016*	0.472	0.257	0.869	−0.740	0.323	0.022*	0.477	0.253	0.899	−0.714	0.350	0.041*	0.489	0.247	0.971
Child general health													−0.572	0.271	0.035*	0.565	0.332	0.959	−0.638	0.285	0.025*	0.528	0.302	0.924
Cognitive ability													0.542	0.182	0.003**	1.719	1.203	2.457	0.610	0.195	0.002**	1.841	1.255	2.700
PCG Education																			0.508	0.503	0.312	1.662	0.621	4.451
Mother Authoritarian (vs. authoritative)																			0.465	0.532	0.382	1.592	0.561	4.514
Mother Permissive (vs. authoritative)																			0.054	0.415	0.897	1.055	0.468	2.378
Mother Neglectful (vs. authoritative)																			0.287	0.911	0.753	1.333	0.223	7.954
PCG conflict																			−0.025	0.017	0.140	0.975	0.943	1.008
PCG closeness																			0.025	0.026	0.324	1.026	0.975	1.079

Table 6 (continued)

	Block 1					Block 2					Block 3					Block 4				
	B	SE B	*p*	e^B	CI (L)	CI (U)	*B*	SE B	*p*	E^B	CI (L)	CI (U)	*B*	SE B	*p*	e^B	CI (L)	CI (U)	*B*	SE B	*p*	e^B	CI (L)	CI (U)
PCG dependence																			0.049	0.044	0.270	1.050	0.963	1.146
PCG health																			0.149	0.160	0.350	1.161	0.849	1.589
PCG depression																			−0.072	0.033	0.027*	0.930	0.873	0.992
Constant	8.083						9.017						9.966						7.458					
Model χ^2	76.263						95.567						110.143						122.012					
Cox & Snell R^2	0.224						0.273						0.307						0.334					

Dependent variable: remission coded as 1, persistence coded as 0. Included cases $N = 249$ (missing data excluded pairwise)

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$

research [3, 21–23], the current study suggests that familial dynamics may not contribute much additional predictive value, with the exception of depression in the primary caregiver. This may be a fruitful target for intervention: future research should investigate whether treating parental depression may have the secondary effect of preventing a child's early mental health difficulties from becoming chronic.

Longitudinal trends in psychopathology

In the GUI data overall, average levels of psychopathology decreased between the ages of 9 and 13. This conflicts with previous international studies, which suggest pathology increases as children age [6, 19]. The application of statistical weights means that this is not attributable to biased attrition. This trend is particularly surprising given the historical contexts in which the two datasets were collected (2007–08 vs. 2011–12). In Ireland, the early-mid 2000s were characterised by rapid economic growth and prosperity, which ended when the country entered severe recession in 2008. Previous GUI analysis found this changing macro-economic context produced a substantial increase in participants' economic vulnerability in Wave 2 relative to Wave 1 [27]. The current analysis indicates that this economic context had not, as yet, negatively affected rates of child psychopathology. Further research with upcoming GUI waves, and with another cohort who were infants during the economic crisis, will clarify whether this resilience holds in the long term.

The reduction in population-wide pathology accords with some previous studies [16, 17]. For instance, Becker et al.'s longitudinal German study, also using the SDQ, showed the proportion scoring in the Abnormal range shrunk over time [17]. This was mostly attributable to a decrease in problematic scores on the hyperactivity subscale. Becker et al. expressed uncertainty regarding whether this was due to treatment effects or natural remission of hyperactivity symptoms. Problematic levels of hyperactivity also showed a longitudinal decrease in the current GUI data. However, here the overall improvement of pathology cannot be attributed to pharmaceutical treatment of ADHD symptoms, since no child in the remission group had been prescribed psychotropic medication. Moreover, very few children who improved held a psychological diagnosis at either 9 or 13, which likely indicates symptoms remitted without intervention from mental health services. More detailed information on service engagement is not available in the existing GUI data.

A key contribution of this study is to highlight the substantial rates of psychopathological discontinuity in a representative community sample. Of those who met criteria for Abnormal SDQ scores aged 9, just two-fifths remained so classified at 13: one-fifth moved into the Borderline range,

and over one-third met the cut-off point for Normal. While SDQ Total Difficulties scores have proven valuable as a screening instrument in cross-sectional data [33], the current results question their utility as a prognostic tool. Descriptive results indicated the SDQ dimensions that most differentiated children with chronic difficulties were high Hyperactivity and Peer Problems scores. Children with elevated scores on these subscales may require particularly intense monitoring and intervention.

Predicting persistence or remission of pathology

Confirmation that remission of emotional and behavioural pathology is common reinforces the importance of identifying the factors associated with that improvement, which may prove useful targets for interventions. The current analysis vindicates previous indications that gender is important, with boys' pathology more likely to prove persistent [17, 19]. The finding that single-carer households predict the persistence of pathology is not entirely consistent with previous literature, where some studies report no relation [19]; further research is required to verify this effect. Interestingly, in contrast to previous research [17], income did not show an independent relationship with the persistence of pathology. This may reflect a ceiling effect: since the regression model was restricted to children showing abnormal levels of pathology at 9, this subsample was already more economically deprived than the total community sample. Within this subsample, income variations had no additional effect on the probability of remission or persistence of problematic behaviours.

The analysis also confirms that a child's general health and cognitive ability affect the persistence of psychopathology. It is known that children with physical illness have increased risk of comorbid mental illness, especially when there is neurological involvement [34]. The current study suggests this risk applies to the persistence as well as incidence of psychopathology. Clinicians working with children showing mental health difficulties should be particularly vigilant when children have poor physical health or low cognitive ability, since both are risk factors for early difficulties becoming chronic in nature. In such cases, the development of mental health assessment and treatment plans should coordinate with relevant professionals in the education system and other medical specialties.

While important in understanding the aetiology of childhood psychopathology, the above socio-demographic and child factors are rarely viable targets of immediate mental health interventions. Family dynamics are more malleable to intervention, and as such of crucial clinical importance. A key finding of this analysis was that the only familial variable at age 9 that independently predicted the persistence of problems at 13 was depression in the primary caregiver. This accords with prior findings linking chronic child psychopathology to past and present maternal depression [24, 25]. However, previous research has found that the statistical effects of maternal depression abate once other variables are taken into account. For instance, Fergusson and Lynskey report that maternal depression was related to conduct and attentional problems in late childhood, but the effects disappeared once socio-economic confounds were controlled [25]. Similarly, Barker et al. report maternal depression in infancy increased risk of later diagnoses, but much of this association was explained by confounds such as early parenthood and maternal substance use [24]. In the current analysis, caregiver depression remained significantly related to the persistence of psychopathology independently of socio-demographic and other parent–child relationship factors.

Parental depression could influence child mental health through numerous different pathways, including shared genetic predisposition and the modelling of parental behaviour. Since the regression model controlled for initial variations in SDQ scores, caregiver depression exacerbating child psychopathology is a more plausible causal direction than the reverse. Nevertheless, it is likely that the relationship is bidirectional and mutually reinforcing. Disentangling these pathways of influence should be a priority for future research. This notwithstanding, the precise nature of causality may be immaterial for the implications for clinical practice and health policy. The finding that persistent child psychopathology is preceded by caregiver depression indicates that treatment should attend to both. The identification of one of these problems should alert clinicians to risks of the other and prompt initiation of appropriate interventions. If the problems are genuinely mutually reinforcing, targeting parental difficulties will have beneficial effects for the child and vice versa. This reinforces the need for joined-up, family systems level mental health services.

Parenting style and child–parent relationships at 9 did not predict remission or persistence of mental health problems at 13. Agnew-Blais et al. also found minimal differences between the familial environments of those with persistent vs. remitting ADHD symptoms [12]. This is interesting, given that parenting courses (e.g. the Incredible Years® programme) [35] are often recommended as a means of delivering interventions to at-risk children. While parenting courses may have beneficial effects for child and family wellbeing generally, the current research suggests parenting behaviours may not be productive targets for interventions seeking to affect mental health trajectories in children already displaying emotional and behavioural problems. However, the current analysis was restricted to those familial variables included in the GUI study; further investigation of other family factors (e.g. sibling relationships, communication styles) may show more promising results. It is also possible that extra-familial environments, such as peer and

education experiences, may represent important sources of environmental influence on psychopathological trajectories. Future research should investigate these possibilities.

Conclusion

This study has several limitations. While the use of a generic measure of psychopathology afforded a holistic overview of the factors involved and ensured sufficient cases for analysis, different types of pathology have different aetiologies. Future research should endeavour to tease apart the trajectories unique to different forms of child psychopathology. Assessing psychopathology using differentiated diagnostic criteria rather than general mental health scales may yield more nuanced results. Analytic depth was also lost by decomposing continuous SDQ scores into three categories, and omitting Borderline cases from the regression analysis. Categorical operationalisations of pathology have the advantage of indicating clear cases of persistence vs. remission in relation to community norms of pathology, and are standard in longitudinal studies of this sort [16–18]. Nevertheless, operationalising psychopathology as a continuous rather than categorical variable would facilitate a more fine-grained analysis. A further limitation is the fact that most measures were reported by the parent rather than child or other informants. Research does suggest that parent reports are more sensitive than self-reports in measuring youth psychopathology [14]. However, SDQ sensitivity is better with multi-informant ratings [33], which were not available in GUI Wave 2. More child-generated data is being collected in the third wave of data collection with this cohort, which is currently ongoing. Tracking how these trends evolve as the cohort moves into adulthood will provide rich insight into the trajectories of early onset mental health difficulties.

The results of this study converge with the emerging empirical consensus that longitudinal remission of early mental health difficulties is common [8–19]. While this does not trivialise the immediate and residual suffering youth mental illness engenders, this message may offer some hope to young people, families and clinicians struggling to manage emotional and behavioural disorders. Moreover, these findings should focus particular empirical and clinical attention on those who do not recover, that is, the 2–3% of children who experience chronic mental health difficulties. The current study provides information that will help identify this subgroup in clinical populations and suggests one viable target for intervention, namely parental mental health. Further research of this type is required to develop more finely honed prediction tools and intervention strategies.

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Compliance with ethical standards

Ethical statement The GUI study received ethical approval and monitoring from the Irish Health Research Board and was performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. All persons gave their informed consent prior to their inclusion in the study.

Conflict of interest The authors declare that they have no conflict of interest.

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