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Research Paper

Bullying victimisation as a mediator in the association between childhood epilepsy and later emotional and behavioural difficulties: Evidence from the British National Child development study

Emma Blundell [a,b,*], Vaughan Bell [a], King-Chi Yau [a]

[a] Research Department of Clinical, Educational and Health Psychology, UCL, London, UK
[b] North London NHS Foundation Trust, UK

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ABSTRACT

Objective: To investigate whether bullying victimisation contributes to associations between childhood epilepsy and emotional and behavioural difficulties into adolescence.
Method: This was a longitudinal population-based, matched cohort study leveraging data from the National Child Development Study of 1958. The study population of 715 individuals included children with confirmed epilepsy at age 7 years and a matched reference population without epilepsy or other chronic health condition. Linear regression analyses investigated whether CWE were more likely than peers to experience emotional and behavioural problems at age 7, 11, and 16 years as reported by parents, with scores subdivided into internalising and externalising scales. We examined the contribution of bullying victimisation to these associations at age 7 years and tested mediation of associations with later outcomes at ages 11 and 16 years.
Results: 65 CWE were matched to 650 children without epilepsy, with baseline covariates balanced after propensity score matching. Linear regression models found CWE at significantly higher risk of internalising and externalising difficulties at all ages (effect sizes from $\beta = 0.11$ to 0.63), except internalising at age 11 years. Bullying victimisation accounted for 29% of the association between epilepsy and internalising at age 7 years (CI, −0.02–1.25; $p = 0.05$) and mediated 18% of the association with internalising at age 16 years (CI, 0.03–0.99; $p = 0.03$).
Significance: Bullying victimisation may partially contribute to elevated internalising difficulties among CWE across development. Targeting bullying victimisation may help reduce psychosocial risk in this population.

1. Introduction

Research has shown that children with epilepsy (CWE) are at a much greater risk for a range of emotional and behavioural difficulties than healthy peers [1,2]. These include anxiety, depression, attention difficulties, and conduct problems [3]. The presence of these difficulties in epilepsy is associated with poorer treatment adherence, poorer seizure control and increased healthcare utilisation [2,4–6]. They can also have a greater impact on quality of life than seizure-related factors [3]. Therefore, there is an urgent need to identify and manage risk factors that contribute to these comorbidities [3]. One consistent psychosocial risk factor for emotional and behavioural difficulties in the general population is bullying victimisation [7]. However, whilst it has been found that CWE are more likely to experience bullying than healthy peers [8], the potential contribution of bullying victimisation to emotional and behavioural difficulties in CWE remains poorly understood.

Epilepsy is one of the most common neurological conditions in childhood characterised by unprovoked, recurrent seizures [9]. Epilepsies result from diverse aetiologies and age of onset varies across epilepsy classifications, with the highest rate in the first year after birth [10,11]. Beyond seizures, CWE often face a burden of emotional (such as anxiety or depression), behavioural (such as attention problems) and neurodevelopmental challenges greater than those observed in children living with other chronic diseases, and which can persist even in cases of symptom remission [12–15]. Reviews have identified a pooled prevalence of anxiety in 18.9% and depression in 13.5% of CWE, whilst rates of ADHD have been estimated as 2.5–5.5 times higher compared to

* Corresponding author.
E-mail address: emma.blundell@ucl.ac.uk (E. Blundell).

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healthy controls [16,17]. Biomedical models have traditionally focused on seizure control, meaning such comorbidities have been under-recognised, under-treated, and/or often associated with epilepsy-specific factors, such as seizure frequency or medication effect [18,19].

There has been increasing recognition of the cognitive and mental health challenges associated with paediatric epilepsy, alongside the development of psychosocial interventions to support wellbeing [20]. Quality of life has also been associated with the social impact of epilepsy, including anxiety around peer acceptance in childhood and the impact of stigma [3,21–23]. Recent findings from a group intervention suggest that social and psychosocial support can improve how CWE manage difficult emotions and increase their confidence in talking about epilepsy with others [24]. From a developmental psychopathology perspective [25], adolescence is a period of critical biological and social transition during which the impact of social factors may be particularly important. At this developmental stage, young people navigate increasingly complex peer relationships and form a sense of identity, supported by the development of social cognitive processes such as cognitive empathy [26,27]. For CWE, social challenges, stigma, and neurological differences may heighten vulnerability to peer difficulties, risking the loss of protective peer support against emotional and behavioural difficulties at this formative time [28].

Bullying victimisation from peers is one of the most robust and preventable psychosocial risk factors for emotional and behavioural difficulties early in life [29]. Victimisation remains associated with a range of adverse social, health, and economic outcomes decades after exposure [30]. Bullying is typically defined as a form of repeated, intentional aggressive behaviour involving a power imbalance between perpetrator and victim of the same age group [31]. Childhood bullying is common worldwide, affecting up to a quarter of typically developing children [32]. However, studies have shown increased prevalence in special populations where developmental and social processes underlying peer relationships may be disrupted, such as those living with neurodevelopmental conditions or chronic illness [33–36]. It is noteworthy that the risk of being bullied for CWE is almost twice as high as that of healthy peers and those with other chronic conditions, net to the effect of confounding variables such as socioeconomic status and disease severity [8,36,37]. Few studies have explored the impact of bullying on CWE specifically [36].

To our knowledge, only a handful of studies have examined longitudinal associations between childhood epilepsy and emotional/behavioural comorbidities [21,38] and no longitudinal studies have investigated whether bullying victimisation mediates the association between childhood epilepsy and these outcomes. Most studies on bullying in CWE have focused on determining prevalence [36,37], whilst the few studies looking at psychosocial correlates are limited by cross-sectional samples and recruitment from specialist clinics [8,39]. Such findings have limited generalisability and preclude understanding of developmental trajectories [40,41].

To address research gaps, this study used a longitudinal birth cohort study to investigate whether the presence of epilepsy at age 7 was associated with emotional/behavioural difficulties (including internalising and externalising difficulties) at ages 7, 11, and 16 years; whether these associations changed after adjusting for bullying victimisation; and if so, whether bullying victimisation mediated the associations between epilepsy and emotional/behavioural difficulties.

2. Methods

2.1. Design and population

This cohort study used the National Child Development Study (NCDS), an ongoing longitudinal investigation of all individuals born in Britain during 1 week in March 1958 (initial $n = 17,415$) [42]. We used information from the initial birth study and surveys conducted at age 7 years (first sweep; 1965), 11 years (second sweep; 1969), and 16 years (third sweep; 1974). We included individuals with available data on biological sex at birth and physical health at age 7 years (Appendix S1).

2.2. Measures

Epilepsy status at age 7 years was established following similar studies [38,43]. A positive response to a parent-report item on history of seizures at age 7 years 'has the child had a fit or convulsion after the first year' was used to select case notes from family doctors that determined epilepsy status [43]. Epilepsy was defined as a history of two unprovoked seizures, based on International League Against Epilepsy recommendation [44], Epilepsy and o. C. a. T. o. t. I. L. A. [45].

Emotional/behavioural difficulties in childhood have often been classified into internalising and externalising difficulties respectively. Guided by this framework, we derived variables from the parent-rated Rutter-A measure administered at age 7, 11, and 16 years, in which parents rated behaviours on a three-point scale (0 = never to 2 = frequently). An internalising scale was created by summing responses to miserable, worries, and fearful; an externalising scale was created by summing responses to destructive, irritable, and disobedient. The maximum score for each scale was 6, with greater scores indicating greater difficulties. These scales do not represent diagnostic constructs but rather capture the relevant emotional and behavioural symptoms available in the dataset, grouped within broader internalising and externalising domains. This approach is consistent with comparable studies and recent harmonisation guidance on mental health measures in cohort studies [38,46,47]. Rutter scales have been shown to have adequate reliability and validity in epidemiological research [48].

Bullying victimisation was derived from a single Rutter-A item for which parents rated 'bullied by other kids' (0 = never to 2 = frequently). We coded exposure by age 7 years (maximum score = 2) and exposure by age 11 years (maximum score = 4), with greater scores indicating greater bullying victimisation. Modest correlations were observed between bullying and internalising ($r = 0.26$) and bullying and externalising ($r = 0.16$) at age 7 years. We addressed potential multicollinearity amongst predictors in regression analyses by calculating variance inflation factors (VIF). All VIF values were below 5, indicating no serious multicollinearity concerns [49].

We measured three potential confounders hypothesised to influence both exposure and outcomes: biological sex, gestational age, and SES (Appendix S2) [50–55]. We defined sex as assigned male or female at birth. We defined gestational age as pre-term or full-term (pre-term $\leq$ 259 days) [56]. We created a composite SES score consistent other research on health disparities [57]. We derived SES scores by coding paternal occupational class at the child's birth (0 = manual, 1 = non-manual) and maternal education (0 = left school at 16 years, 1 = continued past 16 years) as binary variables. These variables were summed to create a composite SES score ranging from 0 to 2, representing low, medium, or high SES. The moderate correlation between SES indicators ($r = 0.53$), supported our decision to generate an aggregated score.

2.3. Propensity score matching

This study used propensity score matching (PSM) to reduce confounding by creating a control group balanced on observed baseline characteristics [58,59]. We first estimated propensity scores for epilepsy diagnosis at age 7 years via logistic regression, using the pre-specified confounders of sex, gestational age, and SES. Based on the estimated propensity score, we matched CWE to healthy children using 1:10 nearest neighbour matching without replacement, within the recommended caliper width of 0.1 of the standard deviation of the logit of the propensity score [60,61]. We excluded children with other neurological or health conditions from the matching process to reduce residual confounding. We checked the balancing properties of the propensity score by comparing standardised differences (Table 1) using a threshold of

Table 1
Baseline characteristics of children with epilepsy and children without epilepsy in full NCDS sample and matched sample.

Variable	Epilepsy (n = 65)% (n)	Full sample (n = 13, 707)			Matched sample (n = 715)		
		No epilepsy (n = 13,642)% (n)	**Standardised difference** (%)	**P value**	**No epilepsy (n = 650)**% (n)	**Standardised difference** (%)	**P value**
Female	46.42 (30)	48.7 (6637)	0.05	0.78	46.2 (300)	< 0.001	1.00
Other neurological condition	13.8 (9)	3.6 (497)	0.37	< 0.001	100 (0)	0.57	< 0.001***
Other chronic health condition	18.5 (12)	7.6 (1030)	0.33	0.002	100 (0)	0.67	< 0.001***
Socioeconomic position			0.14	0.59		< 0.001	1.00
Low	63.1 (41)	60.8 (8300)			63.1 (410)		
Medium	27.7 (18)	25.6 (3497)			27.7 (180)		
High	9.2 (6)	13.5 (1845)			9.2 (60)		
Born pre-term (%)	10.8 (7)	4.3 (590)	0.25	0.02*	10.8 (70)	< 0.001	1.00
Bullying victimisation at age 7			0.33	0.02*		0.33	0.03*
Never	49.2 (32)	65.1 (8882)			64 (416)		
Occasionally	41.5 (27)	29.6 (4038)			32 (208)		
Frequently	9.2 (6)	5.3 (722)			4 (26)		
Bullying victimisation by age 11			0.41	0.003**		0.39	0.01**
Never	35.4 (23)	54.2 (7392)			52.9 (344)		
Occasionally	38.5 (25)	31.7 (4330)			32.8 (213)		
Frequently	26.2 (17)	14.1 (1920)			14.3 (93)		
Internalising behaviours (mean/ SD)							
Age 7	1.78 (1.65)	1.39 (1.31)	0.26	0.02*	1.35 (1.25)	0.30	0.01**
Age 11	1.85 (1.59)	1.53 (1.34)	0.22	0.06	1.54 (1.32)	0.21	0.08
Age 16	1.22 (1.17)	0.90 (1.16)	0.27	0.03*	0.88 (1.16)	0.29	0.03*
Externalising behaviours (mean/ SD)							
Age 7	1.92 (1.81)	1.40 (1.21)	0.34	0.001***	1.29 (1.14)	0.42	< 0.001***
Age 11	1.55 (1.33)	1.21 (1.07)	0.28	0.01**	1.18 (1.05)	0.32	0.01**
Age 16	1.15 (1.50)	0.76 (1.02)	0.31	0.002***	0.70 (0.91)	0.36	< 0.001***

Note. Significance levels $p \leq 0.05$*, $p \leq 0.01$**, $p \leq 0.001$***. For ease of understanding of descriptive analyses, continuous scores have been split at tertiles of their distributions to derive categorical variables.

10% as indication of good balance [62].

2.4. Statistical analysis

All analyses were conducted in R (version 4.4.1). Our analysis code was made publicly available after publication: https://github.com/emmablundell/NCDSepilepsy_bullying_difficulties.git.

For objectives 1 and 2, we used linear regression models to examine associations between epilepsy at age 7 years and outcomes at age 7, 11, and 16 years. Where associations were statistically significant, we assessed whether these changed after adjusting for exposure to bullying victimisation by age 7 years. When modelling outcomes at age 16 years, we adjusted for exposure to bullying victimisation by age 11 years. Objective 3 was addressed only if epilepsy was no longer a significant predictor in models that adjusted for bullying victimisation. In these cases, we quantified the extent to which bullying victimisation accounted for the association between epilepsy and outcomes by comparing estimates from models with and without adjustment for bullying victimisation, to estimate the proportion mediated. All analyses were conducted with the matched sample of children with epilepsy matched to healthy children on biological sex, gestational age, and SES. No further adjustment for confounders was made in analyses due to risk of bias [63]. We adopted a statistical significance threshold of $\alpha = 0.05$ (5%) for all analyses.

We fitted PSM and regression models on participants with data on biological sex at birth and health conditions at age 7 years, and imputed mediator, confounder and outcome data using multiple imputation by chained equations. We imputed 50 datasets using all variables included in the models, plus auxiliary variables previously associated with attrition [64]and those hypothesised to be associated with variables to be imputed (Appendix S3). As sensitivity analyses, we repeated the PSM procedure to permit matching of children with other chronic health or neurological conditions, then repeated all analyses using this sample. We used the 'MatchIt' package for PSM [65] and the 'mediation' package to perform mediation analyses with 5,000 bootstrap simulations [66].

3. Results

3.1. Participants

Of the original cohort of 17,415 children, 11, 996 children had available sex data at birth and health data at age 7 years and thus were retained in the sample. 65 (0.5%) children had confirmed epilepsy at age 7 years, yielding a total matched sample size of $n = 715$ (Fig. 1).

At age 7 years, a greater proportion of CWE were male and from a low socioeconomic position than for healthy children. CWE were reported to have higher internalising/externalising difficulties at all ages (Table 1). At both age 7 years and 11 years, bullying victimisation scores were higher for CWE, males, children from low socioeconomic backgrounds, and children with higher internalising scores (Table S1). In the matched sample (n = 715), bullying victimisation data was missing for 13 children (1.8%) at age 7 years and 137 children (19.2%) at 11 years (Table S5). Missing Rutter-A scores ranged from 1.1% to 32.6% across timepoints and scales, with highest missingness at age 16 years (Table S6). Little's MCAR Test indicated that data were not missing completely at random ($\chi 2 = 44{,}331.62$, df = 2,016, $p < 0.0001$). Consequently, we employed multiple imputation which provides less

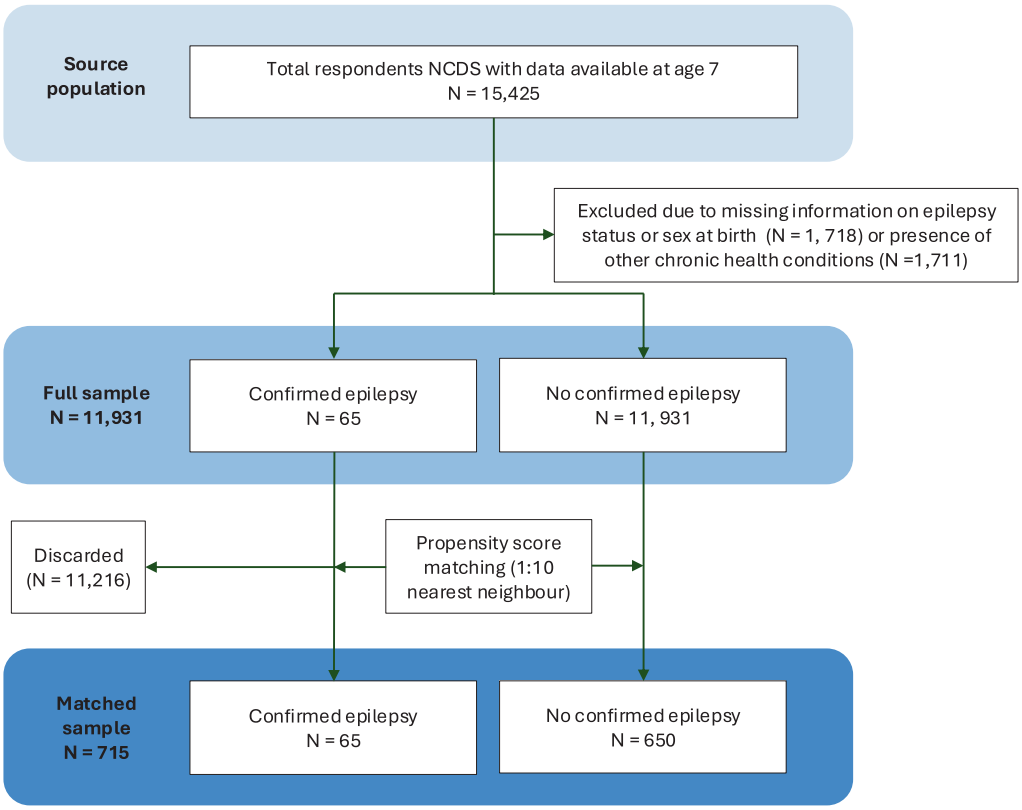

Fig. 1. Sample flowchart.

biased estimates than complete case analysis when data are not missing completely at random [67].

3.2. Is childhood epilepsy associated with emotional/behavioural difficulties?

Epilepsy was significantly associated with greater internalising and externalising difficulties at all ages (effect sizes ranging from β = 0.33 to 0.63), except for internalising at age 11 years (see Table 2).

3.3. Do these associations change after adjusting for bullying victimisation?

Bullying victimisation was significantly associated with internalising and externalising difficulties in all models (effect sizes ranging from β = 0.11 to 0.63), with the exception of internalising at age 11 years. Epilepsy remained a significant predictor of externalising at age 7 years (β = 0.61; CI, 0.29–0.91; p < 0.001), externalising at age 11 years (β = 0.34; CI, 0.06–0.62; p = 0.01) and externalising at age 16 years (β = 0.42; CI; 0.17–0.67; p < 0.001) following adjustment for bullying victimisation. Epilepsy became a non-significant predictor of internalising at age 7 years (β = 0.31; CI, −0.01–0.63; p = 0.06) and internalising at age 16 years (β = 0.27; CI, −002–0.57; p = 0.07) following adjustment for bullying victimisation at age 7 years and 11 years, respectively (see Table 2).

3.4. Does bullying victimisation mediate these associations?

The estimated total causal effect of epilepsy on internalising difficulties at age 7 years (β = 0.43; CI, 0.03–0.85; p = 0.04) was consistent

Table 2
Linear regression models – estimates using propensity score matched sample (n = 715).

Model	Age 7			Age 11			Age 16		
	B	95% CI	P value	B	95% CI	P value	B	95% CI	P value
Epilepsy – bullying victimisation									
Epilepsy	0.20	[0.05, 0.35]	0.008**	0.30	[0.11, 0.48]	0.002**			
Epilepsy – internalising (a)									
Epilepsy	0.43	[0.11, 0.76]	0.01**	0.31	[-0.03, 0.65]	0.08	0.33	[0.03, 0.63]	0.03*
Epilepsy – internalising (b)									
Epilepsy	0.31	[-0.01, 0.63]	0.06				0.27	[-0.02, 0.57]	0.07
Bullying victimisation by age 7	0.63	[0.47, 0.79]	<0.001***						
Bullying victimisation by age 11							0.20	[0.09, 0.32]	<0.001***
Epilepsy – externalising (a)									
Epilepsy	0.63	[0.31, 0.94]	<0.001***	0.38	[0.10, 0.65]	0.01**	0.45	[0.20, 0.70]	<0.001***
Epilepsy – externalising (b)									
Epilepsy	0.61	[0.29, 0.91]	<0.001***	0.34	[0.06, 0.62]	0.01**	0.42	[0.17, 0.67]	<0.001***
Bullying victimisation	0.14	[-0.02, 0.30]	0.08	0.17	[0.03, 0.31]	0.02*			
Bullying victimisation by age 11							0.11	[0.01, 0.21]	0.03*

Note. B = unstandardised regression coefficient. CI = 95% confidence interval. Significance levels p ≤ 0.05*, p ≤ 0.01**, p ≤ 0.001***.

with those found in the initial analyses. The estimated indirect effect was 0.13 (CI, 0.02–0.24; p = 0.02), meaning the proportion of the association between epilepsy and internalising at age 7 years mediated by bullying victimisation was 29% (CI, −0.02–1.25; p = 0.05). For internalising difficulties at age 16 years, the estimated total causal effect of epilepsy was again consistent with initial analyses (β = 0.33; CI, 0.04–0.63; p = 0.02). The estimated indirect effect was 0.05 (CI, 0.01–0.12), meaning the proportion of the association mediated by bullying victimisation was 18% (CI, 0.03–0.99; p = 0.03) (see Table 3).

3.5. Sensitivity analysis

In the sensitivity analysis, 41 matched children were living with a chronic health condition other than epilepsy (6.3%) compared to 12 CWE (18.5%). No matched children were living with another neurological condition, compared to 9 CWE (13.8%). Results for objectives 1–3 were broadly consistent with main analyses, except the proportion of the association mediated by bullying victimisation on internalising at age 7 years (35%; CI, −0.69–1.85) fell slightly below statistical significance (p = 0.06) (Tables S2 and S3).

4. Discussion

This study aimed to investigate the longitudinal associations between epilepsy at age 7 years and emotional/behavioural difficulties at age 7, 11, and 16 years. It also investigated the extent to which bullying victimisation mediated the association between epilepsy and emotional/behavioural difficulties. Building on a small body of longitudinal research, in this UK birth cohort, we found evidence that epilepsy was associated with internalising and externalising difficulties at all ages, except for internalising difficulties at age 11 years. Epilepsy became a non-significant predictor for internalising difficulties at age 7 years and 16 years when controlling for bullying victimisation. In mediation analyses, bullying victimisation mediated 29% and 18% of the association between epilepsy and internalising difficulties at age 7 years and age 16 years, respectively.

Our results support existing evidence that childhood epilepsy is associated with greater emotional/behavioural difficulties than those experienced by healthy peers and children with other health conditions [36]. The non-significant association between epilepsy and internalising at age 11 years was unexpected. This finding may be explained by our study being underpowered, as all confidence intervals were large; however, we found consistent associations at ages 7 and 16 years in both main analyses and sensitivity analyses. Measurement factors may also have contributed. Internalising behaviours are often less readily observed than externalising behaviours, particularly in mid-childhood, and children's emotional symptoms often manifest differently depending on the context, both of which can reduce measurement sensitivity of parent-report measures at this age [68]. Beyond methodological factors, developmental changes in psychosocial risk may contribute to the different patterns observed. Epilepsy is associated with a range of neurodevelopmental comorbidities, including ADHD and ASD, and potential shared underlying vulnerabilities may contribute to heterogeneity in emotional and behavioural profiles across development [69]. Further, social-cognitive processes develop across the transition to adolescence, when peer relationships become particularly important [26]. Developmental shifts, including a temporary decline in cognitive empathy after mid-childhood, may influence how social challenges are experienced and potentially contribute to age-related variation in internalising difficulties [24]. Importantly overall, our findings found that childhood epilepsy is associated with both internalising and externalising difficulties, and that only some of these associations are mediated by the presence of bullying victimisation [38]. The heterogeneity in outcomes supports the suggestion that a range of biopsychosocial factors may be implicated in mental health outcomes for children living with epilepsy [3].

Table 3
Mediation analyses with bullying victimisation as mediator – estimates using propensity score matched sample (n = 715).

Model	Estimate (95% CI)	p-value
Epilepsy – internalising at age 7 years		
Total Effect	0.43 [0.03, 0.85]	0.04*
Direct Effect	0.31 [-0.07, 0.71]	0.12
Indirect Effect	0.13 [0.02, 0.24]	0.02*
Proportion Mediated	0.29 [-0.02, 1.25]	0.05*
Epilepsy – internalising at age 16 years		
Total Effect	0.33 [0.04, 0.63]	0.02*
Direct Effect	0.27 [-0.02, 0.57]	0.07*
Indirect Effect	0.05 [0.01, 0.12]	0.01**
Proportion Mediated	0.18 [0.03, 0.99]	0.03*

Note. CI = 95% confidence interval. Significance levels p ≤ 0.05*, p ≤ 0.01**, p ≤ 0.001***.

Our results extend existing findings by showing that that peer victimisation may partly explain the link between childhood epilepsy and internalising difficulties. We found no similar evidence for externalising difficulties. This discrepancy may reflect the high level of neurodevelopmental comorbidities in epilepsy, such as ADHD that may be more strongly associated with externalising behaviour [16]. It is also plausible that CWE are at higher risk of certain types of victimisation. More research is needed to understand the role of potential contributing factors such as minority status, disease severity and cognitive difficulties on bullying type and persistence [8,33,37], Johnson et al. [70], Johnson et al. [36,71]. Further, it may be that associations are bidirectional [8]. More studies are clearly needed to explore why victimisation may embed risk for internalising difficulties in CWE, including those that look at the association between types of victimisation and structural and functional brain outcomes [72,73].

Since it is a modifiable risk factor, public health strategies have focused on bullying victimisation, including the use of whole school interventions that have demonstrable efficacy in population samples [74–76]. However, psychosocial interventions targeting emotional and behavioural difficulties in CWE have typically prioritised individual-level approaches, such as psychoeducation, cognitive behavioural therapy (CBT) for mood and anxiety, and condition self-management. These interventions have been found to offer clear benefits, including improved epilepsy knowledge, coping strategies, self-efficacy, and quality of life, particularly through structured psychoeducation and CBT-based approaches such as mindfulness [20]. Group-based programmes such as the PIE (Psychosocial Intervention for Epilepsy) intervention have also demonstrated efficacy in enhancing young people's confidence in discussing their epilepsy with peers, promoting adaptive coping [24]. Such approaches may foster long-term resilience against emotional and behavioural difficulties by supporting identity development and confidence within peer relationships.

Our findings suggest that the impact of social context, particularly interpersonal victimisation, on psychosocial outcomes may have been overlooked. This highlights the need for multi-level public health interventions that combine individual and systemic approaches, situating psychosocial support within broader family and community networks rather than locating the effects of epilepsy within the young person alone [77]. Public awareness and attitudes towards epilepsy vary across cultural contexts [78], and treatment gaps highlight a clear need for stigma-reduction and policy-based awareness initiatives to reduce discrimination [79,80]. Education delivered within schools and communities may help address stigma, misconceptions, and perceived loss of autonomy, all of which may affect peer relationships and vulnerability to peer victimisation. Epilepsy nurse specialists are well placed to deliver education within schools [81], whilst including both peers and staff can promote confidence, safety, and social integration of CWE within school activities [82]. However, comprehensive epilepsy education guidelines are yet to be developed and implemented, with notable

training gaps in mainstream schoolsJohnson et al. [70], Johnson et al. [71]. The Italian League Against Epilepsy campaign, provides one example of a national initiative that aimed to disseminate knowledge at scale through mediums such as films and meetings with neurologists [83]. Involving parents in educational efforts and interventions may further support children's participation in school and peer activities, with implications for self-esteem, connectedness, and caregiver burden [84]. Informal peer and parent support spaces within schools or healthcare settings, alongside signposting to third-sector organisations, may also reduce isolation and strengthen social confidence [77].

4.1. Implications

The study has implications. The associations between specific epilepsy factors and victim status remain unclear, as are resilience factors [69]. Bullying thus needs to be routinely screened for in CWE along with mental health measures to ensure clinicians are aware of the psychosocial challenges facing their patients that could increase disease burden. Future research could explore whether bullying victimisation impacts other epilepsy-specific factors that children may be more willing to disclose or clinicians more likely to notice [85]. Bullying may also mediate associations between epilepsy and non-clinical psychosocial outcomes for which they are at higher risk, such as educational attainment. It is noteworthy that victim status in healthy children has been associated with school absenteeism and later bully-victim status, both of which have been found to be higher in CWE [86]. Importantly, epilepsy is not a social behaviour that directly elicits bullying but rather, living with epilepsy may generate vulnerabilities that increase susceptibility to victimisation within certain contexts. Future research could examine how epilepsy moderates the impact of bullying on emotional and behavioural difficulties, to extend findings from this mediation analysis. Another implication for future research is to compare findings with populations living with other biologically driven health conditions, to further demonstrate the relevance of our findings for CWE. Our findings thus lay the groundwork for more research to identify the risk factors for bullying victimisation and the modifiable resilience factors that are protective against its impact on wellbeing.

4.2. Strengths and limitations

Strengths include the use of a large cohort with minimal attrition in childhood surveys. The survey identified epilepsy status using medical records. We used validated scales to assess emotional/behavioural difficulties [47]. The use of propensity scores is considered stronger for controlling for confounders than covariate adjustments and increased the comparability of CWE to peers, thereby assuring confidence in the interpretation of findings. We matched with a 1:10 ratio to increase statistical power overall and reduce the risk of overmatching due to the relatively small number of CWE within the NCDS dataset. It is the first longitudinal study of the impact of bullying victimisation on emotional/behavioural difficulties in CWE and thus carries important research and clinical implications.

There are limitations. We did not adjust for all possible confounders to prevent overmatching and like any other observational study, our analysis may have suffered from residual confounding. The lack of longitudinal data for seizure history precluded us from controlling for symptom remission. Some analyses may have been underpowered due to the small number of CWE identified. Our measures for socioeconomic status were coarse and may not have captured other aspects of poverty-related disadvantage. Future studies could consider using contextual measures of socioeconomic status to better account for health disparities [57]. We used the same parent-report measure for both outcomes and mediating variables. Future studies could use more informants and more sensitive measures including self-report measures, which were not available here [85]. The nature of peer victimisation and prevalence of emotional difficulties may have evolved since data collection, which was in the 1960 s, particularly with the recent rise of social media and cyberbullying, and the Rutter scales used to measure for emotional and behavioural difficulties in the NCDS are no longer routinely used [50,87]. The internalising and externalising scales were comprised of select emotional and behavioural symptoms within these domains and accordingly, findings should be interpreted as reflecting elevations in these specific symptom clusters. Future studies could investigate different types of bullying and replicate findings using scales from current practice or other cohort studies, to parse out these associations.

5. Conclusions

In conclusion, this study found that childhood epilepsy is associated with greater emotional and behavioural difficulties from mid-childhood to adolescence. These associations remained strong even while controlling for confounders including sex, socioeconomic status, and gestational age, and when comparing to children living with other chronic health conditions. We found that the associations between childhood epilepsy and internalising difficulties at age 7 years and at age 16 years were no longer statistically significant after adjusting for bullying victimisation by age 7 years and age 11 years, respectively, and found that bullying victimisation was a part mediator for these associations. Targeting bullying victimisation deserves to be considered as a way to reduce the high level of emotional and behavioural comorbidities in children with epilepsy. Further attention towards bullying victimisation in this vulnerable group is warranted both as a plausible contributory factor for such comorbidities, and a possible preventative treatment target.

6. Data sharing agreement

National Child Development Study data, accessed through the Centre for Longitudinal Studies (CLS) at the UCL Social Research Institute. The data is available to registered researchers via the CLS and the UK Data Service. The Economic and Social Research Council funds the Centre for Longitudinal Studies (CLS) Resource Centre (ES/W013142/1) which provides core support for the CLS cohort studies and makes these data available. CLS does not bear any responsibility for the analysis or interpretation of these data by researchers. Relevant training on information governance has been attended by the investigators. The code used for analysis was made publicly available after publication: https://github.com/emmablundell/NCDSepilepsy_bullying_difficulties.git.

7. Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work the author EB used ChatGPT in order to support with writing code for statistical analysis on R. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the published article.

Ethics statement

The NCDS sweeps included in this study pre-date the establishment of formal ethics committees and were thus subject to internal ethics review only. Informal parental consent was sought via letters explaining the nature of the study; written consent was not obtainedCentre for Longitudinal Studies [88]. Ethics approval for secondary data analysis using the NCDS was granted by UCL Research Ethics Committee on 1st May 2023 (CEHP/2023/593).

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CRediT authorship contribution statement

Emma Blundell: Writing – review & editing, Writing – original draft, Visualization, Software, Project administration, Methodology, Formal analysis, Data curation, Conceptualization. **Vaughan Bell:** Writing – review & editing, Supervision, Conceptualization. **King-Chi Yau:** Writing – review & editing, Supervision, Methodology, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.yebeh.2026.110986.

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