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Maternal cannabis use disorder and offspring behavioral outcomes: findings from a linked data cohort study

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ABSTRACT

Few studies have explored the association between maternal gestational cannabis use and disruptive behavioural disorders (DBDs) in offspring, often relying on self-reported data and small samples. This study aimed to assess the relationship between maternal cannabis use disorder (CUD) during pregnancy and postpartum periods and the risk of disruptive behaviours in offspring. We conducted a population-based retrospective cohort study using linked health data from New South Wales, Australia, for live births between 2003 and 2005. Mothers with CUD were compared to those without, and the risk of DBDs in offspring was estimated. Both CUD and disruptive behavioural disorders were identified using the International Classification of Disease (ICD) codes. Generalised Linear Models (GLMs) with log-binomial regression were fitted to estimate disruptive behavioural disorder risk in children. Statistical significance was set at $p < 0.05$.

After adjusting for key confounders, this study revealed significantly higher risks of disruptive behavioural disorders in children of mothers with CUD during the antenatal [risk ratio (RR) = 3.56, 95 % CI 2.42–5.05], perinatal [RR = 3.55, 95 % CI 2.45–4.98], and postnatal [RR = 2.95, 95 % CI 1.23–6.16] periods compared to non-exposed counterparts. These findings underscore the importance of preconception, antenatal, and postnatal counselling on maternal cannabis use to mitigate neurobehavioral risks in children.

1. Introduction

Disruptive behaviour disorders (DBDs), including oppositional defiant disorders (ODDs) and conduct disorders (CDs), are the most common psychiatric diagnoses in children (Ayano et al., 2024; 2021; Murray et al., 2013; Tandon and Giedinghagen, 2017). The prevalence of DBDs in children and adolescents has significantly increased over the last few decades, with notable gender-specific variations, indicating male children have more than 2.5 times higher prevalence (Ayano et al., 2024; Erskine et al., 2013; Mohammadi et al., 2021; Polanczyk et al., 2015; Vasileva et al., 2021). In addition to genetic influences, environmental factors, including maternal cannabis use (Gjone and Stevenson, 1997; Glasheen et al., 2013; Gu et al., 2024; Latimer et al., 2012; Moore et al., 2023; Whitaker et al., 2006) experienced during pregnancy and in the postnatal periods are widely believed to contribute to the development of DBDs in children.

Cannabis use disorder (CUD) has been increasingly reported among women in the last two decades (Meinhofer et al., 2022), with the prevalence among pregnant women also rising, ranging from 1.01 % to 9.8 % worldwide (Bandoli et al., 2021; Brown et al., 2023; Koto et al., 2022; Lo et al., 2023; Wicken et al., 2022; Young-Wolff et al., 2019). In Australia, approximately one in five pregnant women use cannabis during pregnancy (S. J. Brown et al., 2016; Passey et al., 2014; Stephanie et al., 2016) and 0.3 to 0.7 % of pregnant women are hospitalised due to CUD (Betts et al., 2022; Oni et al., 2022), indicating that cannabis use has become a substantial public health concern.

To date, studies on the link between maternal cannabis use and DBDs in children are limited and have yielded conflicting results. While some studies indicate a link between in-utero cannabis exposure and behavioural problems in offspring, including externalizing/behavioural problems (De Genna et al., 2021; El Marroun et al., 2019; Paul et al., 2021; Sorkhou et al., 2024), CDs (Daha et al., 2020), ODDs (Brianna

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et al., 2023; Moore et al., 2023), aggressive behaviours (Marroun et al., 2011; Moore et al., 2023), and any DBDs (De Genna et al., 2022; McLemore and Richardson, 2016), others report null associations (Godleski et al., 2018; Goldschmidt et al., 2000; Larkby et al., 2011; O'Connell and Fried, 1991; Ruisch et al., 2018). Most of the existing studies are limited to antenatal cannabis exposures to examine its impact on childhood behavioural problems in children and adolescents (Moore et al., 2023; Paul et al., 2021). Additionally, many studies used symptom scales such as the child behavioural checklist (CBCL) to ascertain behavioural problems in offspring and cannabis use measured using self-report data instead of clinical diagnoses, which has potential limitations such as changes in the directions of the true associations between exposure and outcome of interest (Brener et al., 2003; Brown et al., 1992; Burns et al., 2006; Sujan et al., 2022; Young-Wolff et al., 2020). Also, most prior studies relied on relatively small sample sizes, which limited the power of the studies and, in turn, reduced the ability to adjust for key confounders such as socioeconomic index for areas (Boden et al., 2010; Mravčík et al., 2020), maternal smoking (Boden et al., 2010; D'Onofrio et al., 2010; Fergusson et al., 1993; Latimer et al., 2012), maternal psychiatric admissions (Ayano et al., 2021; Glasheen et al., 2013; Halligan et al., 2007; Latimer et al., 2012) and other substance use disorders. Therefore, the existing studies are limited in their ability to examine the risk of DBDs in offspring associated with maternal CUD exposures.

Using large linked administrative data with objectively measured exposure of interest, maternal CUD during antenatal and postnatal periods, and DBDs in children as an outcome (Chiandetti et al., 2017; Shi et al., 2024), this study aimed to examine the association between antenatal and postnatal CUD and the risk of DBDs in offspring.

We hypothesised that exposure to maternal CUD during pregnancy and postnatal periods would markedly increase the risk of DBDs in offspring compared to those non-exposed. The findings of this study could help strengthen preventive strategies and policies in the health sector by mitigating the risk of DBDs in children associated with maternal CUD.

2. Methods and materials

2.1. The study population and data sources

In this study, we used population-based linked administrative data from routinely collected health data sources in New South Wales (NSW), Australia. The cohort sample comprised mother-offspring pairs, including all live births from 01 January 2003 to 31 December 2005.

Mothers with CUD comprised the exposure group, while mothers without CUD served as the comparison group. Both groups were followed from pregnancy until their offspring's first birthdays to identify prenatal and postnatal hospital admissions with CUD diagnoses. Additionally, all offspring were monitored from birth through December 2018, up to age 15 years, to identify hospital admissions and outpatient visits with disruptive behavioural disorders (DBD) diagnoses.

The birth records in the Perinatal Data Collection (PDC) from 01 January 2003 to 31 December 2005 were linked with the Admitted Patients Data Collection (APDC) and outpatients visit (Mental Health Ambulatory data collection: MH-AMB-DC) records that encompass the diagnoses of psychiatric illness and mental health disorders due to substance use, including the principal and secondary diagnoses, spanning from 01 January 2003 to 31 December 2006. The Centre for Health Record Linkage (CHeReL), overseen by the NSW Ministry of Health, did the data linkage. The details of this cohort profile are published elsewhere (Tadesse et al., 2024).

2.2. Study variables

2.2.1. The outcome variable (DBD)

The outcome variable, DBD, was derived from two main data sources: hospital admissions (APDC) and hospital outpatient visits (AMB-MH-DC). DBD diagnoses in children included oppositional defiant disorders (ODDs) and conduct disorders (CDs), identified using ICD-10-AM criteria for mental and behavioural disorders. Conduct disorder diagnoses were identified with codes F91, F91.0, F91.2, F91.8, and F91.9, while ODD diagnoses were identified with code F91.3. Therefore, the overall classification of DBDs in children was derived using ICD codes F91 through F91.9.

2.2.2. Exposure: maternal CUD

Maternal CUDs were ascertained from APDC data sources using the ICD-10-AM diagnostic codes consistent with mental and behavioural disorders due to the use of cannabinoids (F12.0–F12.9). The diagnoses of maternal antenatal CUD were further categorised as occurring during the antenatal and postnatal periods. For maternal CUD diagnosed during pregnancy (yes/no); the episode of care indicating the diagnosis of CUD must fall between the date of conception and the date of delivery. The date of conception was estimated as the delivery date in weeks minus gestational age at delivery in weeks (Betts et al., 2022; Tadesse et al., 2024). By taking the antenatal exposure period as a reference, the postnatal exposure to CUD includes the time elapsed from birth to 12 months postnatally. Additionally, perinatal exposure to CUD encompasses exposures during antenatal and/or postnatal periods.

2.3. Covariates

Existing epidemiological studies have reported several factors associated with both maternal CUD and childhood behavioural disorders. We identified a range of confounding factors for our main analyses, including maternal admissions due to substance use other than CUD-such as alcohol use-, tobacco use-, opioid use-, and stimulant use- disorders (D'Onofrio et al., 2010; Knudsen et al., 2015; Latimer et al., 2012; Meinhofer et al., 2022), maternal psychiatric admissions (Ayano et al., 2021; Glasheen et al., 2013; Halligan et al., 2007; Latimer et al., 2012), maternal medical conditions and adverse birth outcomes such as prematurity at birth, low birth weight, parity, pre-existing and gestational diabetes and hypertension (Elgen et al., 2002; Hack et al., 2009; Hayatbakhsh et al., 2012; Riechi et al., 2011) and socioeconomic status (Boden et al., 2010; Murray and Farrington, 2010). We derive these covariates from three linked data sources: PDC, APDC, and outpatient/ambulatory data collection (MH-AMB-DC) datasets. Furthermore, socio-economic indexes for areas (SEIFA codes) were used by the Australian Bureau of Statistics to assess postcodes based on their relative economic advantage or disadvantage, categorised into quartiles (1st quartile - most disadvantaged and 4th quartile - least disadvantaged) (ABS, 2016).

2.4. Statistical analysis

The risks of DBDs in children associated with exposure to maternal CUD, including antenatal and postnatal periods, were modelled using generalised linear models (GLMs) fitted with a log-binomial regression to estimate relative risks (RRs) with 95 % confidence intervals (CIs) for each exposure period using R 4.3.2. These include socioeconomic status (SEIFA quartiles), maternal admissions for other substance use disorders (tobacco, sedatives, stimulants, opioids, and alcohol), psychiatric conditions (schizophrenia, depression, bipolar, and anxiety disorders), chronic medical conditions (diabetes and hypertension), parity, and child-specific factors such as sex, premature birth (PTB), low birth weight (LBW), small-for-gestational-age (SGA), 5 min APGAR, admission to NICU, and birth plurality (Model 2). Finally, we subsequently further adjusted Model 2 for maternal smoking during pregnancy (Model 3).

Furthermore, we conducted additional analyses to examine the association between maternal admissions due to substance use disorders other than CUD during pregnancy - specifically opioid, tobacco, and

alcohol use disorders and the risk of disruptive behaviour disorders (DBDs) in offspring after adjusting for antenatal cannabis use disorder (CUD) and other covariates. For all models, the risk ratios (RRs) with 95 % confidence intervals (CIs) were estimated and the significance level was declared at a P-value of less than 0.05.

2.5. Ethics

Ethics approval for this study, which used linked health administrative data, was granted by the Cancer Institute NSW, the NSW Population and Health Service Research Ethics Committee (HREC/18/CIPHS/22), and Curtin University Human Research Ethics Committee (HRE/2019/0601/01), ensuring compliance with the highest ethical standards. Data linkage was performed by the Centre for Health Record Linkage (CHeReL), which adheres to strict privacy and data governance practices by separating personal identifiers from de-identified health records. This process, supported by health record custodians and ethical bodies, enables researchers to conduct population-level, ethically approved studies in the public interest without individual consent, thereby minimising bias and safeguarding privacy.

3. Results

3.1. Cohort characteristics

As presented in Fig. 1, a total of 222604 mother-offspring pairs were included in the final complete-case analyses to examine the associations between maternal CUD and DBD risks in children. A total of 461 observations (0.21 %) had missing values for various covariates, which were excluded from the main analysis to perform a complete-case analysis (Fig. 1). In this study, 1319 (0.59 %) and 281 (0.13 %) of offspring of mothers were hospitalised due to CUD during pregnancy and postnatal periods, respectively.

In this study, 1003 children were diagnosed with DBDs. Of these children, 3.4 % and 0.8 % of children with DBDs were exposed to antenatal and postnatal CUD, respectively. Of total offspring diagnosed with DBDs, 30 % were from the most disadvantaged socioeconomic background, 31 % were exposed to maternal smoking during pregnancy, 9.7 % were preterm birth, 6.9 % were low birth weight, and 0.5 % had been exposed to antenatal tobacco use disorder, and 2.4 % were exposed to antenatal maternal depressive disorders. Additionally, male children were more likely to be diagnosed with DBDs than females (69.6 versus 30.4 %, $p < 0.001$) (Table 1).

3.2. Exposure to maternal CUD and risk of DBD in Offspring

We conducted separate analyses to examine the associations between CUD during the antenatal and postnatal periods and the risk of DBDs in offspring. We observed consistently significant associations between antenatal- [adjusted risk ratio (aRR) = 3.69 (95 % CI 2.53, 5.2)], perinatal- [aRR = 3.66 (95 % CI 2.54,5.22)], and postnatal- [aRR = 3.2 (95 % CI 1.33, 6.63)] -periods exposure to CUD and risk of DBDS in offspring, after adjusting for a range of important confounders (Model 2). Further, we noticed slightly attenuated risks of DBDs in offspring associated with exposure to antenatal CUD [aRR = 3.56 (95 % CI 2.42, 5.05)], perinatal CUD [aRR = 3.55 (95 % CI 2.45,4.98)], and postnatal CUD [aRR = 2.95 (95 % CI 1.23, 6.16)] when the subsequent models adjusted for maternal tobacco smoking (Model 3) (Table 2).

We also examined the overlaps between the antenatal and postnatal exposure window periods associated with diagnoses of CUD, and we observed a minimal overlap between the two exposure periods. Additionally, we noticed that the overlaps did not significantly influence the observed associations, with slightly attenuated estimates in the final model (Table S1). Furthermore, we conducted additional analyses to assess the associations between maternal admissions for other substance use disorders (opioid, tobacco, and alcohol use) during pregnancy and the risk of disruptive behavior disorders (DBDs) in offspring. These analyses adjusted for antenatal cannabis use disorder (CUD) and other covariates. Our findings indicate that while other maternal substance use disorders also contribute to the risk of DBDs, the strength of the association varies by substance". For example, offspring prenatally exposed to maternal opioid use disorder had a 2.96-fold higher risk of DBDs, even after adjusting for prenatal CUD and other factors (Table S2).

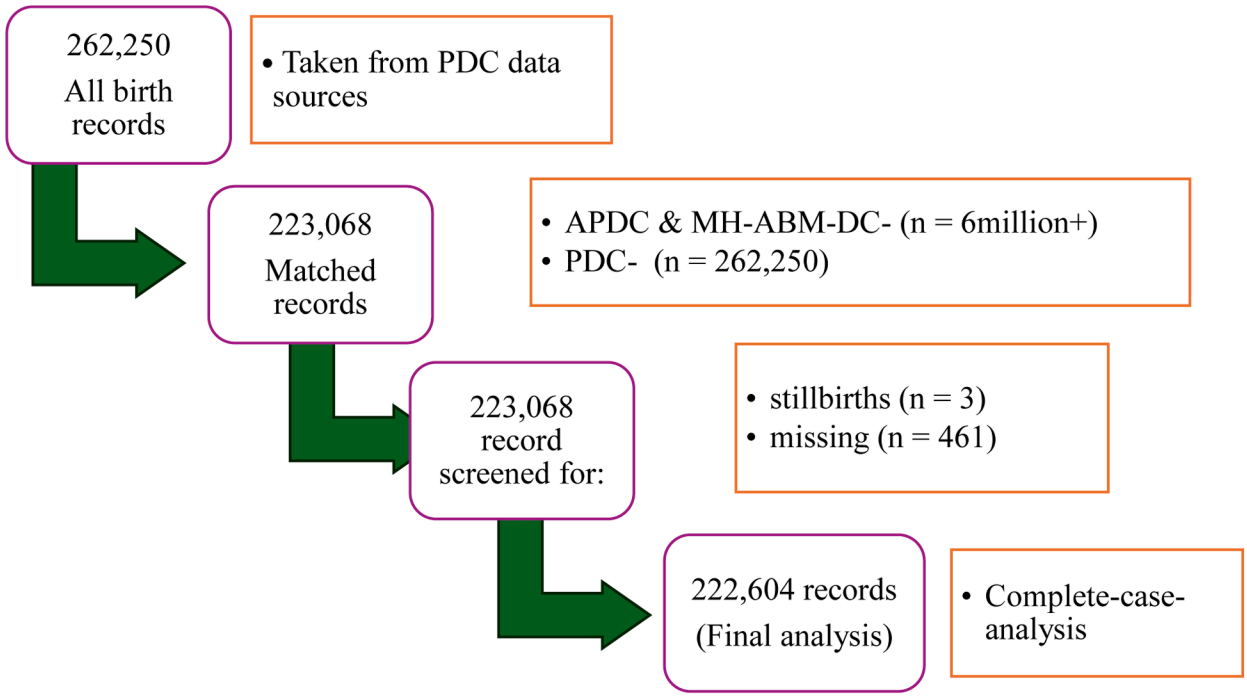

Fig. 1. The birth cohort profile from January 2003 to December 2005 in NSW state and observations included in our final analysis.

Table 1
Maternal demographic, obstetric, mental health, and neonatal factors by diagnosis of disruptive behavioural disorders (DBDs) in offspring ($N = 222,604$).

Potential predictors	Categories (if any)	Overall N (%)	DBDs in offspring		P-value
			Yes, N (%)	No, N (%)	
Maternal age (in years)	<20	8604 (3.9)	89 (8.9)	8515 (3.8)	<0.001
	20-24	31560 (14.2)	207 (20.6)	31353 (14.1)	
	25-29	61382 (27.6)	246 (24.5)	61136 (27.6)	
	30-34	75958 (34.1)	277 (27.6)	75681 (34.2)	
	35+	45100 (20.3)	184 (18.3)	44916 (20.3)	
Socioeconomic status (SEIFA quantile)	1st (most disadvantaged)	58983 (26.5)	301 (30.0)	58682 (26.5)	< 0.001
	2nd	48170 (21.6)	211(21.0)	47959 (21.6)	
	3rd	54715 (24.6)	239 (23.8)	54476 (24.6)	
	4th (least disadvantaged)	60736 (27.3)	252 (25.1)	60484 (27.3)	
Completed maternal education level	Degree & above	42224 (19.0)	241 (24.1)	41983 (19.0)	< 0.01
	Certificate	32903 (14.8)	131 (13.1)	32772 (14.8)	
	Diploma	56835 (25.5)	269 (26.8)	56566 (25.5)	
	Non-school qualifications	61400 (27.6)	185 (18.5)	61215 (27.6)	
	Unknown/not stated	29157 (13.1)	176 (17.6)	28981 (13.1)	
Maternal occupation	Managerial role	35018 (15.7)	117 (11.7)	34901 (15.8)	< 0.01
	Professional worker	64012 (28.8)	223 (22.3)	63789 (28.8)	
	Nonpaid worker	53298 (24.0)	297 (29.6)	53001 (23.9)	
	Tradeswoman	44567 (20.0)	172 (17.2)	44395 (20.0)	
	Unknown/not stated	25624 (11.5)	193 (19.3)	25431 (11.5)	
Child sex	Female	107751 (48.4)	305 (31.1)	107446 (99.7)	<0.001
	Male	114853 (51.6)	698 (69.6)	114155 (99.4)	
Low birth weight at birth (<2500 g)	Yes	11718 (5.3)	69 (6.9)	11649 (5.3)	0.026
	No	210886 (94.7)	934 (93.1)	209952 (94.7)	
Small-for-gestational age (<10th percentile)	Yes	3998 (1.8)	21 (2.1)	3977 (1.8)	0.554
	No	218606 (98.2)	982 (97.9)	217624 (98.2)	
Prematurity at birth (< 37 weeks)	Yes	13549 (6.1)	97 (9.7)	13452 (6.1)	<0.001
	No	209055 (93.9)	906 (90.3)	208149(93.9)	
5 min low APGAR (scores <7)	Yes	2523 (1.1)	18 (1.8)	2505 (1.1)	0.067
	No	220081 (98.9)	985 (98.2)	219096(98.9)	
Admitted to NICU	Yes	5425 (2.4)	48 (4.8)	5377 (2.4)	<0.001
	No	217179 (97.6)	955 (95.2)	216224(97.6)	
Birth plurality	Single	216056 (97.1)	976 (97.3)	215080(97.1)	0.707
	Twins or more	6548 (2.9)	27 (2.7)	6521 (2.9)	
Parity	Nil	92163 (41.4)	485 (48.4)	91678 (41.4)	<0.001
	1	76098 (34.2)	274 (27.3)	75824 (34.2)	
	2+	54343 (24.4)	244 (24.3)	54099 (24.4)	
Smoking in pregnancy	Yes	32110 (14.4)	311 (31.0)	31799 (14.3)	<0.001
	No	190494 (85.6)	692 (69.0)	189802(85.7)	
Pre-existing diabetes mellitus	Yes	1360 (0.6)	4 (0.4)	1356 (0.6)	0.540
	No	221244 (99.4)	999 (99.6)	220245(99.4)	
Gestational diabetes mellites (GDM)	Yes	10144 (4.6)	42 (4.2)	10102 (4.6)	0.627
	No	212460 (95.4)	961 (95.8)	211499(95.4)	
Pre-existing hypertension	Yes	2283 (1.0)	9 (0.9)	2274 (1.0)	0.761
	No	220321 (99.0)	9994 (99.1)	219327(99.0)	
Pregnancy-induced hypertension (PIH)	Yes	11789 (5.3)	70 (7.0)	11719 (5.3)	0.021
	No	210815 (94.7)	933 (93.0)	209882(94.7)	
Maternal mental health & substance use disorders					
Antenatal CUD	Yes	1319 (0.6)	34 (3.4)	1285 (0.6)	<0.001
	No	221285 (99.4)	969 (96.6)	220316(99.4)	
Postnatal CUD	Yes	281 (0.1)	8 (0.8)	273 (0.1)	<0.001
	No	222323 (99.9)	995 (99.2)	221328(99.9)	
Perinatal CUD diagnosis	Yes	1500 (0.7)	38 (3.8)	1462 (0.7)	<0.001
	No	221104 (99.3)	965 (96.6)	220139(99.3)	
Antenatal admission due to substance use other than CUD	Yes	1637 (0.7)	46 (4.6)	1591 (0.7)	<0.001
	No	220967 (99.3)	957 (95.4)	220010 (99.3)	
Antenatal psychiatric admissions	Yes	4271 (1.9)	66 (6.6)	4205 (1.9)	<0.001
	No	218333 (98.1)	937 (93.4)	217396(98.1)	
Postnatal admission due to substance use other than CUD	Yes	580 (0.3)	15 (1.5)	565 (0.3)	<0.001
	No	222024 (99.7)	988 (98.5)	221036 (99.7	
Postnatal psychiatric admissions	Yes	3185 (1.4)	43 (5.6)	2487 (1.1)	<0.001
	No	219419 (98.6)	947 (94.4)	218472(98.9)	

Keynotes: CUD- cannabis use disorder, SEIFA- Socio-economic indexes for areas, DBDs- Disruptive Behavioural Disorder. Maternal hospitalisation due to substance use other than CUD- admissions because of tobacco use-, alcohol use-, stimulant use-, opioid use-, or sedative use disorders. Maternal psychiatric admissions- hospitalisations due to schizophrenia, depression, anxiety, or bipolar disorders.

4. Discussion

4.1. Key findings

This study aimed to explore the association between maternal CUD during pregnancy and the postpartum period and the risk of DBDs in offspring. Additionally, we sought to determine whether adverse neonatal outcomes mediate this relationship.

Our findings suggest that children exposed to CUD during pregnancy and postnatal periods have more than three times higher risk of developing DBDs compared to their unexposed counterparts. These associations remained unaffected after adjusting for key confounders such as

Table 2
Risks of disruptive behavioural disorders (DBDs) in offspring associated with exposure to maternal CUD ($N = 222,604$).

Maternal predictors	DBDs in offspring					
	Model 1 RR (95 % CI)	P-value	Model 2, RR (95 % CI)	P-value	Model 3, RR (95 % CI)	P-value
Antenatal exposure to CUDa	5.89 (4.2, 8.25)	<0.001	3.69 (2.53, 5.2)	<0.001	3.56 (2.42, 5.05)	<0.001
Postnatal exposure to CUDb	6.36 (3.20, 12.64)	< 0.001	3.2 (1.33, 6.63)	< 0.01	2.95 (1.23, 6.16)	< 0.05
Perinatal exposure to CUDc	5.80 (4.14, 7.87)	< 0.001	3.66(2.54,5.22)	< 0.001	3.55 (2.45,4.98)	<0.001

Keynotes: RR- relative risks, CI- confidence intervals, CUD- cannabis use disorder, DBD- disruptive behavioural disorders
Model 1: Crude associations between maternal CUD and DBD risk in children.
Model 2a,c: Adjusted for maternal factors, including maternal age, socioeconomic status measured with SEIFA quartiles, parity, psychiatric admissions (i.e., schizophrenia, depression, anxiety and bipolar disorders), medical conditions (pre-existing and gestational DM and hypertension), maternal hospitalisations due to substance use disorders other than CUD (i.e., alcohol use-, stimulants use-, sedative use-, and opioids use disorders), and child factors: sex, PTB, LBW, SGA, 5 min low APGAR scores, and birth plurality.
Model 2b: In addition to the covariates included in Model 2, this model was further adjusted for adverse neonatal outcomes, including PTB, LBW, 5 min low APGAR scores, and required admission to the NICU.
Model 3a,b,c: Further adjusted for maternal tobacco smoking.

maternal smoking, maternal alcohol use disorder, socioeconomic status, child sex, birth plurality, and maternal psychopathology (i.e., depression, anxiety, schizophrenia, and bipolar disorders), suggesting that maternal CUD may be an independent risk for DBDs in children. These findings contribute to the existing evidence on the association between maternal cannabis use and behavioural problems in children and adolescents (Brianna F.Moore et al., 2023; Marroun et al., 2011; Paul et al., 2021). Our study addresses key gaps in previous research by examining the impact of antenatal, perinatal, and postnatal cannabis exposure on DBDs, while controlling for the mediating roles of adverse neonatal outcomes.

4.2. Interpretation of findings

Our findings are consistent with previous epidemiological studies that suggested exposure to maternal cannabis use was associated with a range of disruptive behavioural problems (i.e., conduct, aggressive, or oppositional/defiant behaviours) in children and adolescents (El Marroun et al., 2019; Marroun et al., 2011; McLemore and Richardson, 2016; Moore et al., 2023; Paul et al., 2021). For example, a 2021 population-based cohort study by Paul and colleagues reported that cannabis exposure, specifically occurring before and after maternal knowledge of pregnancy, was associated with an increased risk of externalising problems in children and adolescents (Paul et al., 2021).

However, other studies reported conflicting findings and no evidence of association (Godleski et al., 2018; Ruisch et al., 2018). For instance, a 2018 study by Ruisch and colleagues, which included three primary studies ($N = 56 – 636$), reported that antenatally exposed children have no increased risk of conduct problems compared to non-exposed children (Ruisch et al., 2018). It is plausible that those studies may not have been adequately powered to detect differences in childhood behavioural problems due to relatively small sample sizes (Godleski et al., 2018; Larkby et al., 2011), which limits the ability of studies to include more potential confounders in the main analysis, potentially obscuring true associations. For instance, it is important to note that no study has adjusted the influence of socioeconomic status and maternal psychopathology, which are the important confounders for the associations between maternal cannabis use and neurobehavioral problems in children and adolescents. Furthermore, while these previous studies have used screening tools (CBCL) to ascertain childhood behavioural problems, we used diagnostic tools (ICD-10-AM). Additionally, most of these prior studies predominantly relied on self-reported data to investigate the relationship between maternal cannabis use and the risk of behavioural problems in children. Self-reported data are prone to limitations such as under-reporting and recall bias (Brener et al., 2003; Brown et al., 1992), which may influence the observed associations. Further, while much of the existing literature studied the risk of gestational cannabis use, we have comprehensively examined the risks of exposure to maternal CUD at different exposure points (i.e., antenatal, perinatal, and postnatal) associated with DBDs in children.

4.3. Biologically plausible mechanisms

Several animal and human studies have investigated the neurobiological mechanisms by which antenatal cannabis exposure affects neurodevelopmental and neurobehavioral conditions in exposed children. Existing evidence suggests that cannabinoid ingredients, specially Δ9-tetrahydrocannabinol (THC), can pass through the placental and breast lactations (Ayonrinde et al., 2021; Blackard and Tennes, 1984; Little and VanBeveren, 1996), which has the potential to affect the expression of key genes for neural development, leading to neurotransmitter and neurobehavioral disturbances and brain development impairment (Houston et al., 2014; Narouze, 2021; Navarrete et al., 2020; Thomason et al., 2021). For example, a study by Thomason and colleagues suggested that in-utero exposure to cannabis is associated with variation in human brain hippocampal functional connectivity, which results in alterations in foetal dorsolateral, medial and superior frontal, insula, anterior temporal, and posterior cingulate connectivity (Thomason et al., 2021). Similarly, other studies from longitudinal studies reported that THC exposure during pregnancy might alter dopamine receptors (i.e., D1 and D2), which significantly influences neurodevelopmental outcomes (Jutras-Aswad et al., 2009), thereby impairing neurobehavioral outcomes in later life (Morris et al., 2011; Szutorisz and Hurd, 2018; Wu et al., 2011). Furthermore, an animal study by Natale et al. (2020) indicated that maternal exposure to Δ9-THC effectively compromised fetal growth, which provides a potential mechanism for the fetal growth restriction observed in rats who use cannabis during pregnancy. Thus, it is plausible that exposure to cannabis during pregnancy, perinatal, and postnatal periods could influence the fundamental brain developmental processes, thereby increasing the risk of behavioural disorders in exposed offspring.

Alternatively, although antenatal cannabis use is associated with higher environmental risks, it also involves some familial or polygenetic risk scores (PRSs) (Gu et al., 2024), which may increase the risk of behavioural disorders in children and adolescents. Nevertheless, our study did not provide direct evidence supporting the involvement of these mechanisms, while these observations are plausible.

4.4. Implication of the findings

The implications of these findings are significant for shaping health policies aimed at reducing cannabis use among pregnant and lactating women to lower neurobehavioral risks in offspring. This research enhances the existing literature by underscoring the association between maternal CUD and a heightened risk of DBDs in offspring. By establishing this link, the study underscores the need for targeted

interventions and public health strategies to mitigate these risks, ultimately improving long-term outcomes for children.

4.5. Strengths and limitations of the study

The present study has notable strengths. Firstly, we used a large sample size derived from population-based and clinical data with standardised and validated measurement tools to measure both the exposure of interest (CUD) and outcome of interest (DBDs), which are the main strengths of this study. Secondly, it stands out as the first study to examine the risk of disruptive behavioural disorders in the children of mothers with CUD across antenatal and postnatal periods using high-quality linked data. Thirdly, our study's adequate sample size enables the inclusion of more potential confounders in the main analysis, potentially enhancing the reliability of the observed associations. For example, prior research underscores the necessity of elucidating the intricate relationships between tobacco use, cannabis use, and neurodevelopmental and neurobehavioral outcomes, a complexity that our study thoroughly addresses (Rosen et al., 2015; von Ehrenstein et al., 2021).

This study recognises and acknowledges several limitations. It is important to note that gaps in data completeness will be a limitation when capturing DBD diagnoses and maternal CUD, even though we used linked health registries. Specifically, our study included only hospitalised mothers, potentially missing individuals with less severe maternal CUD who do not require hospitalisation. Additionally, despite efforts to include data from both inpatient and outpatient settings, some DBD diagnoses may still be under-documented or misclassified. The reliance on registry data introduces the potential for coding errors or diagnoses that may not align with standard clinical criteria. For example, while ICD-10-AM codes were used to classify disruptive behavior disorders (DBDs), including ODD and CD, diagnoses made at very young ages may not fully reflect the clinical consensus for these conditions. Furthermore, the small number of events stratified by CD and ODD limited our ability to conduct separate analyses for these subtypes. Consequently, we examined the impact of maternal cannabis use disorder (CUD) on the broader category of DBDs, which includes both CD and ODD, as identified through ICD-10-AM codes. While this approach enhanced statistical power, it also necessitates caution in interpreting findings related to specific DBD subtypes.

Furthermore, the lack of data on paternal substance use- and mental health-related disorders in this study suggests that paternal factors may also play a role in the observed associations. For instance, epidemiological studies reported that paternal characteristics can influence maternal cannabis use during pregnancy (Epstein et al., 2018; McLaughlin et al., 2012). Additionally, paternal behavioural factors have been linked to offspring's psychiatric, neurodevelopmental, and neurobehavioral outcomes (Kosty et al., 2015; Sujan et al., 2022; Xerxa et al., 2021). Thus, it is important to note that these unmeasured paternal factors may influence the observed associations.

Finally, we do not have data for the residual and familial confounders, such as genetics. Studies suggest that maternal cannabis use is associated with familial or polygenetic risk scores (PRSs) (PRSs) (Gu et al., 2024), which may increase the risk of behavioural disorders in children and adolescents. These imply that these factors might explain the observed associations. This suggests caution is warranted when interpreting and translating the observed associations. Additionally, future studies are required to address these limitations.

5. Conclusion

In our study, we found that exposure to maternal CUD during antenatal, perinatal, and postnatal periods consistently increases the risk of DBDs in children compared to non-exposed counterparts. This study adds to the body of evidence on the need for early screening of CUD for women who plan to give birth, are pregnant and are lactating to mitigate the risk of childhood behavioural disorders in their offspring. Future research should employ advanced causal inferential techniques to understand the observed associations.

CRediT authorship contribution statement

Abay Woday Tadesse: Writing – review & editing, Writing – original draft, Software, Methodology, Formal analysis, Data curation, Conceptualization. **Berihun Assefa Dachew:** Writing – review & editing, Supervision, Methodology. **Getinet Ayano:** Writing – review & editing, Supervision. **Kim Betts:** Writing – review & editing, Supervision. **Rosa Alati:** Writing – review & editing, Supervision.

Declaration of competing interest

All authors have no conflicts of interest to disclose.

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Supplementary materials

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