Journal of Psychosomatic Research 204 (2026) 112610

Contents lists available at ScienceDirect

Journal of Psychosomatic Research

journal homepage: www.elsevier.com/locate/jpsychores

Maternal pre- and postnatal depression and anxiety: Impacts on childhood asthma and its phenotypes

Eetu Kanerva [a,*], Minna Lukkarinen [b], Marika Leppänen [c], Bernd Pape [d], Päivi Rautava [e], Max Karukivi [a]

[a] Department of Adolescent Psychiatry, University of Turku, and Turku University Hospital, Turku, Finland
[b] The Department of Pediatrics and Adolescent Medicine, University of Turku and Turku University Hospital, Turku, Finland
[c] Preventive Medicine and Psychiatry, University of Turku and Turku University Hospital, Turku, Finland
[d] Department of Mathematics and Statistics, University of Vaasa, and Department of Biostatistics, Turku University and Turku University Hospital, Turku, Finland
[e] Department of Public Health, University of Turku and Turku Clinical Research Centre and Turku University Hospital, Turku, Finland

ARTICLE INFO

Keywords:
Asthma
Atopic asthma
Child
Maternal anxiety
Maternal depression
Non-atopic asthma

ABSTRACT

Background: Maternal psychological distress during pregnancy is known to elevate the risk of offspring asthma, but the impact of the timing of the distress remains poorly understood.
Objectives: To assess the individual and combined effects of maternal prenatal and postnatal depressive and anxiety disorders, both separately and longitudinally, on offspring asthma and its phenotypes.
Methods: Healthcare register data on 310,701 children born 2001–2006 and their 232,240 mothers were collected. Maternal depressive disorder was defined by diagnoses F30, F31, F32–F34 and F38 and anxiety disorder as F40–F42, F44–F45 and F48. Timing of disorder was defined as prenatal (from one year before until labor) and postnatal periods (from birth until three years postpartum). Child outcomes were overall asthma diagnosis J45–J46 at 7–12 years, further separated into allergic J45.0 and non-allergic J45.1 asthma phenotypes.
Results: Altogether, 19,000 (6.1%) children had asthma, 6517 (2.8%) mothers had depression, and 4189 (1.8%) had anxiety disorder. Child overall asthma was associated with maternal prenatal depression (adjusted odds ratio 1.28; 95% confidence interval 1.08–1.53) and anxiety disorders (1.30; 1.07–1.57), and with postnatal anxiety disorders (1.33; 1.15–1.54). Both maternal postnatal depression (1.36; 1.06–1.74) and anxiety disorders (1.45; 1.06–2.00) were associated with non-atopic asthma, and postnatal anxiety was associated with atopic asthma (1.34; 1.07–1.67). The comorbidity or longitudinality of maternal depressive and anxiety disorders didn't affect the associations.
Conclusion: Maternal depressive and anxiety disorders were associated with offspring asthma, varying by phenotype and timing. The postnatal effect was significant, suggesting independent associations and possibly distinct pathways in child respiratory morbidity.

1. Introduction

Childhood asthma is a globally common [1] disease that impairs a child's quality of life. [2] The evidence regarding the etiology of childhood asthma remains inconclusive, possibly due to the heterogeneity of the disease itself. [3] At the turn of the century, leading theories of allergen exposure [4] as a significant etiological factor for asthma were challenged [5] and during recent decades, several other theories regarding its multifactorial etiology have come into the spotlight. Environmental factors, such as sensitization to house dust mites and aeroallergens, seem to play a major role in asthma development. [6,7] Asthma has hereditary features [8,9] particularly through the maternal lineage. [10,11] In addition, growing evidence shows that family socioeconomic status, [12] parental educational level, [13] and residential neighborhood [14] could contribute to the development of childhood asthma. Furthermore, intrauterine programming predisposing to asthma may be a result of various prenatal factors. [15]

Wright et al. paved the way for theories linking maternal prenatal

Abbreviations: aOR, adjusted Odds Ratio; CI, Confidence Interval; MBR, Medical Birth Register; OR, odds ratio; SD, Standard deviation; SES, socioeconomic status; THL, the Finnish Institute for Health and Welfare..

* Corresponding author at: Department of Adolescent Psychiatry, Turku University Hospital, 20521 Turku, Finland.
E-mail address: eepkan@utu.fi (E. Kanerva).

https://doi.org/10.1016/j.jpsychores.2026.112610
Received 10 November 2025; Received in revised form 3 February 2026; Accepted 16 February 2026
Available online 27 February 2026

stress and mental health related development of childhood asthma. [16] Soon after, additional studies followed, highlighting an association between caregiver's stress and child's wheezing and asthma. [15,16] Interestingly, in one of the first cohort studies on the subject, Cookson et al. demonstrated a notable association between maternal prenatal anxiety symptoms and offspring asthma, particularly the non-atopic asthma phenotype. [17] Since then, subsequent register-based studies have reported similar findings regarding maternal depression and child's non-atopic asthma [18] with meta-analyses further supporting the association. [19–21]

Recently, the focus of prenatal etiological studies on the development and morbidity of childhood asthma has shifted toward identifying the critical developmental window - whether the exposure of maternal depression or anxiety during pregnancy or in early infancy has more pronounced effect. [22] The timing of this exposure has been shown to have mixed impacts. Prolonged exposure of depressive or anxiety disorders or symptoms from pregnancy through early childhood may have a stronger impact than exposure limited to either period. [17,23] In our recent study, we found that maternal mental health disorders occurring from one year before up to three years after childbirth were associated with childhood asthma and more prominently with the non-atopic asthma phenotype. [24] Our study results rose further questions regarding how the most prevalent maternal mental health disorders in our previous study, depressive and anxiety disorders, affect different asthma phenotypes, an area that has not yet been sufficiently studied.

Other forms of maternal psychological stress around pregnancy, such as negative life events, have shown similar associations with child's asthma as comparable to those of maternal depressive and anxiety symptoms. [17,25–28] Previous register-based studies examining the association between maternal depressive and anxiety disorders have often not included all the diagnoses given in the health system or assessed possible differences between asthma phenotypes. [23,29] In addition, earlier studies have primarily focused only on children not yet of school age [17,23,29,30] or relied mainly on questionnaire-based data or subjective parental reports of the disease, lacking studies including physician-given diagnoses. [17,21] Thus, due to the tendency of spontaneous remission of early childhood asthma, [31] a longer follow-up period was warranted in the present study.

To provide new insight into the effects of maternal depressive and anxiety disorders on child's respiratory morbidity, we utilized data from our large Finnish birth cohort and nationwide registers. Specifically, we aimed to examine the prenatal and postnatal timing, comorbidity and longitudinal patterns of these disorders. Our study aim was to investigate the associations between maternal prenatal and postnatal depressive and anxiety disorders with child's asthma and its phenotypes between ages 7 and 12. We hypothesized that maternal depressive and anxiety disorders would be associated with offspring asthma and particularly non-atopic asthma.

2. Methods

This is a register-based retrospective study of Finnish children born between January 1, 2001 and December 31, 2006 (N = 341,632) and their mothers (N = 232,240) (Fig. 1). Children with unknown gestational age (N = 1248), severe syndromes (N = 11,746), premature birth before 37 gestational weeks (N = 17,338), or perinatal death (N = 599) were excluded. No exclusion criteria were applied to mothers. Data were collected until 31.12.2022. The study protocol adhered to the principles of the Declaration of Helsinki and was approved by the Finnish Institute for Health and Welfare (THL/5954/14.06.00/2022) and the Ethics Committee of Turku University Hospital (J44/19). As this was a retrospective register-based study, informed consent from participants was not required, and they were not contacted. The legal basis for processing personal data is public interest and scientific research (EU General Data Protection Regulation 2016/679 (GDPR), Article(1)(e) and Article 9(2)(j); Data Protection Act, Sections 4 and 6).

2.1. Maternal depression and anxiety

Diagnosis codes for maternal depressive and anxiety disorders and asthma were collected from the Finnish Hospital Discharge Register. [32] The International Classification of Diseases (ICD) [33] is used in Finland; the 8th revision (ICD-8) from 1969, the 9th (ICD-9) from 1987 and the 10th (ICD-10) from 1996 onward (Fig. 1E, Supplementary data).

Mothers were classified as having a depressive disorder during exposure period if they were diagnosed with F30, F31, F32–F34 or F38, and anxiety with F40–F42, F44–45 and F48. The prenatal period included diagnosis codes set one year before child birth up to birth to

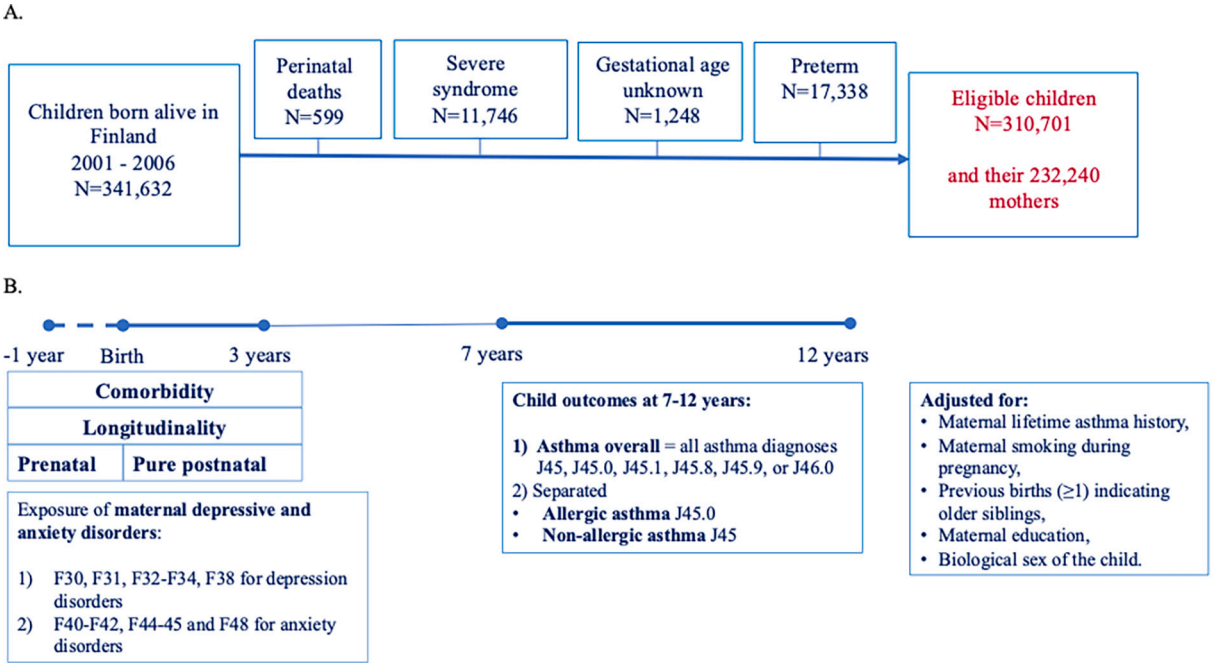

Fig. 1. A) Study flow chart and B) study timeline and setting.

include pregnancy. However, this could extend to the postnatal period as given the typical trajectories of these disorders and the limitations of register data, we could not rule out the possibility of postnatal effects in this group although the diagnosis was no longer observed. The postnatal period included new diagnoses from birth up to three years of child age, clarifying the group as purely postnatal as possible.

2.2. Offspring asthma

The outcome of child's asthma overall was met if they had received asthma diagnosis between ages 7 and 12 years. The ICD-10 codes for asthma were J45.0 for atopic, J45.1 for non-atopic, J45.8 for mixed, J45.9 for other or unspecified asthma, and J46 for status asthmaticus. To avoid multiple classifications, children were categorized according to their latest asthma diagnosis. Children with J46.0 as their latest asthma diagnosis were reclassified by their most recent diagnosis of asthma phenotype J45.0 or J45.1, and if none were recorded, they were categorized into the subgroup of J45.8 and J45.9. In addition, children diagnosed with J45 without a more specific phenotype were categorized as children with J46.0. In Finland, childhood asthma diagnosis is mainly set by pediatricians and is based on the national and international recommendations of typical asthma symptoms and lung function tests indicating bronchial hyperresponsiveness. [34]

2.3. Confounders

Confounding factors included previously established risk factors for childhood asthma such as maternal lifetime history of asthma, smoking during pregnancy, prior births as the proxy for older siblings, maternal occupational status as the proxy for maternal socioeconomical status (SES), and the biological sex of the child. [24] Data on the gestational age and sex of the child, maternal smoking during pregnancy and occupational status were obtained from the Medical Birth Register (MBR). [35] Maternal lifetime asthma was defined as having ever received an asthma diagnosis code of J45, J45.0, J45.1, J45.8 or J45.9 according to ICD-10, or 493.xx according to ICD-8 or ICD-9 during lifetime. Occupational status was originally retrieved from the MBR and subsequently classified into four classes using the International Standard Classification of Occupations. [36] Here, the group of upper white collar workers represented the highest occupational status and was used as the reference group. It was compared to lower white-collar workers, blue-collar workers and others (homemakers, students, pensioners), listed from second highest to the lowest group.

2.4. Statistics

Comparisons of background variables between asthmatic and non-asthmatic children, as well as between asthma phenotypes, were performed using *t*-tests for continuous variables and chi-square tests for categorical variables. The logistic regression analyses were used to study the associations between maternal depressive or anxiety disorders and offspring asthma overall. Regression analyses were repeated separately for the asthma phenotypes of atopic and non-atopic asthma and other asthma. All analyses were adjusted for maternal lifetime asthma history, smoking during pregnancy, prior births, maternal occupational status, and the biological sex of the child. Our previous study showed that maternal smoking, asthma history, lower occupational status and child's biological male sex were more prevalent in the asthmatic group, and thus these covariates were included in this study. [24]

First, to assess the effect of comorbidity between depression and anxiety, an interaction term for depression and anxiety was included in the analyses. These interaction terms were omitted from the final analyses, as they were not statistically significant in any of the models indicating that comorbidity between depression and anxiety did not alter the individual effect of either type of maternal psychiatric morbidity. Second, to assess the effect of longitudinal exposure, an interaction term was included in the analyses to capture the presence of depressive or anxiety disorder across pre- and postnatal periods. These interaction terms were removed from the final analyses due to non-significance, indicating that the presence or absence of depression or anxiety in one period, prenatal or postnatal, did not affect the presence or absence of the same condition in the other period.

Finally, to assess the impact of timing of exposure of maternal depression and anxiety, the prenatal and postnatal periods were analyzed separately. The prenatal period was operationally defined as the time frame from one year prior to childbirth up to the time of delivery; however, diagnostic coding from this period may have extended into the postnatal timeframe. The postnatal period included only diagnoses occurring from birth until the child's third birthday. Thus, the postnatal period was considered purely postnatal, representing new-onset disorders not diagnosed during the prenatal period. This approach helped to minimize collinearity between prenatal and postnatal maternal psychiatric morbidity and enabled us to study the impact of *de novo* postnatal exposure. Two-sided *p*-values below 0.05 were considered statistically significant. The analyses were carried out using SAS for Windows version 9.4 (SAS Institute Inc., Cary, NC, USA).

3. Results

3.1. Characteristics

Totally 310,70 children and their 232,240 mothers were eligible for the study (Fig. 1). Of the mothers, 6517 (2.8%) were diagnosed with depressive disorders, of whom 2002 (0.86%) prenatally and 4515 (1.94%) postnatally. Anxiety disorders were diagnosed in 4189 (1.8%), including 1680 (0.72%) prenatally and 2509 (1.1%) postnatally. Concomitant depressive and anxiety disorders were identified in 1607 (0.69%) mothers. Mothers with either depressive or anxiety disorder, or both, were more often diagnosed with asthma than those without depression or anxiety (Tables E1–E3 in Supplementary data).

In total, 19,000 (6.1%) children were diagnosed with asthma overall between 7 and 12 years of age (Table 1). Phenotypes were distributed as follows: 3196 (16.8%) had non-atopic asthma (J45.1), 7794 (41.0%) had atopic asthma (J45.0), and the rest, 8010 (42.2%) children, had other asthma as their latest asthma diagnosis. Children who had been exposed to maternal depressive or anxiety disorder either pre- or

Table 1
Characteristics, all children, non-asthmatics and asthmatics. *P*-value between non-asthmatics *vs.* asthmatics.

	All children (*N* = 310,701)	Non-asthmatics (*N* = 291,701/ 93.9%)	Asthmatics (*N* = 19,000/ 6.1%)	*p*-value[a]
Maternal prenatal depression: from one year before until labor, N (%)	2020 (0.65)	1843 (0.63)	177 (0.93)	**<0.001**
Maternal prenatal anxiety: from one year before until labor, N (%)	1690 (0.54)	1537 (0.53)	153 (0.81)	**<0.001**
Maternal postnatal depression: from childbirth until three years of age, N (%)	5231 (1.68)	4825 (1.65)	406 (2.14)	**<0.001**
Maternal postnatal anxiety: from childbirth until three years of age, N (%)	2962 (0.95)	2693 (0.93)	269 (1.42)	**<0.001**

Presented as numbers (percentages); [a]*p*-value between non-asthmatics and asthmatics. Chi-Square -test. *p*-values <0.05 in bold.

postnatally had more asthma compared to the non-exposed (Table 1). (See Table 2.)

3.2. Comorbidity and longitudinal exposure of maternal depressive and anxiety disorders

Interaction terms for the comorbidity of the maternal depressive and anxiety disorders were omitted from the final analyses, as they were not statistically significant (all $p > 0.05$; range 0.081–1.00) (Table E4 in Supplementary data). Similarly, the interaction terms for the longitudinal exposure of these disorders failed to reach statistical significance and were therefore excluded from the final analyses ($p > 0.05$; 0.41–0.93) (Table E5 in Supplementary data).

3.3. Effect of prenatal and postnatal maternal depressive and anxiety disorders on childhood asthma

Pre- and postnatal periods were analyzed separately to assess the effect of timing of exposure of maternal depression and anxiety.

In the unadjusted analyses, maternal prenatal depression was associated with offspring asthma overall and other asthma. Maternal prenatal anxiety was associated with similar degree to asthma overall, atopic asthma and other asthma. Maternal *de novo* postnatal depression was associated with all child asthma phenotypes and similarly, maternal *de novo* postnatal anxiety was associated with all asthma phenotypes (Table 3).

In the adjusted analyses, findings of maternal prenatal depression and anxiety remained rather similar. Maternal *de novo* postnatal depression disorders were associated with child's non-atopic asthma, whereas maternal *de novo* postnatal anxiety disorders were associated with child asthma overall, non-atopic asthma and atopic asthma in the adjusted analyses (Table 4).

4. Discussion

In this study, we found that both maternal depressive and anxiety disorders were associated with childhood asthma between 7 and 12. The novel finding was that the impact of exposure timing varied. Childhood asthma overall was associated with maternal depressive and anxiety disorders occurring prenatally, but with only anxiety disorders occurring in the postnatal period. Among the asthma phenotypes, non-atopic asthma was associated with maternal depressive and anxiety

Table 2
Characteristics of the population and offspring asthma between the age of 7–12.

	Atopic asthma (N = 7794/19,000, 41.0%)	Non-atopic asthma (N = 3196/19,000, 16.8%)	Other asthma (N = 8010/19,000, 42.2%)	p-value[a]
Maternal prenatal depression: from one year before until labor, N (%)	60 (0.77)	26 (0.81)	91 (1.14)	0.8134
Maternal prenatal anxiety: from one year before until labor, N (%)	57 (0.73)	24 (0.75)	72 (0.90)	0.9131
Maternal postnatal depression: from childbirth until three years of age, N (%)	162 (2.08)	82 (2.57)	162 (2.02)	0.1154
Maternal postnatal anxiety: from childbirth until three years of age, N (%)	107 (1.37)	53 (1.66)	109 (1.36)	0.2565

Presented as numbers (percentages); [a]p-value between non-atopic asthma and atopic asthma, Chi-Square -test. p-values <0.05 in bold.

Table 3
Risk of asthma at 7 to 12 years of age; unadjusted analyses.

	Asthma overall (OR, 95% CI)	Atopic asthma (OR, 95% CI)	Non-atopic asthma (OR, 95% CI)	Other asthma (OR, 95% CI)
Maternal prenatal depression: from one year before until labor	**1.48 (1.27–1.73)**	1.19 (0.92–1.54)	1.26 (0.85–1.85)	**1.79 (1.45–2.21)**
Maternal prenatal anxiety: from one year before until labor	**1.53 (1.30–1.81)**	**1.36 (1.04–1.77)**	1.39 (0.93–2.08)	**1.69 (1.33–2.14)**
Maternal postnatal depression: from childbirth until three years of age	**1.30 (1.17–1.44)**	**1.25 (1.07–1.46)**	**1.55 (1.24–1.93)**	**1.21 (1.04–1.42)**
Maternal postnatal anxiety: from childbirth until three years of age	**1.54 (1.36–1.75)**	**1.46 (1.21–1.78)**	**1.77 (1.34–2.32)**	**1.45 (1.20–1.76)**

OR, odds ratio; CI, confidence interval. Unadjusted results. All bold text under $p < 0.05$, tested with Wald Chi-Square.

Table 4
Risk of asthma at 7 to 12 years of age; adjusted analyses.

	Asthma overall (aOR, 95% CI)	Atopic asthma (aOR, 95% CI)	Non-atopic asthma (aOR, 95% CI)	Other asthma (aOR, 95% CI)
Maternal prenatal depression: from one year before until labor	**1.28 (1.08–1.53)**	1.08 (0.81–1.43)	0.88 (0.55–1.42)	**1.59 (1.26–2.01)**
Maternal prenatal anxiety: from one year before until labor	**1.30 (1.07–1.57)**	1.14 (0.84–1.55)	1.33 (0.85–2.07)	**1.37 (1.05–1.79)**
Maternal postnatal depression: from childbirth until three years of age	1.08 (0.96–1.22)	1.03 (0.85–1.23)	**1.36 (1.06–1.74)**	1.01 (0.84–1.21)
Maternal postnatal anxiety: from childbirth until three years of age	**1.33 (1.15–1.54)**	**1.34 (1.07–1.67)**	**1.45 (1.06–2.00)**	1.22 (0.98–1.53)

aOR, adjusted odds ratio; CI, confidence interval. All results were adjusted for maternal smoking during pregnancy, maternal asthma history, maternal occupational status by using the upper white-collar worker group as the reference group for other groups, maternal parity and child sex. $p < 0.05$ in bold text, tested with Wald Chi-Square.

disorders postnatally, but atopic asthma only with anxiety disorders. Other types of asthma were associated with both of the studied mental disorders in the prenatal period.

The novelties of our study are several. The effects of maternal depressive and anxiety disorders on childhood asthma at this age and accounting for different asthma phenotypes have not previously been examined to this extent. The independent impact of clearly defined *de novo* postnatal exposure for both disorders, especially on non-atopic asthma, raises important questions regarding disorder- and timing-specific pathophysiological mechanisms, and highlights the need for further research. Our findings complement the results of our previous study [24] and emphasize that both the type and timing of the maternal mental disorder are important in the context of childhood asthma.

Our results regarding the categorization of atopic and non-atopic asthma are in line with previous studies on maternal depression and anxiety that have also addressed asthma phenotypes. Cookson and colleagues (2009) noted that maternal prenatal anxiety symptoms continuing postnatally were associated with childhood non-atopic asthma. [17] However, no difference between maternal depression and anxiety symptoms were observed. Also, Ramratnam et al. (2021) showed that moderate-wheeze, low-atopy -phenotype in children was associated with maternal stress and depression during the first three years of life. [37] Brew and colleagues also reported that asthma was more prevalent among children exposed to cumulative maternal psychological distress from pre-conception through early childhood, which aligns with the results of our prenatally and postnatally exposed groups. [23] In their study, in contrast to our findings, prenatally occurring maternal depressive and anxiety disorders had the same effect if also occurring postnatally. This implicates that, in our study population, from the perspective of childhood asthma risk, if a child was exposed to maternal depression or anxiety one year before birth, the effect did not change if or if not the same exposure continued postnatally.

Maternal depressive and anxiety disorders each represent a distinct risk for childhood asthma, depending on the disorder type and the timing of the exposure. In contrast to previous studies finding no significant difference between depressive and anxiety disorders, [17,23] we observed differential associations. Specifically, when a child was first exposed to maternal depressive or anxiety disorders during the first three years of life, the risk was more prevalent and prone to non-atopic asthma phenotype. This finding highlights the possibility of different pathways in these associations.

In our study, atopic asthma, which generally considered to be more Th2-driven, lymphocyte- and eosinophil rich, and has been shown to be affected by maternal stress in previous literature, [3,38,39] was associated only with the postnatal maternal anxiety exposure. Non-atopic asthma, which is more Th1-mediated (or Th2-low), and characterized by neutrophil and macrophage involvement, [3,40] was significantly associated with both maternal depression and anxiety disorders occurring postnatally. Although both asthma phenotypes share similarities in their clinical presentation and at the cellular level, research on non-atopic asthma remains limited, and the pathophysiology of this subtype is not yet well characterized. [3,40–42]

There are few hypotheses for maternal psychological stress as a potential factor in the development childhood asthma. One proposed mechanism is that maternal depression and anxiety disorders during pregnancy and in early childhood may alter the function of hypothalamus-pituitary-adrenal (HPA) -axis, leading to dysregulated glucocorticoid secretion patterns that predispose offspring to asthma. [43] During the prenatal period, elevated stress hormone levels due to maternal depressive or anxiety disorders can interfere with placental regulation, allowing these hormones to cross the placenta and disrupt the fetal HPA-axis development and glucocorticoid balance, which may contribute to increased susceptibility to asthma in offspring. [44,45] These glucocorticoids may also modulate the immune system to be less sensitive to type 2 inflammatory responses, resulting in diminished allergic sensitization in the offspring. [46] Conversely, some studies have reported a more proallergic cytokine profile present in pregnant women experiencing depressive and anxiety symptoms, which could similarly affect the child's respiratory health. [47]

The first three years of life are also a critical period in child's biologic development. During this time, maternal psychological distress (*e.g.* depression or anxiety) can affect the relationship between the child and the caregiver, which is shown to lead to long-term alterations in cortisol levels and stress reactivity in the children, [48] thus possibly elevating the child's risk of developing asthma later in life. Maternal smoking during pregnancy, prior births, SES and the biological sex of the child had similar associations with the childhood asthma as already shown in our first study, therefore, they were not examined in further detail in this study. [24]

Regarding maternal depressive and anxiety disorders and their co-occurrence, these conditions have historically been classified as distinct disorders. [49] However, they frequently co-occur, and the perceptions of the diseases have evolved across the diagnostic manuals of DSM (III to IV) and ICD (9 to 10) during recent decades, and the evidence of some overlapping in symptoms, similar biological components and responses to treatment is observed in the disorders. [50,51] Despite these similarities, there are still notable differences between the disorders, such as recognizable neural signatures and genetic profiles for the disorders. [52,53] In our study, the statistically nonsignificant interaction between the two disorders supports the interpretation that they are separate disorders. Given the fact that in our study, diagnoses of the maternal mental health disorders were made in secondary healthcare by physicians, we consider the diagnostic assessments to be accurate and differential diagnoses appropriately conducted. Other psychiatric co-morbidities, such as schizophrenia or delusional disorders, were not assessed in detail, as the majority of the maternal mental health diagnoses in our data were depressive or anxiety disorders. [24] Nevertheless, more severe mental health disorders have also been associated with childhood asthma, as reported by Heuckendorff et al. [34]

This study has several strengths. First, our data utilize high-quality Finnish registers. Second, the dataset is large and comprehensive, covering all Finnish children born between 2001 and 2006 and their mothers. Third, the extensive sample size enabled us to integrate multiple registers in order to create multifactorial analyses. For example, the information of the occupational status was used as the proxy for the family socioeconomic status and was combined with health care data. Also, we were able to access primary health care diagnoses from year 2011 onward, which completed the asthma diagnosis data for children born in 2004–2006 and further supports the generalizability of our study. In addition to its breadth, the data are excessive also in terms of longitudinality, as it covers all of the register data of the children and their mothers from their inception to year 2022 (Fig. 1E, Supplementary data).

We also acknowledge the limitations. Maternal mental health diagnoses were primarily derived from secondary healthcare, which most likely resulted in an underestimation of the prevalence of maternal depression and anxiety disorders during the study period. The combined prevalence of these disorders in our study is 4.6% is lower than the reported 18–25% in prior studies. [54,55] Furthermore, diagnoses from the private sector were not available, which may have contributed to the lower observed prevalence. On the other hand, due to the register-based nature of the study, the identified cases of depressive and anxiety disorders clearly represent clinically significant disorders as the diagnoses were set in secondary health care. Comparable epidemiological studies have lacked physician-given diagnoses, [54,55] so it is plausible that the true prevalence of the maternal depressive and anxiety disorders in the pre- and postnatal periods lies between the estimates observed in our study and those reported in earlier studies. In children, the inaccuracy of asthma diagnoses may be due to possible differences in patient record systems that complicated the data extraction and human error when physicians set the diagnosis codes. The nonspecific asthma diagnosis of

J45 was recorded in 1375 children and represented 7,2% of all asthmatics, and may have included more specific asthma phenotypes that we were unable to account for. We speculate that this may have overestimated the significance of the group of "other asthma", thus possibly hiding a more marked association of other asthma phenotypes. In our study design, the children who were prenatally exposed to maternal depressive or anxiety disorders were not categorically excluded from the prenatal group if the mother was also diagnosed postnatally. Thus, the exposure of maternal prenatal disorders may have included children who were exposed to maternal pre- and postnatal disorders longitudinally. Noteworthy is that this approach reflects a clinically realistic scenario, as maternal perinatal mental health disorders tend to persist for several years. [56] We also recognize that some mothers diagnosed with postnatal mental health disorders may have experienced symptoms already during the prenatal period that did not yet meet diagnostic thresholds. Likewise, the control group may have included mothers whose depressive and anxiety disorders were mild, undiagnosed or untreated and therefore unrecorded. Because the data were diagnosis-based and pharmacy records were unavailable, we were unable to identify mothers or children without proper diagnosis but still having used or currently using medication for depressive or anxiety disorder or asthma. Paternal data were also unavailable for the present study.

In conclusion, this study provides new and detailed insights into the role of prenatal and postnatal periods in relation to the development of child's respiratory health. We demonstrate that the effects of maternal depressive and anxiety disorders on childhood asthma are independent of one another and with respect to timing and comorbidity. Our findings further support the association of prenatal depressive and anxiety disorders and childhood asthma. The novel finding was that *de novo* postnatal maternal depressive and anxiety disorders appeared to be more specifically associated with non-atopic asthma in children. These results emphasize the importance of clinical guidelines at family planning and health clinics that address maternal mental health prior and during pregnancy and in early childhood, considering their potential impact on the child's future respiratory health.

CRediT authorship contribution statement

Eetu Kanerva: Writing – review & editing, Writing – original draft, Visualization, Methodology, Investigation, Formal analysis, Conceptualization. **Minna Lukkarinen:** Writing – review & editing, Writing – original draft, Visualization, Methodology, Investigation, Conceptualization. **Marika Leppänen:** Writing – review & editing, Visualization, Investigation. **Bernd Pape:** Writing – review & editing, Formal analysis, Data curation. **Päivi Rautava:** Writing – review & editing, Project administration. **Max Karukivi:** Writing – review & editing, Visualization, Methodology, Investigation, Conceptualization.

Funding

The study was conducted independently of the funding sources, which had no role in its design, data collection, analysis or interpretation, execution, nor in the preparation, writing, review, approval or submission of the manuscript for publication. The funding sources among the authors were the following: the Ministry of Social Affairs and Health of Finland, Helsinki (Päivi Rautava, Marika Leppänen), State Funding for University Level Research of Health (Päivi Rautava), the Finnish State Grants for Clinical Research (Päivi Rautava), Signe and Ane Gyllenberg Foundation, Helsinki (Eetu Kanerva) and the Finnish Allergy, Skin and Asthma Foundation, Helsinki (Eetu Kanerva) - all in Finland.

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.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jpsychores.2026.112610.

References

[1] R. Guthold, E.W. Johansson, C.D. Mathers, D.A. Ross, Global and regional levels and trends of child and adolescent morbidity from 2000 to 2016: an analysis of years lost due to disability (YLDs), BMJ Glob. Health 6 (3) (Mar. 2021) e004996, https://doi.org/10.1136/bmjgh-2021-004996.
[2] D. Zhang, J. Zheng, The burden of childhood asthma by age group, 1990–2019: a systematic analysis of Global Burden of Disease 2019 data, Front. Pediatr. 10 (2022), https://doi.org/10.3389/fped.2022.823399. Accessed: July 28, 2023. [Online].
[3] M.E. Kuruvilla, F.E.-H. Lee, G.B. Lee, Understanding asthma phenotypes, endotypes, and mechanisms of disease, Clin Rev Allergy Immunol 56 (2) (Apr. 2019) 219–233, https://doi.org/10.1007/s12016-018-8712-1.
[4] T.A.E. Platts-Mills, G. Rakes, P.W. Heymann, The relevance of allergen exposure to the development of asthma in childhood, J. Allergy Clin. Immunol. 105 (2) (Feb. 2000) S503–S508, https://doi.org/10.1016/S0091-6749(00)90051-4.
[5] J. Douwes, N. Pearce, Asthma and the westernization "package", Int. J. Epidemiol. 31 (6) (Dec. 2002) 1098–1102, https://doi.org/10.1093/ije/31.6.1098.
[6] J.M. Gaffin, W. Kanchongkittiphon, W. Phipatanakul, Reprint of: perinatal and early childhood environmental factors influencing allergic asthma immunopathogenesis, Int. Immunopharmacol. 23 (1) (Nov. 2014) 337–346, https://doi.org/10.1016/j.intimp.2014.09.028.
[7] G.T. O'Connor, et al., Early-life home environment and risk of asthma among inner-city children, J. Allergy Clin. Immunol. 141 (4) (Apr. 2018) 1468–1475, https://doi.org/10.1016/j.jaci.2017.06.040.
[8] Z.W. El-Husseini, R. Gosens, F. Dekker, G.H. Koppelman, The genetics of asthma and the promise of genomics-guided drug target discovery, Lancet Respir. Med. 8 (10) (Oct. 2020) 1045–1056, https://doi.org/10.1016/S2213-2600(20)30363-5.
[9] E.M.S. Paaso, M.S. Jaakkola, T.K. Lajunen, T.T. Hugg, J.J.K. Jaakkola, The importance of family history in asthma during the first 27 years of life, Am. J. Respir. Crit. Care Med. 188 (5) (Sept. 2013) 624–626, https://doi.org/10.1164/rccm.201212-2236LE.
[10] R.H. Lim, L. Kobzik, M. Dahl, Risk for asthma in offspring of asthmatic mothers versus fathers: a meta-analysis, PloS One 5 (4) (Apr. 2010) e10134, https://doi.org/10.1371/journal.pone.0010134.
[11] C. Venter, et al., Incidence and timing of offspring asthma, wheeze, allergic rhinitis, atopic dermatitis, and food allergy and association with maternal history of asthma and allergic rhinitis, World Allergy Organ. J. 14 (3) (Mar. 2021), https://doi.org/10.1016/j.waojou.2021.100526.
[12] A. Zanobetti, et al., Childhood asthma incidence, early and persistent wheeze, and neighborhood socioeconomic factors in the ECHO/CREW consortium, JAMA Pediatr. 176 (8) (Aug. 2022) 759–767, https://doi.org/10.1001/jamapediatrics.2022.1446.
[13] E. Hafkamp-de Groen, et al., Socioeconomic and sociodemographic factors associated with asthma related outcomes in early childhood: the generation R study, PloS One 8 (11) (Nov. 2013) e78266, https://doi.org/10.1371/journal.pone.0078266.
[14] S. Teixeira, A. Zuberi, Neighborhood social and environmental factors and asthma among children living in low-income neighborhoods: the importance of informal social control, Fam. Community Health 41 (4) (2018) 214–224, https://doi.org/10.1097/FCH.0000000000000202.
[15] R.J. Wright, Stress and childhood asthma risk: overlapping evidence from animal studies and epidemiologic research, Allergy Asthma Clin. Immunol. Off. J. Can. Soc. Allergy Clin. Immunol. 4 (1) (Mar. 2008) 29–36, https://doi.org/10.1186/1710-1492-4-1-29.
[16] R.J. Wright, S. Cohen, V. Carey, S.T. Weiss, D.R. Gold, Parental stress as a predictor of wheezing in infancy, Am. J. Respir. Crit. Care Med. 165 (3) (Feb. 2002) 358–365, https://doi.org/10.1164/ajrccm.165.3.2102016.
[17] H. Cookson, R. Granell, C. Joinson, Y. Ben-Shlomo, A.J. Henderson, Mothers' anxiety during pregnancy is associated with asthma in their children, J. Allergy Clin. Immunol. 123 (4) (Apr. 2009) 847–853.e11, https://doi.org/10.1016/j.jaci.2009.01.042.
[18] X. Liu, J. Olsen, L.H. Pedersen, E. Agerbo, W. Yuan, J. Li, Antidepressant use during pregnancy and asthma in the offspring, Pediatrics 135 (4) (Apr. 2015) e911–e917, https://doi.org/10.1542/peds.2014-4073.
[19] K.F.E. van de Loo, M.M.H.J. van Gelder, J. Roukema, N. Roeleveld, P.J.F. M. Merkus, C.M. Verhaak, Prenatal maternal psychological stress and childhood asthma and wheezing: a meta-analysis, Eur. Respir. J. 47 (1) (Jan. 2016) 133–146, https://doi.org/10.1183/13993003.00299-2015.
[20] C. Flanigan, A. Sheikh, A. DunnGalvin, B.K. Brew, C. Almqvist, B.I. Nwaru, Prenatal maternal psychosocial stress and offspring's asthma and allergic disease: a systematic review and meta-analysis, Clin. Exp. Allergy 48 (4) (2018) 403–414, https://doi.org/10.1111/cea.13091.
[21] A.D. Rosa, Does maternal anxiety and depression increase the risk of asthma in the offspring? A systematic review and meta-analysis, Eur. Rev. Med. Pharmacol. Sci. 28 (3) (2024) 1066–1076, https://doi.org/10.26355/eurrev_202402_35343.

[22] M.J. Rosa, A.G. Lee, R.J. Wright, Evidence establishing a link between prenatal and early-life stress and asthma development, Curr. Opin. Allergy Clin. Immunol. 18 (2) (Apr. 2018) 148, https://doi.org/10.1097/ACI.0000000000000421.
[23] B.K. Brew, C. Lundholm, A. Viktorin, P. Lichtenstein, H. Larsson, C. Almqvist, Longitudinal depression or anxiety in mothers and offspring asthma: a Swedish population-based study, Int. J. Epidemiol. 47 (1) (Feb. 2018) 166–174, https://doi.org/10.1093/ije/dyx208.
[24] E.P. Kanerva, M.M. Lukkarinen, M.H. Leppänen, B.K. Pape, P.T.K. Rautava, M.R. J. Karukivi, Maternal mental health disorders associate with their child's asthma: a register-based study, Brain Behav. Immun. - Health 45 (May 2025) 100979, https://doi.org/10.1016/j.bbih.2025.100979.
[25] M.C. Magnus, et al., Association of maternal psychosocial stress with increased risk of asthma development in offspring, Am. J. Epidemiol. 187 (6) (June 2018) 1199–1209, https://doi.org/10.1093/aje/kwx366.
[26] A. Ruohomäki, et al., The impact of postpartum depressive symptoms on self-reported infant health and analgesic consumption at the age of 12 months: a prospective cohort study, J. Psychiatr. Res. 136 (Apr. 2021) 388–397, https://doi.org/10.1016/j.jpsychires.2021.02.036.
[27] E. Puosi, et al., Maternal prenatal psychological distress associates with offspring early-life wheezing – FinnBrain Birth Cohort, Pediatr. Allergy Immunol. 33 (1) (Jan. 2022) e13706, https://doi.org/10.1111/pai.13706.
[28] C.S. Alcala, et al., Longitudinal assessment of maternal depression and early childhood asthma and wheeze: effect modification by child sex, Pediatr. Pulmonol. 58 (1) (2023) 98–106, https://doi.org/10.1002/ppul.26164.
[29] S. Heuckendorff, M.N. Johansen, C. Overgaard, S.P. Johnsen, Y. Kelly, K. Fonager, Parental mental health, socioeconomic position and the risk of asthma in children—a nationwide Danish register study, Eur. J. Public Health 32 (1) (Dec. 2021) 14–20, https://doi.org/10.1093/eurpub/ckab205.
[30] A.P. van der Leek, et al., Maternal distress during pregnancy and recurrence in early childhood predicts atopic dermatitis and asthma in childhood, Chest 158 (1) (July 2020) 57–67, https://doi.org/10.1016/j.chest.2020.01.052.
[31] O. Oluwole, et al., The course of asthma: a population-based 10-year study examining asthma remission in children diagnosed with asthma in preschool, Pediatr. Pulmonol. 55 (8) (2020) 1924–1935, https://doi.org/10.1002/ppul.24881.
[32] R. Sund, Quality of the Finnish hospital discharge register: a systematic review, Scand. J. Public Health 40 (6) (Aug. 2012) 505–515, https://doi.org/10.1177/1403494812456637.
[33] World Health Organization, International Statistical Classification of Diseases and Related Health Problems, vol. 11th Edition, Available from, https://icd.who.int/, 2019.
[34] Asthma, Current Care Guidelines. Working Group Set up by the Finnish Medical Society Duodecim and the Finnish Cardiac Society, The Finnish Medical Society Duodecim, Helsinki, 2022 (accessed September 8, 2025). Available from, www.kaypahoito.fi.
[35] M. Gissler, J. Shelley, Quality of data on subsequent events in a routine medical birth register, Med. Inform. Internet Med. 27 (1) (Mar. 2002) 33–38, https://doi.org/10.1080/14639230110119234.
[36] A.C. Michalos., 'International Standard Classification of Occupations (ISCO)', in Encyclopedia of Quality of Life and Well-Being Research, vol. 2014, Springer, Dordecht, https://doi.org/10.1007/978-94-007-0753-5.
[37] S. Ramratnam, et al., Maternal stress and depression are associated with respiratory phenotypes in urban children, J. Allergy Clin. Immunol. 148 (1) (July 2021) 120–127, https://doi.org/10.1016/j.jaci.2021.03.005.
[38] R. Lim, A.V. Fedulov, L. Kobzik, Maternal stress during pregnancy increases neonatal allergy susceptibility: role of glucocorticoids, Am. J. Physiol. Lung Cell. Mol. Physiol. 307 (2) (July 2014) L141–L148, https://doi.org/10.1152/ajplung.00250.2013.
[39] B. Medsker, E. Forno, H. Simhan, J.C. Celedón, Prenatal stress, prematurity, and asthma, Obstet. Gynecol. Surv. 70 (12) (Dec. 2015) 773–779, https://doi.org/10.1097/OGX.0000000000000256.
[40] P. Pillai, C.J. Corrigan, S. Ying, Airway epithelium in atopic and nonatopic asthma: similarities and differences, ISRN Allergy Dec. 2011 (2011) 195846, https://doi.org/10.5402/2011/195846.
[41] L.A. Conrad, M.D. Cabana, D. Rastogi, Defining pediatric asthma: phenotypes to endotypes and beyond, Pediatr. Res. 90 (1) (July 2021) 45–51, https://doi.org/10.1038/s41390-020-01231-6.
[42] I.D. Pavord, et al., After asthma: redefining airways diseases, Lancet 391 (10118) (Jan. 2018) 350–400, https://doi.org/10.1016/S0140-6736(17)30879-6.
[43] V. Glover, T.G. O'Connor, K. O'Donnell, Prenatal stress and the programming of the HPA axis, Neurosci. Biobehav. Rev. 35 (1) (Sept. 2010) 17–22, https://doi.org/10.1016/j.neubiorev.2009.11.008.
[44] K. O'Donnell, T.G. O'Connor, V. Glover, Prenatal stress and neurodevelopment of the child: focus on the HPA axis and role of the placenta, Dev. Neurosci. 31 (4) (2009) 285–292, https://doi.org/10.1159/000216539.
[45] V. Glover, Prenatal stress and its effects on the fetus and the child: possible underlying biological mechanisms, Adv. Neurobiol. 10 (2015) 269–283, https://doi.org/10.1007/978-1-4939-1372-5_13.
[46] J.V. Fahy, Type 2 inflammation in asthma — present in most, absent in many, Nat. Rev. Immunol. 15 (1) (Jan. 2015) 57–65, https://doi.org/10.1038/nri3786.
[47] L. Karlsson, et al., Cytokine profile and maternal depression and anxiety symptoms in mid-pregnancy-the FinnBrain Birth Cohort Study, Arch. Womens Ment. Health 20 (1) (Feb. 2017) 39–48, https://doi.org/10.1007/s00737-016-0672-y.
[48] T.J. Barry, et al., Maternal postnatal depression predicts altered offspring biological stress reactivity in adulthood, Psychoneuroendocrinology 52 (Feb. 2015) 251–260, https://doi.org/10.1016/j.psyneuen.2014.12.003.
[49] K.S. Dobson, The relationship between anxiety and depression, Clin. Psychol. Rev. 5 (4) (1985) 307–324, https://doi.org/10.1016/0272-7358(85)90010-8.
[50] H.-J. Möller, B. Bandelow, H.-P. Volz, U.B. Barnikol, E. Seifritz, S. Kasper, The relevance of "mixed anxiety and depression" as a diagnostic category in clinical practice, Eur. Arch. Psychiatry Clin. Neurosci. 266 (8) (2016) 725–736, https://doi.org/10.1007/s00406-016-0684-7.
[51] R.C. Kessler, et al., Anxious and non-anxious major depressive disorder in the World Health Organization World Mental Health Surveys, Epidemiol. Psychiatr. Sci. 24 (3) (June 2015) 210–226, https://doi.org/10.1017/S2045796015000189.
[52] P. Zhukovsky, et al., Multiscale neural signatures of major depressive, anxiety, and stress-related disorders, Proc. Natl. Acad. Sci. U.S.A. 119 (23) (June 2022) e2204433119, https://doi.org/10.1073/pnas.2204433119.
[53] B.J. Coombes, et al., The genetic contribution to the comorbidity of depression and anxiety: a multi-site electronic health records study of almost 178 000 people, Psychol. Med. 53 (15) (Nov. 2023) 7368–7374, https://doi.org/10.1017/S0033291723000983.
[54] C.-L. Dennis, K. Falah-Hassani, R. Shiri, Prevalence of antenatal and postnatal anxiety: systematic review and meta-analysis, Br. J. Psychiatry 210 (5) (May 2017) 315–323, https://doi.org/10.1192/bjp.bp.116.187179.
[55] L. Underwood, K. Waldie, S. D'Souza, E.R. Peterson, S. Morton, A review of longitudinal studies on antenatal and postnatal depression, Arch. Womens Ment. Health 19 (5) (Oct. 2016) 711–720, https://doi.org/10.1007/s00737-016-0629-1.
[56] J.A. Dipietro, K.A. Costigan, H.L. Sipsma, Continuity in self-report measures of maternal anxiety, stress, and depressive symptoms from pregnancy through two years postpartum, J. Psychosom. Obstet. Gynaecol. 29 (2) (June 2008) 115–124, https://doi.org/10.1080/01674820701701546.