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Review

Effects of parental mental illness on children's physical health: systematic review and meta-analysis

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Background

Children of parents with mental disorder face multiple challenges.

Aims

To summarise evidence about parental mental disorder and child physical health.

Method

We searched seven databases for cohort or case–control studies quantifying associations between parental mental disorders (substance use, psychotic, mood, anxiety, obsessive–compulsive, post-traumatic stress and eating) and offspring physical health. Studies were excluded if: they reported perinatal outcomes only (<28 days) or outcomes after age 18; they measured outcome prior to exposure; or the sample was drawn from diseased children. A meta-analysis was conducted. The protocol was registered on the PROSPERO database (CRD42017072620).

Results

Searches revealed 15 945 non-duplicated studies. Forty-one studies met our inclusion criteria: ten investigated accidents/injuries; eight asthma; three other atopic diseases; ten overweight/obesity; ten studied other illnesses (eight from low-and middle-income countries (LMICs)). Half of the studies investigated maternal perinatal mental health, 17% investigated paternal mental disorder and 87% examined maternal depression. Meta-analysis revealed significantly higher rates of injuries (OR = 1.15, 95% CI 1.04–1.26), asthma (OR = 1.26, 95% CI 1.12–1.41) and outcomes recorded in LMICs (malnutrition: OR = 2.55, 95% CI 1.74–3.73; diarrhoea: OR = 2.16, 95% CI 1.65–2.84). Evidence was inconclusive for obesity and other atopic disorders.

Conclusions

Children of parents with mental disorder have health disadvantages; however, the evidence base is limited to risks for offspring following postnatal depression in mothers and there is little focus on fathers in the literature. Understanding the physical health risks of these vulnerable children is vital to improving lives. Future work should focus on discovering mechanisms linking physical and mental health across generations.

Declaration of interest

None.

Keywords

Epidemiology; childhood experience; depressive disorders; perinatal psychiatry; low- and middle-income countries.

Only recently has improving the lives of the 20% of children estimated to have a parent with a mental disorder[1–3] become a public health priority.[4–6] Thus, despite these children being likely to experience multiple deprivations and challenges, notably little is known about them or their health needs. Most of the information available about these children relates to their higher risk of developing psychiatric[7] and neurodevelopmental problems.[8–10] Prior research has shown that children of parents with mental disorder have a considerably increased risk of congenital anomalies,[11] of having been born with obstetric complications, including premature birth and low birthweight,[12] of being stillborn and of premature death (the last persists into adulthood).[13,14] However, little attention has been paid to a link between parental mental disorder and poorer offspring physical health, defined here as diseases such as asthma or diabetes affecting somatic rather than mental health. This is important not least because poor physical health has a detrimental effect on a child's development, with chronic ill health affecting social functioning[15] and academic progress.[16,17] Also, health disparities in childhood often persist into adulthood, leading to lower life expectancy.[18] Moreover, such a readily identifiable high-risk group could be a suitable target for early interventions.

This systematic review examined whether having a parent with a mental illness increases the risk of physical illness throughout childhood. Prior reviews have focused on maternal depression or anxiety and specific child health outcomes of obesity[19–21] and asthma.[22–24] Most of these included self-reported outcomes, cross-sectional designs and studies in which the childhood illness precedes exposure to the parental illness. Our review and meta-analysis included a broad range of parental mental disorders, specified *a priori*, and any clinically diagnosed child physical health outcome. We limited the scope of the review by using strict definitions of exposure and outcome and by pre-specifying the type of study design. This enabled us to summarise the literature on physical health of children whose parents have mental illness, focusing on studies of higher quality and with clearly defined measures.

Method

This review was developed according to the Centre for Reviews and Dissemination's 'Guidance for undertaking reviews in health care'.[25] The protocol was registered on the PROSPERO database (reference: CRD42017072620).

Selection criteria

Cohort or case–control studies that quantify associations between parental mental disorders and physical health in the offspring were included. This review focused on childhood disease; therefore, we excluded studies reporting outcomes occurring only in the neonatal period (0–28 days) or beyond age 18. Studies were also excluded if: the outcome was measured prior to exposure; the sample size was <10; or the cohort sample was drawn from diseased children. If more than one study had overlapping study populations

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and definitions of exposure and outcome, the study with the largest sample was chosen.

We specified *a priori* parental mental disorders that were of interest: substance use disorders (ICD-10 category: F10–19); schizophrenia, schizotypal and delusional disorders (F20–29); mood disorders (F30–39); anxiety disorders (F40–41); obsessive–compulsive disorders (F42); post-traumatic stress disorder (F43.1); and eating disorders (F50). We included studies where: mental illness was defined by a clinical diagnosis, using ICD or DSM criteria; mental illness was measured using a peer-reviewed instrument; or where the parent received treatment for a mental disorder.

We included studies reporting any physical disease in the offspring, clinically diagnosed and in the World Health Organizations' ICD-10 framework. This excluded psychological or neurobehavioural disorders (i.e. chapter V of ICD-10) and any disorder categorised in ICD-10 chapter VIII: 'Symptoms, signs and abnormal clinical and laboratory findings, not elsewhere classified'.

Search strategy

We searched the following databases for published papers, reports, conference abstracts and theses: MEDLINE; PsycINFO; PsycARTICLES; Embase; Web of Science; ProQuest ASSIA. We included studies published within the dates 1 January 1970 to the search date (21 September 2017), and did not initially include language restrictions.

We searched using the following general terms: (children) AND (parent) AND (mental illness) AND (physical disease) AND (case–control OR cohort study). We included terms for the specific mental illnesses and, for the purposes of conducting the search, an *a priori* list of common diseases of childhood that was developed by clinical collaborators (R.P. and K.M.A.). We included MESH terms and included variants and synonyms using truncations and wildcards where helpful. The full search strategy can be found in supplementary Appendix A available at https://doi.org/10.1192/bjp.2019.216. Following screening, we sought to identify further studies by hand-searching review papers arising from our searches. We also conducted hand-searches of reference lists of identified papers and we conducted a cited-reference search using Web of Science.

Screening

Duplicates were removed and then two reviewers (M.P. and A.K.) screened titles and abstracts, initially piloting 250 papers to ensure that consistent features were selected. All studies categorised as 'yes' or 'maybe' for inclusion were extracted for full text screening. A full-text screening tool was piloted by three reviewers (M.P., A.K. and J.G.) using 30 papers and then the same three reviewers divided all full-text papers between them so that each paper was screened twice. Disagreements and ambiguities were resolved in a group discussion, calling on a fourth reviewer (K.M.A.) as necessary. Lack of resources meant that we excluded papers that were not in English.

Data extraction and analysis

Data extraction was carried out by two reviewers (M.P. and A.K.), extracting data on: outcome type, exposure type; assessment instrument used; timing of exposure and outcome; sample size; study setting; statistical model; variables used in adjustment; and effect size. Study quality was assessed using the National Institutes of Health (NIH) Quality Assessment Tools using checklists developed for cohort and case–control studies.[26]

When studies used multiple adjusted models, we report from the model with the most covariates, unless we judged that any of the extra variables were on the causal pathway between exposure and outcome. When studies presented multiple effect sizes from multiple exposures we report only those exposures considered by the studies' authors to be the most severe and most chronic. All extracted effect sizes are presented in supplementary Appendix B. For meta-analysis, when a study reported multiple outcomes we selected the most frequent and when studies reported the effect of maternal and paternal exposure we selected estimates associated with maternal exposure.

For evidence synthesis, studies were grouped according to outcome type. For each grouping, estimates were converted into odds ratios (where possible) and pooled odds ratios were estimated using random-effects meta-analyses. Between-study heterogeneity was estimated using the I^2 statistic. Two sensitivity analyses were conducted. First, we determined the robustness of the meta-analyses to removing studies that were ranked poor quality. Second, where appropriate we compared estimates by type of study (case–control versus cohort design). Analyses were done using Stata 14 for Windows using the *metan* command.

Results

The database searches yielded 15 945 non-duplicate studies (Fig. 1). Of these, 251 were considered for full-text screening and 221 were excluded (94 failed the outcome criteria; 37 failed the exposure criteria; 36 were non-relevant study designs; 12 were reviews; 12 selected a diseased-only cohort; 7 were in a language other than English; 7 did not report sufficient data; 6 were outside the age range; 4 had exposure subsequent to disease; 2 were nested within a larger study; and we were unable to locate the full text for 4). This resulted in 30 studies for inclusion. After additional searches, a further 11 studies were included (5 from other reviews, 5 from reference lists of included papers and 1 from the cited reference search), giving a final total of 41 studies.

Overview of included studies

The vast majority of included studies (31/41) investigated exposure to parental depression; 7 investigated anxiety, 5 substance misuse and 1 psychotic disorder (Table 1). One study defined exposure as 'psychiatric morbidity', another as 'common mental disorder' and another considered the effect of post-traumatic stress disorder. None investigated the effect of parental eating, bipolar or obsessive–compulsive disorders. All studies examined maternal mental disorder; 17% (7/41) also examined exposure to paternal mental disorder. Forty-two percent of studies (17/41) measured exposure only during the perinatal period, defined here as from the start of pregnancy to 1 year after the birth. The median age at the last follow-up was 5 years (interquartile range IQR = 2–7.5) and the median sample size was 1696 (IQR = 294–12 618).

The majority (26) were prospective cohort studies; 8 were retrospective cohort studies and 7 were case–control studies. Thirty-nine percent (16) came from Europe (half of which were from Scandinavia) and 29% (12) were from North America. Sixty-eight percent (28) were carried out after 2010 and the earliest study was from 1981.

Studies were grouped into the categories according to their outcomes: accidents and injuries ($n = 10$); asthma ($n = 8$); other atopic diseases ($n = 3$); overweight and obesity ($n = 10$). The remaining studies were split between those from a low- and middle-income country (LMIC) setting ($n = 8$) (mainly consisting of studies examining diarrhoea or malnutrition) and those from a high-income country setting ($n = 2$).

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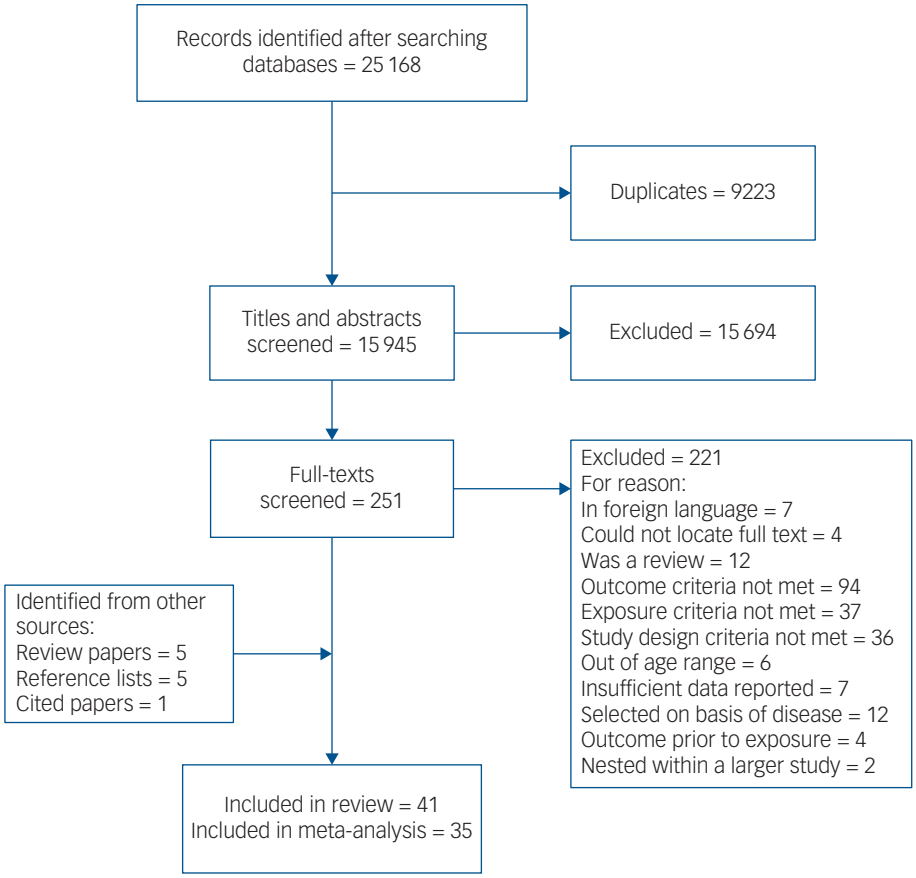

Fig. 1 Flow diagram for studies included in systematic review.

Accidents or injuries (ten studies)

Of the ten studies that reported accidents and injuries,[27–36] nine[28–36] and a combined sample of 314 132 children were included in the pooled analysis for accidents and injuries (Fig. 2). This revealed a 15% increase in the likelihood of a child having an accident or injury if they were exposed to parental mental illness (OR_{pool} = 1.15, 95% CI 1.04–1.26, I^2 = 76.4%).

Five of the six studies that considered the risk of offspring accidents or injuries associated with maternal depression found a positive association[27–31] and one study, which was the smallest and the only one to examine paternal as well as maternal depression, did not find an effect[32] (Table 2). Most studies examined outcomes in the first 5 years; however, one study of a birth cohort of 1265 found an effect of maternal depression on traumatic brain injuries up to age 15.[28]

Three studies examined parental substance use disorder and injuries.[33–35] One reported a doubling in the risk of traumatic brain injury if either parent had misused alcohol[33] and another small study (n = 125) estimated a similar effect of maternal substance misuse,[34] although the lower confidence interval included a null effect. A large retrospective cohort study from Finland (n = 113 813) did not find an effect of maternal or paternal substance misuse on risk of injuries in the first 6 years.[35]

One study examined accidents in the first year of life for 199 children with maternal psychotic disorder matched to 787 children without.[36] They did not find an increased risk of accidents associated with maternal exposure.

Asthma (eight studies)

From eight studies[37–44] and a combined sample of 450 202 children, we estimated a 19% increase in the odds of childhood asthma for children exposed to parental mental illness (OR_{pool} = 1.19, 95% CI 1.08–1.32, I^2 = 77.0%).

Six of the eight studies found a positive association between maternal depression or anxiety and childhood asthma.[37–41] The remaining two studies also had effect sizes indicating a positive relationship, albeit with confidence intervals that include a null effect.[42,43] Three of these studies also reported on exposure to paternal mental disorder[39–41] but only one detected an effect.[40] Those that investigated short-term versus chronic depression found a greater effect of the latter.[38,44] One study that looked at depression exposure during pregnancy versus exposure postnatally did not find a difference.[40]

Other atopic diseases (three studies)

In the pooled analysis, using three studies[45–47] and 23 471 children, there was inconclusive evidence for an association between parental mental illness and childhood atopy (OR_{pool} = 1.36, 95% CI 0.91–2.03, I^2 = 92.9%).

However, a large cohort study from Taiwan reported a positive effect of maternal depression on risk of infant eczema in the first 6 months of life.[45] One study reported an association between atopic dermatitis and maternal anxiety, but not maternal depression[46] and another reported an association with maternal (but not paternal) depression for inhalant (but not food) allergies.[47]

Overweight or obesity (ten studies)

The pooled analysis for the effect of parental mental illness and being overweight or obese in childhood included seven studies[50–54,56–57] and 36 309 children. To facilitate pooling, overweight was selected in the meta-analysis. The pooled estimate showed borderline

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Table 1 Descriptive summary of the 41 studies included in the systematic review

Variable	*n* (%)
Study design	
Case–control study	7 (17.1)
Cohort study	34 (82.9)
Prospective	26 (63.4)
Retrospective	8 (19.5)
Origin of sample	
Africa	4 (9.8)
Australasia	2 (4.9)
Asia	4 (9.8)
Europe	16 (39.0)
North America	12 (29.3)
South America	3 (7.3)
Year study published	
Before 2000	2 (4.9)
2000–2004	4 (9.8)
2005–2009	7 (17.1)
2010–2014	15 (36.6)
After 2015	13 (31.7)
Outcome	
Accidents/injuries	10 (24.4)
Asthma	8 (19.5)
Other atopic diseases	3 (7.3)
Overweight/obesity	10 (24.4)
Malnutrition	5 (12.2)
Diarrhoea	3 (7.3)
Other	2 (4.9)
Mental disorder[a]	
Depression	31 (75.6)
Anxiety	7 (17.1)
Substance misuse	5 (12.2)
Other	4 (9.8)
Parent in whom mental disorder was measured[a]	
Mothers	41 (100.0)
Fathers	7 (17.1)

a. These categories not mutually exclusive.

evidence to conclude a positive association ($OR_{pool} = 1.16$, 95% CI 0.97–1.39, $I^2 = 55.0\%$). The three studies unsuitable for pooling reported equivocal results.[48,49,55]

Of the nine studies that examined childhood obesity and its association with maternal depression or anxiety, one small cohort study ($n = 160$) estimated a positive association,[48] seven were equivocal[49–55] and one reported a negative effect.[56] Of the three studies that examined duration of maternal depressive disorder, two found an increased risk associated with cumulative exposure to depression, but not of exposure to periodic depression.[51,54] Two large prospective cohort studies, based in The Netherlands and the USA,[52,53] indicated an effect of maternal depression on childhood overweight in their unadjusted analyses, but when adjusted for potential confounders (including maternal/paternal body mass index) this effect disappeared.

One Finnish study of 4525 teenagers that examined the association between parental harmful drinking and offspring obesity did not find an effect.[57]

Other studies from low- and middle-income countries (eight studies)

Five studies from LMICs with a combined sample of 851 children investigated the effect of maternal mental illness on childhood malnutrition.[58–62] The pooled risk of malnutrition was more than double for exposed compared with unexposed children ($OR_{pool} = 2.55$, 95% CI 1.74–3.73, $I^2 = 0.0\%$).

Three studies and 13 430 children were pooled to investigate the link between perinatal maternal depression and diarrhoea or gastrointestinal infection and, similarly, the odds of diarrhoea doubled for exposed children ($OR_{pool} = 2.16$, 95% CI 1.65–2.84, $I^2 = 52.0\%$).[63–65]

Other studies from high-income countries (two studies)

One retrospective cohort study of 107 587 children in the UK reported an association between maternal perinatal depression and offspring gastrointestinal infection and respiratory tract infections.[66] Another retrospective cohort study of 2552 children of mothers with alcohol or substance use disorder in Finland found a marginal effect on diseases of the eye, ear and mastoid process.[67]

Study quality

Overall, 10 studies were graded 'good', 19 studies were 'fair' and 12 studies were graded 'poor' (supplementary Appendix C). Particular methodological problems were the lack of clarity regarding sample selection and the measurements used, and overlapping timing of exposure and outcome. Removing studies that were graded 'poor' (supplementary Appendix D) made little difference to the pooled estimates. Also, for the two meta-analyses that included case–control studies, the results were consistent by type of study design (supplementary Appendix E).

Discussion

Summary of findings

For the first time, this systematic review summarises current evidence on risk for poor physical health in offspring of parents with mental disorder. Overall, this detailed evidence synthesis paints a picture of relatively poor physical health in the children of parents with mental disorder, with pooled effect estimates revealing an increased risk of injuries, asthma, malnutrition and diarrhoea. However, we highlight striking gaps in the evidence: over three-quarters of the studies focused exclusively on maternal depression and, of those, half on postnatal depression; there is little information about children of parents with mental disorders other than depression or anxiety; and few studies investigated the impact of paternal mental disorder.

In total, 63% of studies (26/41) reported an effect of parental mental disorder on physical health outcomes in the offspring. A further 17% of studies (7/41) estimated a positive association, albeit with 95% confidence intervals that include a null effect. Most of the identified studies examined the relationship between exposure to parental mental illness (predominantly maternal postnatal depression) and risk of childhood injuries ($n = 10$), obesity ($n = 10$) or asthma ($n = 8$). Of note, no eligible studies assessed risk of childhood cancers, diabetes, epilepsy or migraine and only one assessed the impact of serious mental disorders such as schizophrenia or bipolar disorder on childhood physical health. Similarly, no studies assessed effects of dual diagnosis, maternal or paternal personality disorder or eating disorders.

Research in context

We identified evidence of an increased risk of childhood accidents or injuries associated with parental mental illness ($OR_{pool} = 1.15$, 95% CI 1.04–1.26).[27–35] Self-reported data have shown that periods of depression affect a mother's ability to supervise her child.[68] Substance misuse, particularly alcohol dependence, is associated with violent behaviour,[69] which may confer additional risk of injury to the child. The one study that examined the effect of psychosis on risk of childhood accidents did not find an effect, although the confidence intervals indicate that it was underpowered to detect

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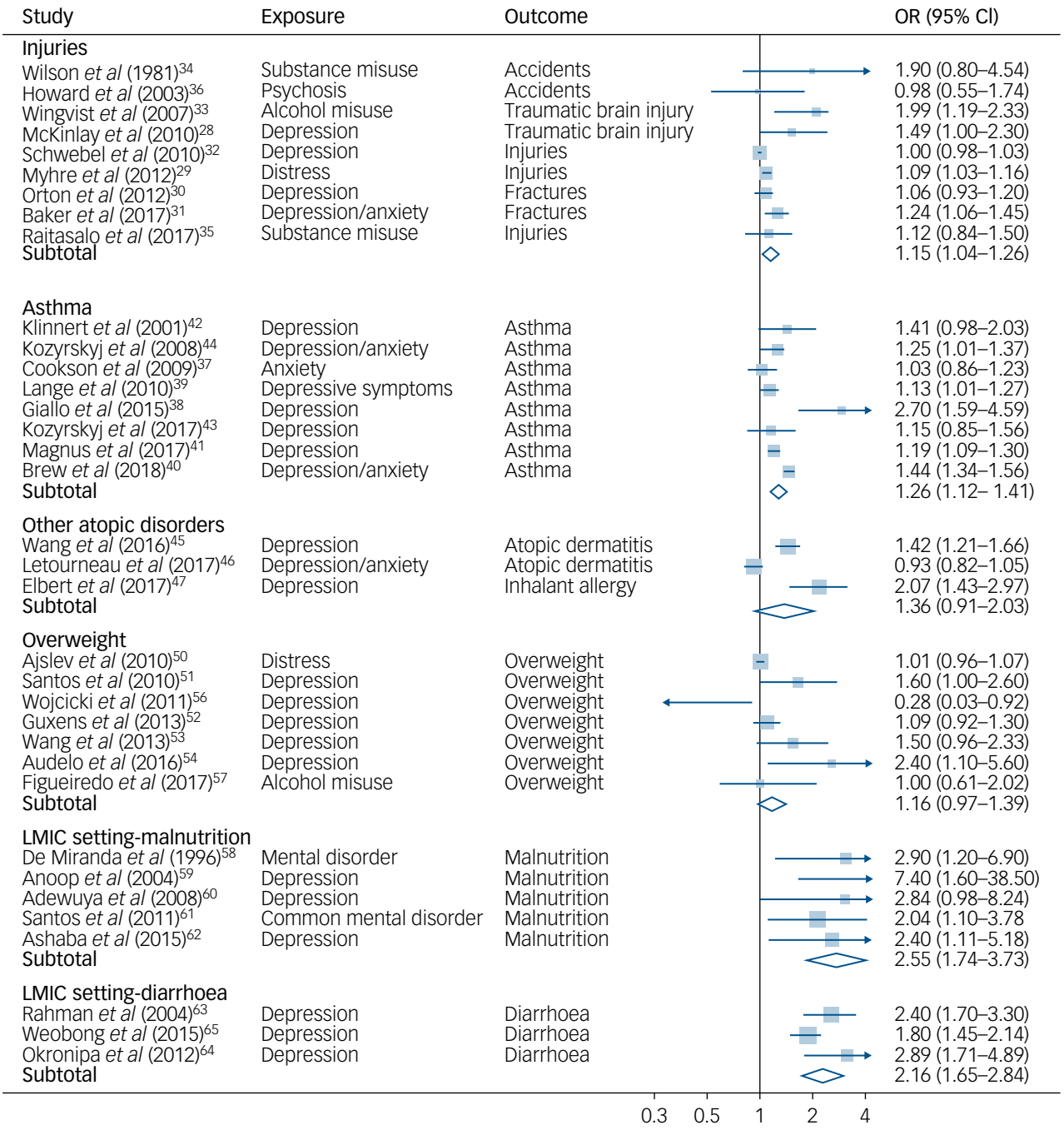

Fig. 2 Forest plot displaying estimates and pooled estimates of the effect of childhood exposure to parental mental illness on childhood disease.

less than a doubling in the rate of accidents.[36] All but two of these studies on accident and injury followed children in the first 6 years of life and therefore we do not know whether the risk is ameliorated by school entrance.

We highlight evidence to of an increased risk of childhood asthma associated with parental mental illness ($OR_{pool} = 1.26$, 95% CI 1.12–1.26) and some evidence to suggest an increased risk of other atopic disorders ($OR_{pool} = 1.36$, 95% CI 0.91–2.03). The association was observed most strongly in studies in which the exposure was categorised as severe[37] or chronic.[40,45] Prior research has reported psychosis and atopic disorders clustering in individuals[70,71] and families.[71] The familial link between mental and atopic disorders could arise as a result of shared aetiological factors but also as a result of the effects of adversity. Parental mental disorder increases risks for a range of adversities during childhood, such as poverty[72,73] and trauma.[74] Exposure to adversity and stress can alter a child's immune response,[75] which in turn increases the risk for atopy.[76] Yet, the link between mental disorders and atopy is likely complex and might include combinations of direct and indirect effects of parental disorder, as well as shared environmental factors (such as smoking).

The evidence for a link between parental mental illness and childhood overweight or obesity is inconclusive. Prior reviews that included cross-sectional studies did report a correlation between maternal depression or anxiety and childhood obesity;[19,21] another that included only prospective designs found

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Table 2 Summary of analyses from studies included in the systematic review

Study	Design, country, *n*	Exposure (age measured)	Outcome (age measured)	Effect measure: estimate (95% CI)
Accidents				
Wilson 1981[34]	Prosp. matched cohort, USA, 125	Substance misuse (8 wks gest. to birth)	Accidents (0–1 y)	OR: 1.90 (0.80–4.54)
Howard 2003[36]	Retro. matched cohort, UK, 986	Psychosis (2 y prior to birth)	Accidents (0–1 y)	OR: 0.98 (0.55–1.74)
Phelan 2007[27]	Prosp. cohort, USA, 1106	Unit increase in depression score (0–6 y)	Injuries (0–6 y)	OR: 1.04 (1.01–1.08)
Winqvist 2007[33]	Retro. cohort Finland, 12 058	Maternal or paternal alcohol misuse (0–14 y)	Traumatic brain injury (0–14 y)	aRR: 1.99 (1.19–2.33)
McKinlay 2010[28]	Prosp. cohort, NZ, 1265	Depression (6–13 y)	Traumatic brain injury (0–15 y)	HR: 1.49 (1.0–2.3)
Schwebel 2010[32]	Prosp. cohort, USA, 584	Depression (5–6 y)	Injuries (7–11 y)	OR Maternal: 1.00 (0.98–1.03) Paternal: 1.00 (0.97–1.03)
Myhre 2012[29]	Prosp. cohort, Norway, 26 087	Distress (1.5 y)	Injuries (1.5–3 y)	OR: 1.09 (1.03–1.16)
Orton 2012[30]	Matched nested case–control study, UK, 104 512	Depression (pregnancy to 6 m)	Types of injuries (0–5 y)	aOR Poisonings: 1.45 (1.24–1.70) OR Fractures: 1.06 (0.93–1.20) aOR Thermal injuries: 1.15 (1.02–1.32)
Baker 2017[31]	Retro. cohort, UK, 54 702	Depression or anxiety (6 m before pregnancy to 5 y)	Types of injuries (0–5 y)	aIRR Poisonings: 2.30 (1.93–2.75) Fractures: 1.24 (1.06–1.44) Burns: 1.53 (1.29–1.81) Serious injuries: 0.95 (0.60–1.50)
Raitasalo 2017[35]	Retro. cohort, Finland, 113 813	Substance misuse (4 y before birth to 6 y)	Injuries (0–6 y)	aOR Maternal: 1.12 (0.84–1.50) Paternal: 1.03 (0.86–1.23)
Asthma				
Klinnert 2001[42]	Prosp. cohort, USA, 133	Depression (6 y)	Asthma (6–8 y)	OR: 1.41 (0.98–2.03)
Kozyrskyj 2008[44]	Retro. cohort, Canada, 13 907	Long-term mood or anxiety disorder (0–6 y)	Asthma (7 y)	aOR: 1.25 (1.01–1.37)
Cookson 2009[37]	Prosp. cohort, UK, 5810	Anxiety (32 wks pregnancy and 8 wks)	Asthma (7.5 y)	aOR 32 wks: 1.03 (0.86–1.23) 8 wks: 1.12 (0.84–1.50)
Lange 2010[39]	Prosp. twin cohort, Puerto Rico, 339 (pairs)	Depression symptoms (0–1 y)	Asthma (3 y)	aOR: 1.13 (1.01–1.27)
Giallo 2015[38]	Prosp. cohort, Australia, 4165	Persistent and high depression symptoms (3 m to 7 y)	Asthma (6–7 y)	aOR: 2.70 (1.59–4.59)
Kozyrskyj 2017[43]	Prosp. cohort, Canada, 1696	Depression (0–18 m)	Asthma (10 y)	aOR: 1.15 (0.85–1.56)
Magnus 2017[41]	Prosp. cohort, Norway, 63 626	Major depression (18 wks pregnancy)	Asthma (7 y)	aOR Maternal: 1.19 (1.09–1.30) Paternal: 0.95 (0.81–1.11)
Brew 2018[40]	Retro. cohort, Sweden, 360 526	Depression or anxiety (continuously through preconception, pregnancy and postnatally)	Asthma (5 y)	aOR Maternal: 1.44 (1.34–1.56) Paternal: 1.11 (1.01–1.21)
Other atopic diseases				
Wang 2016[45]	Prosp. cohort, Taiwan, 18 024	Depression (6 m)	Atopic dermatitis (3 y)	aOR: 1.42 (1.21–1.66)
Letourneau 2017[46]	Prosp. cohort, Canada, 242	Depression or anxiety (3 m)	Atopic dermatitis (18 m)	aOR: 0.93 (0.82–1.05)
Elbert 2017[47]	Prosp. cohort, Netherlands, 5205	Depression (2nd trimester and 3 y)	Allergy (9–10 y)	aOR Maternal/inhalant: 2.07 (1.43–2.97) Maternal/food: 0.75 (0.29–0.97) Paternal/inhalant: 1.58 (0.89–2.80) Paternal/food: 0.87 (0.18–4.14)
Obesity/overweight				
Bronte-Tinkew 2007[49]	Prosp. cohort, USA, 8693	Depression (9 m)	Overweight (2 y)	Path coefficient: 0.025, $P = 0.29$
De Sousa 2009[48]	Case–control, India, 160	Depression score (mean: 9.53 y)	Obesity	Weight, mean (s.d.), kg: Normal 3.69 (2.95), Obese: 7.75 (4.31), $P = 0.29$
Ajslev 2010[50]	Prosp. cohort, Denmark, 21 121	Distress (6 m)	Overweight (7 y)	aOR: 1.01 (0.96–1.07)
Santos 2010[51]	Prosp. cohort, Brazil, 3792	Depression (3, 12, 24, 48 m)	Overweight (0–4 y)	aOR: 1.6 (1.0–2.6)
Wojcicki 2011[56]	Prosp. cohort, US, 166	Depression (2nd trimester and 4–6 wks)	Overweight (6 m– 2 y)	aOR: 0.28 (0.03–0.92)

(*Continued*)

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Table 2 (*Continued*)

Study	Design, country, *n*	Exposure (age measured)	Outcome (age measured)	Effect measure: estimate (95% CI)
Guxens 2013[52]	Prosp. cohort, Netherlands, 5283	Depression (20 wks)	Overweight (3 m–4 y)	aOR Maternal: 1.09 (0.92–1.30) Paternal: 0.71 (0.46–1.09)
Wang 2013[53]	Prosp. cohort, USA, 1090	Depression (1, 24 and 36 m)	Overweight (24 m–1 2 y)	aOR: 1.50 (0.96–2.33)
Audelo 2016[54]	Prosp. cohort, USA, 332	Depression (1, 3.5 and 7 y	Obesity (7 y)	aOR: 2.4 (1.1–5.6)
Blanco 2017[55]	Matched case–control, Spain, 100	Depression score (8–12 y)	Obesity (8 –12 y)	Weight, mean (s.d.), kg: Normal 7.28 (5.57), Obese: 9.56 (8.11), $P = 0.086$
Figueiredo 2017[57]	Prosp. cohort, Finland, 4525	Alcohol misuse (9–14 y)	Overweight (9–14 y)	aRR: 1.00 (0.81–1.23)
Other studies from low and middle-income countries				
De Miranda 1996[58]	Case–control, Brazil, 105	Mental disorder (0–2 y)	malnutrition (0–2 y)	aOR 2.9 (1.2 –6.9)
Anoop 2004[59]	Matched case–control, India, 144	Depression (1 m)	Malnutrition (6–12 m)	aOR: 7.4 (1.6–38.5)
Rahman 2004[63]	Prosp. cohort, Pakistan, 320	Depression (3rd trimester and 2, 6, and 12 m)	Diarrhoea	OR 2.4 (1.7–3.3)
Adewuya 2008[60]	Prosp. matched cohort, Nigeria, 142	Depression (6 m)	Malnutrition (9 m)	OR: 2.84 (0.98–8.24)
Santos 2011[61]	Matched case–control, Brazil, 294	Common mental disorder (0–5 y)	Malnutrition (0–5 y)	aOR: 2.04 (1.10–3.78)
Okronipa 2012[64]	Prosp. cohort, Ghana, 492	Depression (0–50 days)	Diarrhoea (0–3 m)	aOR: 2.89 (1.71–4.89)
Ashaba 2015[62]	Matched case–control, Uganda, 166	Depression (1–5 y)	Malnutrition (1–5 y)	OR: 2.4 (1.11–5.18)
Weobong 2015[65]	Prosp. cohort, Ghana, 12 618	Depression (4–12 wks)	Diarrhoea (0 –1 y)	aOR: 1.80 (1.45–2.14)
Other studies from high-income countries				
Ban 2010[66]	Retro. cohort, UK, 107 587	Depression (pregnancy to 6 m)	Infection (0–4 y)	RR: GI 1.40 (1.37–1.42) LRTI 1.27 (1.22–1.32)
Sarkola 2011[67]	Retro. matched cohort, Finland, 2552	Alcohol or substance misuse (pregnancy)	Diseases of the eye, ear or mastoid process (0–5 y)	OR: 1.34 (1.03–1.73)

Prosp., prospective; retro., retrospective; gest., gestation; a, adjusted analyses; HR, hazard ratio; IRR, incidence rate ratio; OR, odds ratio; RR, risk ratio; m, month; y, year; wks, weeks; GI, gastrointestinal infection; LRTI, lower respiratory tract infection.
a. From the most severe and chronic exposures reported in each study. Other relevant analyses are reported in supplementary Appendix B.

a link with chronic, but not with episodic (mostly postnatal), maternal depression.[20] All of the studies we identified that investigated this relationship used prospective cohort or case–control designs, which generally had smaller samples than studies that used registry-based cohorts. Therefore, the lack of definitive findings may be due to lack of power of individual studies.

All the studies from LMICs reported a positive association between maternal depression and offspring malnutrition or diarrhoea. Half of these exclusively examined the effect of postnatal depression in the first year after birth. Recent reviews have reported rates of maternal postnatal depression in LMICs of around 20%,[77] considerably higher than the 6.5–12.9% seen in affluent Western populations.[78] Evidence also suggests that maternal mental ill health is associated with both poor fetal and child growth.[79,80] Therefore, maternal mental health has been highlighted as a priority target for screening mothers in these settings.[81]

Strengths and limitations

The review highlights how children of parents with mental illness have multiple physical health challenges, on top of previously identified mortality[13,85] and neurodevelopmental risks.[7,86] Several strengths of our review reinforce the findings. First, although the different outcomes under consideration preclude direct comparison of estimates, there is general consistency in the effect sizes estimated, lending weight to the evidence. Also, we use strict definitions of study design; this means that the estimates are better approximations of effect sizes than if weaker study designs were used: i.e. cross-sectional studies. Therefore, we indicate where potential mechanistic explanations should be explored, so that modifiable factors might be identified to help this vulnerable group. The included studies come from heterogeneous samples, countries, settings, measures and designs. This strengthens the conclusions of the review because, as the results generally show consistency, we can assume that the findings are independent of these factors.

To date, this is the most comprehensive review and meta-analysis of the associations between parental mental illness and offspring physical illness during childhood, however there are a number of limitations. Few studies investigated the effect of both maternal and paternal mental illness and, generally, they noted weaker effects for paternal exposure.[36,41,42,53] This indicates that maternal condition plays a more important role in the risk of offspring physical ill health, either as a result of intrauterine exposures, or through early childhood experiences or both; it also provides some evidence against a purely genetic cause for these relationships.

All the identified studies come from observational settings and are therefore subject to confounding bias. Most attempted to account for this bias, primarily using regression adjustment. Notably, most of the adjusted analyses were closer to a null effect size. This indicates that, overall, factors that increase the risk of parental mental illness are also likely to increase the risk of poor physical health in offspring (and *vice versa*). One obvious candidate for a variable of this kind is socioeconomic or multiple deprivation, inextricably linked with parental mental ill health[82] and poor child physical health.[18,83]

Despite adjustment for confounders, we must be cautious before attributing the causes of poor child physical health to parental mental disorders. First, not all the potential confounders are likely to be identified and measured in data available to researchers. Two of the more recent studies tried to account for residual confounding from familial factors by investigating whether associations still persist using sibling or cousin analysis[40,41] or when investigating paternal exposure.[40] Second, some studies may be subject to overadjustment, where analyses adjust for variables on the pathway between the exposure and the outcome. For example, poor fetal growth is associated with both prenatal maternal mental disorder[84] as well as many health outcomes. Therefore, this might be the mechanism by which prenatal maternal mental disorder influences child health and studies that adjust for this might be underestimating the effect of maternal mental illness on child health. Third, although we excluded studies in which it was clear that the outcome was measured before the exposure occurred, for many studies this was unclear. Therefore, we cannot rule out that at least some portion of the results were because poor child health affects parental mental illness and not *vice versa*. Fourth, although we actively tried to include unpublished research, all the identified studies were from the published literature. Therefore, it might well be that some positive findings are the result of publication bias.

Implications for future research and policy

This systematic review shines a stark light on the gaps in our knowledge about the physical health of children whose parents have mental illness, and highlights a need to shift the focus of research towards parental mental disorders other than maternal postnatal depression. Future studies should interrogate the extent to which antenatal, perinatal and postnatal exposures have differential effects on offspring's risk of physical illness. Also, maternal mental disorder may pose more risk to child physical health than paternal disorder but there is a strong need for future studies to include paternal exposure where possible if we are to understand the mechanisms behind these effects in particular outcomes. This chimes well with recent calls for research and policy to place a greater emphasis on the role of fathers in children's lives.[87]

Future research should interrogate the associations highlighted here to explore behavioural, environmental and genetic causes. To do this we highlight a need to develop fresh approaches to understanding the links between child physical health and parental mental health. These include the necessity of accounting for key confounders and to consider the use of alternative design strategies, including negative controls, sibling or quasi-experimental designs.[88] If future research is to be able to deepen our understanding of when and how these vulnerable children are at risk of preventable illnesses, large high-quality cohorts must be identified. For example, to investigate mechanisms of childhood asthma (still the most common childhood illness) we need to look at effects of different parental illnesses and effects of maternal versus paternal mental disorder. Combining data across such cohorts from different countries may offer such an opportunity.

Finally, from a policy perspective, such approaches can offer the detail needed to plan resource allocation and develop new service provision. Studies describing patterns of healthcare utilisation by these children and parents may be particularly valuable for this.

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