# Prenatal and Postpartum Depression in Fathers and Its Association With Maternal Depression

## A Meta-analysis

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**Context** It is well established that maternal prenatal and postpartum depression is prevalent and has negative personal, family, and child developmental outcomes. Paternal depression during this period may have similar characteristics, but data are based on an emerging and currently inconsistent literature.

**Objective** To describe point estimates and variability in rates of paternal prenatal and postpartum depression over time and its association with maternal depression.

**Data Sources** Studies that documented depression in fathers between the first trimester and the first postpartum year were identified through MEDLINE, PsycINFO, EMBASE, Google Scholar, dissertation abstracts, and reference lists for the period between January 1980 and October 2009.

**Study Selection** Studies that reported identified cases within the selected time frame were included, yielding a total of 43 studies involving 28 004 participants after duplicate reports and data were excluded.

**Data Extraction** Information on rates of paternal and maternal depression, as well as reported paternal-maternal depressive correlations, was extracted independently by 2 raters. Effect sizes were calculated using logits, which were back-transformed and reported as proportions. Random-effects models of event rates were used because of significant heterogeneity. Moderator analyses included timing, measurement method, and study location. Study quality ratings were calculated and used for sensitivity analysis. Publication bias was evaluated with funnel plots and the Egger method.

**Data Synthesis** Substantial heterogeneity was observed among rates of paternal depression, with a meta-estimate of 10.4% (95% confidence interval [CI], 8.5%-12.7%). Higher rates of depression were reported during the 3- to 6-month postpartum period (25.6%; 95% CI, 17.3%-36.1%). The correlation between paternal and maternal depression was positive and moderate in size ($r$=0.308; 95% CI, 0.228-0.384). No evidence of significant publication bias was detected.

**Conclusions** Prenatal and postpartum depression was evident in about 10% of men in the reviewed studies and was relatively higher in the 3- to 6-month postpartum period. Paternal depression also showed a moderate positive correlation with maternal depression.

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THE PREVALENCE, RISK FACTORS, and effects of depression among new fathers are poorly understood. Although a large body of research on maternal depression documents incidence rates between 10% and 30% and negative family and child developmental outcomes,[1-3] paternal prenatal and postpartum depression has received little attention from researchers and clinicians.[4] The emerging literature on paternal depression suggests that, like their maternal counterparts, fathers are at increased risk of depression in the postpartum[5] and gestational periods.[6-8] Moreover, several studies have now documented negative child outcomes associated with paternal prenatal and postpartum depression.[9,10]

Although recent literature has addressed this phenomenon, studies in paternal prenatal and postpartum depression are troubled by inconsistent methods, clinical heterogeneity, and prevalence estimates that vary considerably.[5,7,11-20] To date, only 2 reviews on prenatal and postpartum depression in fathers have been published, but neither sought to quantitatively synthesize or resolve the discrepancies across studies, methods, or other issues.[5,11] We conducted the present meta-analysis of depression in expecting and new fathers to (1) estimate paternal depression between the first trimester and 1 year postpartum; (2) describe differences across time within this period; (3) examine the association between paternal and maternal depression; (4) estimate the prevalence of maternal prenatal and postpartum depression identified in paternal depression studies; and (5) identify how published rates

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of paternal depression were affected by methodological factors such as measurement method, study location, and sample risk status.

## METHODS

### Search Strategy

We used 3 methods to identify studies for this meta-analysis. First, we used the reference lists of the most relevant reviews.[5,11] Next, we searched MEDLINE, PsycINFO, Dissertation Abstracts International, EMBASE, and Google Scholar using the search terms *depression, paternal, father, postnatal, postpartum, prenatal, antenatal,* and *perinatal*. Finally, we used the "ancestry approach,"[21] which involves consulting the reference lists of retrieved articles to find earlier relevant studies. Because of the emergent nature of this body of observational research literature, an inclusive approach to study selection was used.[22,23] Therefore, we included all relevant and accessible journal articles, dissertations, and book chapters that were produced between January 1980 and October 2009 that assessed paternal depression during pregnancy, the first postpartum year, or both.

### Study Selection

Studies that reported an estimated number of depression cases among identified fathers were included. This resulted in the exclusion of several studies that reported mean scores for symptom severity because the exact number of cases could not be clearly determined. Several articles[9,10,24-27] used data from common databases and were excluded to avoid duplication of data. Several studies measured depression on multiple occasions. In these cases, 1 depression measure per time period was selected based on these priorities: (1) structured interviews; (2) measures with demonstrated generality in men (eg, Beck Depression Inventory[28]) vs adaptations of maternal measures (eg, Edinburgh Postnatal Depression Scale[29]); and (3) measures with greater specificity for depression (eg, Beck Depression Inventory[28] vs General Health Questionnaire[30]). We excluded studies that selected fathers based on established maternal mental health problems because this could bias meta-analytic estimates. Also, because the identified studies of teen fathers were characterized by significant economic and social stressors, only studies of fathers aged 18 years or older were included.

### Data Abstraction and Quality Assessment

The 2 authors used a standardized coding manual (available from the authors on request) to extract the following data from articles: author names, publication year, sample size, period of assessment, study sample risk (0 or 1; high risk coded when a study denoted this clearly, including medically assisted pregnancies and infants with feeding, sleeping, or crying problems), location, sample size, response rate, number of fathers identified as depressed, number of mothers identified as depressed (when assessed), and correlation between maternal and paternal depressive symptoms. The coding manual was developed a priori and modified after use in several studies. Coding was done independently then aggregated, with disagreements resolved through discussion and consensus. Although quality assessment can be reliably conducted in meta-analyses of experimental studies, its use in observational research is controversial, with no clear consensus on rating methods or their appropriate use in analysis. As such, we used a simple objective rating system (based on the meta-analysis of similar data by Bennett et al[2]) that coded studies on a scale of 0 to 10, assigning 2 points each for sampling method (systematic or probability vs convenience or not reported), presence of clearly stated inclusion criteria, racial/ethnic diversity (≥20% minority), educational diversity (≤80% at 1 educational level), and response rate (reported at ≥60%). Studies that did not report these methodological issues received lower scores. Because evidence on the validity of quality ratings in observational research is lacking, we adopted the approach of Stroup et al[23] of broadly including studies and using sensitivity analysis to determine incremental effects of lower-quality studies.

### Effect Size and Statistical Analysis

**Primary Outcome.** The primary outcome was the point prevalence rate of paternal depression, defined as the number of cases divided by the total number of study participants. We coded these into both simple proportional effect sizes (by dividing the number of cases by the sample size) and logit units, as a direct transformation of these proportions. In this context, the logit transformation was used to form an unbounded (in contrast to the 0-to-1 bounded nature of proportions) estimate to facilitate moderator analysis.[31] After analysis, logit units were back-transformed to proportions for the purposes of reporting.

**Secondary Outcomes.** Secondary outcomes included rates of depression in female partners, which we coded as raw proportions and logit units, and standardized zero-order correlations between paternal and maternal depressive symptoms (when measured with a continuous or ordinal scale).

All major analyses were conducted with Comprehensive Meta-Analysis, version 2.0.[32] In general, random-effects models are argued to better address heterogeneity between studies and study populations, allowing for greater flexibility in parsing effect size variability. Moreover, they are less influenced by extreme variations in sample size.[22] Because studies in this meta-analysis are characterized by heterogeneity and highly variable sample sizes, random-effects models were used. Heterogeneity among study point estimates was assessed with the Q statistic, with magnitude of heterogeneity being evaluated with the $I^2$ index.[31] When reported, all confidence intervals (CIs) reflect a 95% criterion.

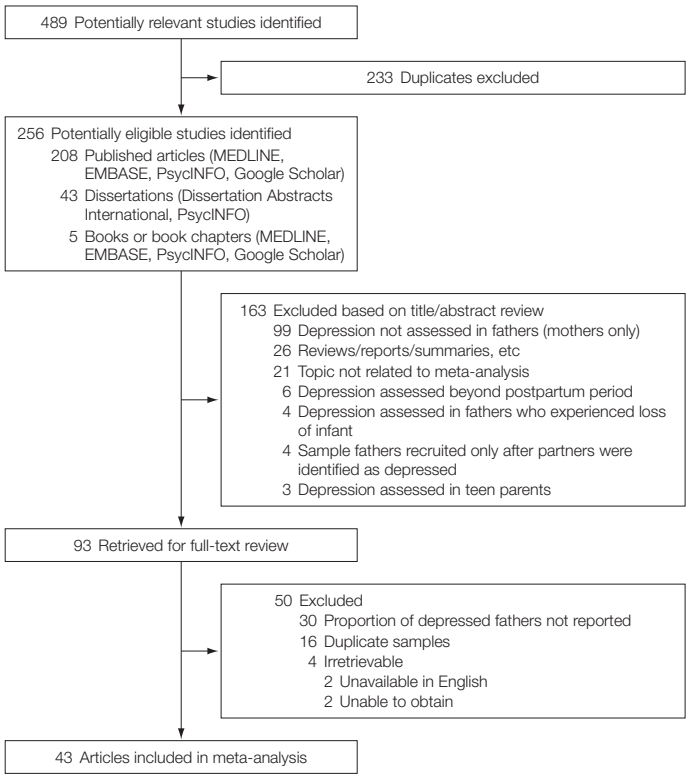
We examined the following determinants of primary and secondary outcomes: period of measurement, risk sta-

tus of the sample (eg, infant problems, medically assisted fertilization), and case identification method (interview vs rating scale). Study location was also coded because previous work has identified geographic variations in postpartum depression.[33] Because the timing of paternal depression vis-à-vis childbirth is of great interest in this study, period of measurement was coded into blocks that included (1) first trimester to 6 months' gestation, (2) greater than 6 months' gestation to birth, (3) immediately postbirth to 3 months postpartum, (4) greater than 3 months postpartum to 6 months postpartum, and (5) greater than 6 months postpartum to 12 months postpartum.

Publication bias was assessed by visually inspecting funnel plots and applying the regression intercept of Egger et al.[34] In addition, we used the fail-safe procedure of Orwin,[35] which is based on effect sizes that would be considered practically insignificant rather than the traditional null-effect reference. This generated a number of unpublished studies with effects at the estimated population base rate for adult male depression[36] that would be needed to move estimates to a nonsignificant difference from base rates.

To assess the robustness of the results, we performed sensitivity analyses by sequentially removing each study and rerunning the analysis. We also conducted a separate analysis excluding studies with quality ratings in the lowest third to determine if potential methodological weaknesses influenced meta-analytic estimates.

**Figure 1.** Study Selection for Inclusion in Meta-analysis




## RESULTS

### Included Studies

Of the initial 256 identified studies, most (n=163) were excluded because they were not applicable to the present meta-analysis (eg, articles on other topics, depression not assessed in fathers, reviews or summaries, beyond postpartum period, infant death, teen parents). Of those that were reviewed in full text, 30 were excluded because the proportion of depressive cases was not reported, 16 reported on a sample already included in the present study, and 4 were not retrievable (FIGURE 1). After a thorough review, 43 studies met the inclusion criteria for this meta-analysis[6-8,13-20,37-68] (TABLE). Of these studies, 23 reported rates of paternal depression at 2 or more time points and 20 reported a single observation. Because the inclusion of multiple effect sizes from a single sample would compromise the independence assumption of meta-analysis,[22] primary analyses used the earliest reported estimate, as this generally reflects a larger preattrition sample size.

The Table provides details on the characteristics of the 43 studies. Studies originated in 16 countries, with the United States contributing the most (n=17 studies). Most studies (n=40) used a self-report rating scale as the primary case definition method, with the remainder (n=3) using a structured or semistructured interview. Three studies enrolled men from higher-risk samples. Two studies used population-based sampling procedures embedded within larger birth cohort studies, but most studies (n=30) recruited from maternity or postpartum units, the remainder coming from parenting/prenatal classes and other health services. Thirty studies reported response rates greater than 70%. In addition to reporting paternal depression, 35 studies reported rates of partners' maternal depression and 14 reported the correlation between maternal and paternal depressive symptoms. Sample sizes varied widely across studies (N=23-10 975), with a median of 130 participants (first quartile=80; third quartile=307). In all, using initial sample sizes across the 43 studies, a total of 28 004 participants are represented in this meta-analysis.

### Tests for Heterogeneity

According to the criteria set by Higgins and Thompson,[69] the heterogeneity in published rates of paternal depression was statistically significant and

**Table.** Characteristics of Studies Included in Meta-analysis

| Source (Study Location) and Time of Assessment | Depression Measure (Cutoff) | No. of Participants (Women)[a] | Depressed, No. (%) | | Correlation Between Men and Women[b] |
|---|---|---|---|---|---|
| | | | Men | Women | |
| **Onset of paternal depression at gestation <6 mo** | | | | | |
| Areias et al,[17] 1996 (Portugal) | SADS | | | | |
| 6 mo gestation | | 42 (54) | 2 (4.8) | 9 (16.7) | |
| 3 mo postpartum | | 12 (24) | 2 (8.3) | 17 (67) | |
| 12 mo postpartum | | 42 (54) | 10 (23.8) | 20 (37) | |
| Condon et al,[6] 2004 (Australia) | | | | | |
| 5.75 mo gestation | EPDS (>12) | 312 | 16 (5.2) | | |
| | GHQ (>5) | | 57 (18.2) | | |
| | MHI-5 (<17) | | 14 (4.6) | | |
| 3 mo postpartum | EPDS (>12) | 276 | 5 (1.9) | | |
| | GHQ (>5) | | 31 (11.3) | | |
| | MHI-5 (<17) | | 4 (1.5) | | |
| 6 mo postpartum | EPDS (>12) | 241 | 5 (2.1) | | |
| | GHQ (>5) | | 27 (11.2) | | |
| | MHI-5 (<17) | | 4 (1.7) | | |
| 12 mo postpartum | EPDS (>12) | 222 | 5 (2.3) | | |
| | GHQ (>5) | | 23 (10.4) | | |
| | MHI-5 (<17) | | 7 (3.1) | | |
| Fawcett and York,[14] 1986 (US) | BDI (>9) | | | | |
| 3.5 mo gestation | | 23 | 1 (4.3) | 6 (26.1) | |
| 9 mo gestation | | 24 | 2 (8.3) | 8 (33.3) | |
| 1.5 mo postpartum | | 23 | 3 (13) | 6 (26.1) | |
| Field et al,[43] 2006 (US) | | | | | |
| 5 mo gestation | CES-D (>15) | 156 | 50 (32) | 56 (36) | |
| Fletcher et al,[44] 2008 (Australia) | | | | | |
| Sometime during gestation | EPDS (>9) | 307 | 16 (5.3) | | |
| | EPDS (>6) | 307 | 48 (15.5) | | |
| Frost,[45] 1996 (US) | CES-D (>15) | | | | |
| 5 mo gestation | | 527 | 75 (14.2) | 353 (67) | 0.23 |
| 1 mo postpartum | | 476 | 67 (14) | 100 (21) | 0.16 |
| 4 mo postpartum | | 442 | 46 (10.6) | 93 (21) | 0.17 |
| Matthey et al,[20] 2000 (Australia) | Multiple measures used to designate cases[c] | | | | |
| 5.5 mo gestation | | 152 | 8 (5.3) | 19 (12.3) | 0.18 |
| 1.5 mo postpartum | | 141 | 4 (2.8) | 11 (7.7) | 0.22 |
| 4 mo postpartum | | 125 | 4 (3.2) | 12 (9.7) | 0.18 |
| 12 mo postpartum | | 128 | 6 (4.7) | 16 (12.4) | 0.32 |
| Ramchandani et al,[55] 2008 (UK) | EPDS (>12) | 10 975 | | | 0.26-0.31 |
| 4.5 mo gestation | | | 426 (3.9) | | |
| 2 mo postpartum | | | 399 (3.6) | | |
| 8 mo postpartum | | | 378 (3.4) | | |
| 21 mo postpartum | | | 425 (3.9) | | |
| van den Berg et al,[7] 2009 (the Netherlands) | | | | | |
| 4 mo gestation | BSI (>15) | 3083 (3822) | 364 (11.8) | 409 (10.7) | |
| **Onset of paternal depression at gestation 6-9 mo** | | | | | |
| Atkinson and Rickel,[37] 1984 (US) | BDI (>9) | 78 | | | |
| 8 mo gestation | | | 10 (13) | 23 (29) | |
| 2 mo postpartum | | | 10 (13) | 20 (26) | |
| Bourne,[60] 2006 (US) | CES-D (>8) | | | | |
| 8 mo gestation | | 120 | 17 (14) | 48 (40) | 0.17 |
| 12 mo postpartum | | 87 | 8 (9) | 23 (27) | 0.08 |
| Escribè-Agüir et al,[8] 2008 (Spain) | | | | | |
| 8.25 mo gestation | EPDS (>10) | 669 (687) | 43 (6.5) | 71 (10.3) | |
| Hall and Long,[63] 2007 (Canada) | CES-D (>16) | 98 (91) | | | |
| 8.75 mo gestation | | | 11 (11.2) | 30 (33) | 0.27 |
| 2.5 mo postpartum | | | 21 (21.4) | 16 (17.6) | 0.21 |

*(continued)*

large in magnitude ($Q=825.081$; $P<.001$; $I^2=94.910$; $\tau^2=0.470$). Maternal depression also demonstrated significant heterogeneity across studies ($Q=1394.968$; $P<.001$; $I^2=97.563$; $\tau^2=0.792$), but the evidence for heterogeneity among correlations between maternal and paternal depressive symptoms was equivocal ($Q=89.906$; $P<.001$; $I^2=85.540$; $\tau^2=0.019$).

### Primary Outcomes

The overall random-effects estimate of paternal depression was 10.4% (95% CI, 8.5%-12.7%) (FIGURE 2). Although no significant differences in depression rates were observed between higher- and lower-risk samples (lower risk, 10.1%; 95% CI, 8.2%-12.4%; higher risk, 15.6%; 95% CI, 5.6%-36.5%; $Q=0.721$; $P=.40$), moderator analyses revealed 3 significant factors. First, there was considerable variability between different time periods vis-à-vis birth ($Q=20.256$; $P<.001$), with the 3- to 6-month postpartum period showing the highest rate (25.6%; 95% CI, 17.3%-36.1%) and the first 3 postpartum months showing the lowest rate (19 studies; 7.7%; 95% CI, 5.3%-11.1%). Second, national origin of the study accounted for variability in depression rates of fathers ($Q=7.108$; $P=.008$), with the US studies reporting an average rate of 14.1% (95% CI, 10.9%-18.0%) and international studies reporting an average rate of 8.2% (95% CI, 5.9%-11.1%). Finally, interview-based case definition methods were associated with lower overall prevalence estimates (rating scale, 11.0%; 95% CI, 8.9%-13.5%; interview, 4.9%; 95% CI, 3.6%-6.7%; $Q=18.236$; $P<.001$). Because paternal age and family size were inconsistently reported, conclusions could not be drawn regarding the moderator effects of either.

Maternal depression had a meta-analytic point estimate of 23.8% (95% CI, 18.7%-29.7%). Time period was a significant determinant of maternal depression ($Q=22.156$; $P<.001$), with higher rates reported during the 3- to 6-month postpartum period (41.6%).

Measurement method (rating scale, 25.5%; 95% CI, 20.0%-31.9%; interview, 9.8%; 95% CI, 5.9%-15.8%) was also a significant predictor of maternal depression rate ($Q=12.773$; $P<.001$). Study location (United States, 29.6; 95% CI, 19.3%-42.5%; international, 19.7%; 95% CI, 15.0%-25.4%) demonstrated a trend toward higher rates in the United States ($Q=2.599$; $P=.107$).

The overall random-effects estimate of maternal-paternal depressive symptom correlation was significantly larger than 0 and moderate in magnitude ($r=0.308$; 95% CI, 0.228-0.384).

### Tests for Publication Bias

Visual inspection of funnel plots (available from the authors) revealed no obvious evidence of publication bias. Quantitative evaluation of publication bias, as measured by the Egger intercept, was nonsignificant ($P=.15$). Finally, the Orwin fail-safe procedure, using a base rate of 3%,[36] determined that 1444 unpublished studies at or below this level would be needed to bring the overall meta-analytic estimate of prenatal and postpartum depression to a nonsignificant difference from the base rate.

### Sensitivity Analyses

Robustness of meta-analytic findings was examined by sequentially removing each study and reanalyzing the remaining data set (producing a new analysis for each study removed). No study affected the meta-analytic estimate more than 0.5%. Removing studies with quality ratings in the lowest 33% decreased the meta-analytic estimate of paternal depression by only 0.6% (from 10.4% to 9.8%). The pattern of differences across time periods, measurement methods, and study locations remained essentially unchanged in direction and magnitude.

## COMMENT

In this meta-analysis of paternal prenatal and postpartum depression and its correlation with maternal depres-

**Table.** Characteristics of Studies Included in Meta-analysis (continued)

| Source (Study Location) and Time of Assessment | Depression Measure (Cutoff) | No. of Participants (Women)[a] | Depressed, No. (%) | | Correlation Between Men and Women[b] |
|---|---|---|---|---|---|
| | | | Men | Women | |
| **Onset of paternal depression at gestation 6-9 mo (continued)** | | | | | |
| Keeton et al,[49] 2008 (US) | CES-D (>15) | 140 | | | |
| 9.04 mo gestation | | | 21 (15) | 62 (44) | |
| 1.3 mo postpartum | | | 17 (12) | 36 (26) | |
| 4.57 mo postpartum | | | 17 (12) | 41 (29) | |
| 6.68 mo postpartum | | | 18 (13) | 36 (26) | |
| 12.81 mo postpartum | | | 15 (11) | 35 (25) | |
| Leathers and Kelley,[12] 2000 (US) | CES-D (>16) | 124 | | | |
| 6.5 mo gestation | | | 9 (7.3) | 38 (30.6) | |
| 3.75 mo postpartum | | | 8 (6.5) | 14 (11.3) | |
| Morse et al,[52] 2000 (Australia) | EPDS (>9) | | | | |
| 6.25 mo gestation | | 251 | 30 (12) | 49 (19.5) | |
| 9 mo gestation | | 204 | 18 (8.7) | 45 (21.1) | |
| 1 mo postpartum | | 166 | 10 (6) | 38 (21.6) | |
| 4 mo postpartum | | 151 | 9 (5.8) | 23 (13.9) | |
| Raskin et al,[59] 1990 (US) | CES-D (>15) | 86 | | | |
| 8.5 mo gestation | | | 16 (18.6) | 24 (28) | 0.09 |
| 2 mo postpartum | | | 18 (21) | 18 (21) | 0.05 |
| Sandberg,[66] 1986 (US) | BDI (>9) | 50 | | | |
| 9.5 mo gestation | | | 8 (16) | 24 (48) | |
| 0.25 mo postpartum | | | 4 (8) | 17 (34) | |
| **Onset of paternal depression at postpartum <3 mo** | | | | | |
| Ballard et al,[19] 1994 (UK) | | | | | |
| 1.5 mo postpartum | EPDS (>12) | 178 | 16 (9) | 49 (27.5) | |
| 6 mo postpartum | EPDS (>12) | 148 | 8 (5.4) | 38 (25.7) | |
| 6 mo postpartum | PAS | 148 | 6 (4.1) | 23 (15.5) | |
| Carro et al,[39] 1993 (US) | | | | | |
| 1 mo postpartum | BDI (>9) | 70 | 7 (10) | 20 (29) | 0.25 |
| Davé et al,[61] 2005 (UK) | | 48 | | | |
| 1.25 mo postpartum | HADS (>7) | | 4 (8) | | |
| | EPDS (>12) | | 4 (8) | | |
| Edhborg et al,[41] 2005 (Sweden) | EPDS (>9) | 106 | | | |
| 0.25 mo postpartum | | | 3 (2.8) | 22 (20.8) | |
| 2 mo postpartum | | | 1 (0.9) | 10 (9.4) | |
| Edhborg,[62] 2008 (Sweden) | EPDS (>10) | | | | |
| 0.25 mo postpartum | | 132 (167) | 4 (3) | 40 (24)[d] | |
| 2 mo postpartum | | 113 (155) | 2 (1.8) | 19 (12) | |
| Ferketich and Mercer,[42] 1995 (US) | CES-D (>15) | 172 | | | |
| 0.5 mo postpartum | | | 36 (20.9) | | |
| 1 mo postpartum | | | 30 (17.4) | | |
| 4 mo postpartum | | | 25 (14.5) | | |
| 8 mo postpartum | | | 28 (16.3) | | |
| Gao et al,[46] 2009 (China) | | | | | |
| 1.5 mo postpartum | EPDS (>12) | 130 | 14 (10.8) | 18 (13.8) | 0.37 |
| Goodman,[47] 2008 (US) | | | | | |
| 2.5 mo postpartum | EPDS (>9) | 128 | 17 (13.3) | 36 (28) | 0.34 |
| Greenhalgh et al,[48] 2000 (UK) | EPDS (>12) | | | | |
| 0.25 mo postpartum | | 78 | 5 (6.4) | | |
| 1.5 mo postpartum | | 64 | 4 (6.3) | | |
| Hjelmstedt and Collins,[13] 2008 (Sweden)[e] | | | | | |
| 2 mo postpartum | EPDS (>9) | 53 | 4 (7.5) | | |
| Lane et al,[50] 1997 (Ireland) | EPDS (>12) | | | | |
| 0.1 mo postpartum | | 175 (289) | 6 (3) | 33 (11.4) | |
| 1.5 mo postpartum | | 175 (224) | 2 (1.2) | 24 (10.7) | |

*(continued)*

**Table.** Characteristics of Studies Included in Meta-analysis (continued)

| Source (Study Location) and Time of Assessment | Depression Measure (Cutoff) | No. of Participants (Women)[a] | Depressed, No. (%) | | Correlation Between Men and Women[b] |
|---|---|---|---|---|---|
| | | | Men | Women | |
| **Onset of paternal depression at postpartum <3 mo (continued)** | | | | | |
| Madsen and Juhl,[15] 2007 (Denmark) | | | | | |
| 1.5 mo postpartum | EPDS (>9) | 542 | 27 (5) | | |
| 1.5 mo postpartum | GMDS (>12) | 529 | 18 (3.4) | | |
| Matthey et al,[64] 2001 (Australia) | | | | | |
| 1.6 mo postpartum | DIS | 208 (230) | 6 (2.9) | 24 (10.4) | |
| Mezulis et al,[65] 2004 (US) | CES-D (>15) | 350 | | | 0.12[f] |
| 1 mo postpartum | | | 55 (15.6) | 41 (11.6) | |
| 4 mo postpartum | | | 47 (13.3) | 31 (8.8) | |
| 12 mo postpartum | | | 36 (10.2) | 21 (5.9) | |
| Pinheiro et al,[54] 2006 (Brazil) | | | | | |
| 2.25 mo postpartum | BDI (>9) | 386 | 46 (11.9) | 91 (23.6) | 0.52[g] |
| Skari et al,[56] 2002 (Norway) | GHQ (>1)[h] | | | | |
| 0.25 mo postpartum | | 115 (126) | 2 (1.7) | 7 (5.6) | |
| 1.5 mo postpartum | | 103 (109) | 2 (1.9) | 1 (0.9) | |
| 6 mo postpartum | | 84 (91) | 1 (1.2) | 2 (2.2) | |
| 0.25 mo postpartum | GHQ (>5)[i] | 115 (124) | 13 (11.3) | 46 (37.1) | |
| 1.5 mo postpartum | | 102 (108) | 11 (10.8) | 23 (21.3) | |
| 6 mo postpartum | | 84 (91) | 9 (10.7) | 17 (18.7) | |
| Soliday et al,[57] 1999 (US) | | | | | |
| 0.79 mo postpartum | CES-D (>16) | 51 | 13 (25.5) | 20 (39.2) | 0.29 |
| Thorpe et al,[68] 1992 (UK/Greece) | | | | | |
| 1 mo postpartum | EPDS (>12) | 267 (281) | 2 (0.7) | 35 (12.5) | |
| Wang and Chen,[58] 2006 (Taiwan) | | | | | |
| 1.5 mo postpartum | BDI (>9) | 83 | 26 (31.3) | 33 (39.8) | |
| **Onset of paternal depression at postpartum 3-6 mo** | | | | | |
| Bielawska-Batorowicz and Kossakowska-Petrycka,[38] 2006 (Poland) | | | | | |
| 4.5 mo postpartum | EPDS (>12) | 80 | 22 (27.5) | 25 (31.2) | 0.76 |
| Dudley et al,[40] 2001 (Australia)[e] | | | | | |
| 3.9 mo postpartum | EPDS (>10) | 93 (158) | 11 (11.8) | 75 (47.5)[d] | 0.33 |
| | GHQ (>4) | 93 | 43 (46.2) | | 0.27 |
| | BDI (>9) | 92 | 16 (17.4) | | 0.29 |
| Smart and Hiscock,[67] 2007 (Australia)[e] | | | | | |
| 3.75 mo postpartum | EPDS (>9) | 59 (71) | 18 (30) | 32 (45)[j] | |
| 4.5 mo postpartum | | 53 (59) | 10 (19) | 9 (15) | |
| **Onset of paternal depression at postpartum >6 mo** | | | | | |
| Bronte-Tinkew et al,[16] 2007 (US) | | | | | |
| 12 mo postpartum | CIDI-SF | 2137 | 115 (5.4) | 143 (6.7) | |
| Leathers et al,[51] 1997 (US) | | | | | |
| 6 mo postpartum | CES-D (>15) | 55 | 10 (18) | 17 (31) | |
| Paulson et al,[10] 2006 (US) | | | | | |
| 9 mo postpartum | CES-D (>9) | 5089 | 509 (10) | 712 (14) | |

Abbreviations: BDI, Beck Depression Inventory; BSI, Brief Symptom Inventory; CES-D, Center of Epidemiologic Studies Depression Scale; CIDI-SF, Composite International Diagnostic Interview Short Form; DIS, Diagnostic Interview Schedule; EPDS, Edinburgh Postnatal Depression Inventory; GHQ, General Health Questionnaire; GMDS, Gotland Male Depression Scale; HADS, Hospital Anxiety Depression Scale; MHI-5, Mental Health Index of the Short Form–36 health survey; PAS, Psychiatric Assessment Schedule; SADS, Schedule for Affective Disorders and Schizophrenia.

[a] Numbers in parentheses represent the number of women who participated at each time point. If a number in parentheses does not appear in this column and a percentage of depressed women is reported in the table, the number of female participants is the same as the number of male participants.

[b] All correlations are Pearson *r* correlation coefficients unless otherwise noted.

[c] Fathers were assessed for depression using the BDI (>16 from time 1 through time 4) and the GHQ (>7 at time 1 and time 4 only). Mothers were assessed using the BDI (>16 at time 1, time 3, and time 4), EPDS (>12 at time 2 only), and GHQ (>7 at time 1 and time 4 only).

[d] Percentage of depressed women based on EPDS greater than 9.

[e] The studies by Hjelmstedt and Collins[13] (child conceived through assisted reproductive technology), Dudley et al,[40] and Smart and Hiscock[67] (infant crying, sleeping, or eating problems in both) were considered to include high-risk individuals.

[f] Point biserial.

[g] Spearman correlation.

[h] A GHQ depression subscale case score greater than 1 indicates clinically important depression.

[i] A GHQ total case score greater than 5 indicates clinically important psychological distress.

[j] Percentages of depressed women based on EPDS greater than 12.

sion, wide variation was observed in reported rates of depression for fathers and mothers. The overall meta-analytic rate of paternal depression between the first trimester and 1 year postpartum was 10.4%. Since recent national data on base rates of depression in men place the 12-month prevalence at 4.8%,[70] this suggests that paternal prenatal and postpartum depression represents a significant public health concern. It must be noted that considerable variability was observed in reported rates of paternal depression. Although timing of measurement, study location, and measurement method were significant predictors, they accounted for only a small amount of overall heterogeneity. In terms of timing, fathers experienced the highest rates of depression 3 to 6 months postpartum, although the small number of studies measuring paternal depression during this period suggests cautious interpretation. Differences were also observed across study locations, with higher rates of prenatal and postpartum depression reported in the United States (14.1% vs 8.2% internationally). Questionnaire methods of case identification produced somewhat higher rates than did interview methods, although this should be interpreted cautiously because of the small number of studies that used interviews. Surprisingly, sample risk status was not a determinant of depression rates.

Maternal depression demonstrated considerable heterogeneity. This varied by time period, with a peak rate of 41.6% in the 3- to 6-month postpartum period, and by measurement method (higher rates with rating scales). Our random-effects estimate is somewhat larger than that of some reports,[1,2] with the variability in rates being clearly observable.

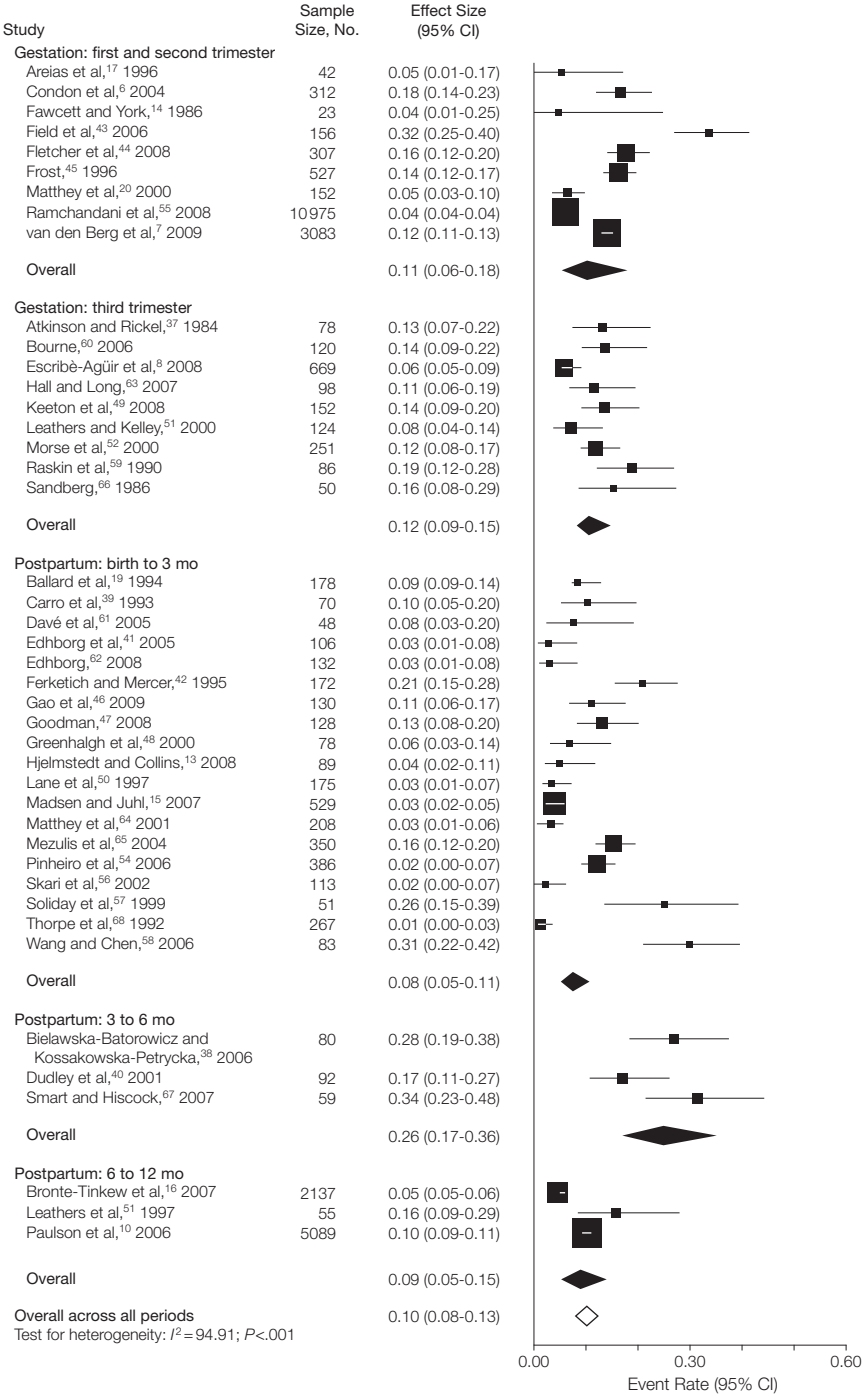
In both men and women, the potential causes of unexplained heterogeneity are varied. Although interview vs self-report questionnaire methods were compared, there were too many different questionnaires and interviews to conduct an instrument-by-instru-

ment moderator analysis. Sample location and undetermined sample characteristics may also contribute to heterogeneity. One possible source of heterogeneity is the liberal inclusion of cases that can be classified as minor depression, a category that includes individuals with depressive symptoms and impairment who do not meet strict criteria, either by severity, number, or duration of symptoms, for major depressive disorder.[71] Few of the studies reported herein explicitly described minor depression, but the use of rating scale cutoff scores suggests that strict diagnostic criteria for major depressive disorder were not used to identify cases. This interpretation is also consistent with the lower rates of identification observed in studies using structured interviews, a method that is typically more conservative in making a clinical diagnosis.

Another area of focus for this meta-analysis was the correlation between maternal and paternal depressive symptoms. Examining this effect in the context of paternal prenatal and postpartum depression is important, as paternal depression has been examined almost exclusively in the context of fathers paired with index mothers or children. Moreover, an extant meta-analysis of maternal postpartum depression suggests that marital satisfaction, a close correlate of depression, is among the strongest predictors of maternal depression.[72] In our meta-analysis, 12 of the 14 correlations were significant (meta-analytic estimate, 0.308), a moderate association by most standards. Although other authors have suggested that maternal depression may play some causal role in paternal depression,[5,11,40] none of the studies included in this meta-analysis suggest direction of causal influence. Studies that speak to direction of effect are of great interest, particularly for their implications for screening and prevention, but strong evidence of this is not yet available.

Our study has several limitations. First, because studies used variable methods of measuring and reporting de-

**Figure 2.** Prevalence of Paternal Birth-Related Depression From Gestation to 1 Year Postpartum




Overall effects were calculated through random-effects model estimates, with separate calculations for overall effects within each period and across all periods. Effect sizes were calculated via a logit transformation of rates (number of reported cases divided by the sample size), which were back-transformed to proportions after estimates and standard errors were computed. Studies are stratified by period of assessment. For studies that assessed depression at multiple time points, only the earliest estimate is reported. Data marker size corresponds to study sample size.

pression in different time periods, time frame–specific prevalence cannot be clearly established, limiting interpretation to the rate of depression observed at that point in time. Also, since point estimates are drawn from a pool of heterogeneous studies, many of which did not use strong population-based sampling methods, there is a potential of bias in our results from studies' methodological weaknesses. These may not have been adequately accounted for by our simplified method of quality rating. The method of identifying depressed cases was highly variable across studies, thereby limiting the specificity of our primary outcome. However, this variability in case identification accurately reflects inconsistencies in both applied and basic research into prenatal and postpartum depression.[5] Removing relatively weaker studies in sensitivity analysis left effects essentially unchanged. We did not find substantial evidence of publication bias in this area, and fail-safe analysis suggested that our findings are robust to unpublished null findings.

With these limitations in mind, this meta-analysis allows us to draw several conclusions regarding paternal prenatal and postpartum depression. First, a significant number of expecting and new fathers experience depression during this period. Second, expecting and new fathers in the United States experience depression at marginally higher rates than do fathers internationally, a finding that bears further investigation vis-à-vis varying social norms and postpartum work practices cross-nationally. Third, there is a moderate correlation between depression in fathers and mothers. There are many implications of these findings. The observation that expecting and new fathers disproportionately experience depression suggests that more efforts should be made to improve screening and referral, particularly in light of the mounting evidence that early paternal depression may have substantial emotional, behavioral, and developmental effects on children.[10,55] The correlation between paternal and maternal depression also suggests a screening rubric[73]—depression in one parent should prompt clinical attention to the other. Likewise, prevention and intervention efforts for depression in parents might be focused on the couple and family rather than the individual.

Future research in this area should focus on parents together to examine the onset and joint course of depression in new parents. This may increase our capacity for early identification of parental depression, add leverage for prevention and treatment, and increase the understanding of how parental depression conveys risk to infants and young children.

**Author Contributions:** Dr Paulson had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.
*Study concept and design:* Paulson.
*Acquisition of data:* Paulson, Bazemore.
*Analysis and interpretation of data:* Paulson, Bazemore.
*Drafting of the manuscript:* Paulson, Bazemore.
*Critical revision of the manuscript for important intellectual content:* Paulson, Bazemore.
*Statistical analysis:* Paulson, Bazemore.
*Administrative, technical, or material support:* Paulson, Bazemore.
*Study supervision:* Paulson.
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