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Abstract

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Keywords	depression; umbrella review; risk factors; meta-analyses; psychiatry; prevention
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TRANSLATIONAL PSYCHIATRY RESEARCH GROUP

Prof. Alan Schatzberg, MD
Editor-in-Chief
Journal of Psychiatric Research

Fortaleza, October 2nd, 2017

Dear Editor,

On behalf of my co-authors, I would like to submit the manuscript entitled "**Mapping Risk Factors for Depression Across the Lifespan: an Umbrella Review of Meta-Analyses and Mendelian Randomisation studies"** as a review article to *Journal of Psychiatric Research*.

This work represents a collaborative and systematic effort to provide an evidence synthesis of environmental risk factors for depression across the lifespan. In addition, we provided both a quantitative and qualitative assessment of available evidence. Sensitivity analyses were conducted, and associations supported by convincing and highly suggestive evidence were identified. The limitations as well as research and clinical implications of these findings are critically discussed.

We believe that our manuscript could be of interest to the broad and interdisciplinary audience of the *Journal of Psychiatric Research*.

Neither this manuscript nor any of its parts have been published or submitted elsewhere. In addition, this work had not been presented at scientific meetings.

We are at your entire disposal during the entire submission/review process of our work. We thank you in advance for the time dedicated to the evaluation our manuscript.

Sincerely yours,



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Abstract

The development of depression may involve a complex interplay of environmental and genetic risk factors. PubMed and PsycInfo databases were searched through August 3rd, 2017 to identify meta-analyses and Mendelian randomization (MR) studies of environmental risk factors associated with depression. For each eligible meta-analysis, we estimated the summary effect size and its 95% confidence interval (CI) by random-effects modelling, the 95% prediction interval, heterogeneity with I^2, and evidence of small-study effects and of excess significance bias. Seventy-six articles (70 meta-analytic reviews and 6 MR studies) met eligibility criteria; 134 meta-analytic syntheses from 1283 primary studies were included. While 109 associations were nominally significant ($p<0.05$), only 8 associations met criteria for convincing evidence and, when limited to prospective studies, convincing evidence was seen for 5 of them (widowhood, physical abuse during childhood, obesity, having 4-5 metabolic risk factors, sexual dysfunction, job strain). In studies in which depression was assessed through a structured diagnostic interview, only associations with widowhood, job strain, and being a Gulf War veteran were supported by convincing evidence. MR studies provided no consistent evidence for causal effects for obesity, smoking, and alcohol consumption. Current findings may have clinical and research implications for the early identification of individuals at-risk for depression.

Keywords: depression; umbrella review; risk factors; meta-analyses; psychiatry; prevention

Mapping Risk Factors for Depression Across the Lifespan: an Umbrella Review of Meta-Analyses and Mendelian Randomization studies

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Abstract

The development of depression may involve a complex interplay of environmental and genetic risk factors. PubMed and PsycInfo databases were searched through August 3rd, 2017 to identify meta-analyses and Mendelian randomisation (MR) studies of environmental risk factors associated with depression. For each eligible meta-analysis, we estimated the summary effect size and its 95% confidence interval (CI) by random-effects modelling, the 95% prediction interval, heterogeneity with I^2, and evidence of small-study effects and of excess significance bias. Seventy-six articles (70 meta-analytic reviews and 6 MR studies) met eligibility criteria; 134 meta-analytic syntheses from 1283 primary studies were included. While 109 associations were nominally significant ($p<0.05$), only 8 associations met criteria for convincing evidence and, when limited to prospective studies, convincing evidence was seen for 5 of them (widowhood, physical abuse during childhood, obesity, having 4-5 metabolic risk factors, sexual dysfunction, job strain). In studies in which depression was assessed through a structured diagnostic interview, only associations with widowhood, job strain, and being a Gulf War veteran were supported by convincing evidence. MR studies provided no consistent evidence for causal effects for obesity, smoking, and alcohol consumption. Current findings may have clinical and research implications for the early identification of individuals at-risk for depression.

Keywords: depression; umbrella review; risk factors; meta-analyses; psychiatry; prevention

1. Introduction

Major depressive disorder is a leading cause of disability worldwide (Eke et al., 2016), and is associated with psychosocial dysfunction (Birnbaum et al., 2010), increased health care use (Birnbaum et al., 2010), and excess mortality (Walker et al., 2015). Data from the World Mental Health (WMH) Survey indicates that the lifetime prevalence of a major depressive episode significantly varies across countries, and was found to be higher in higher-income (14.6%) compared to lower- and middle-income countries (11.1%) (Bromet et al., 2011).

Depression is a complex multi-factorial disorder (Otte et al., 2016), and heritability of the broad depression phenotype has been estimated to be approximately 37% (Flint and Kendler, 2014). Several environmental risk factors have been posited to contribute to the development of depression including but not limited to childhood maltreatment (Li, M. et al., 2016), childhood loss of a parent (Bifulco et al., 1987; Harris et al., 1986), lack of adequate parental care (Bifulco et al., 1987; Harris et al., 1986; Parker et al., 1997), unemployment, lower educational attainment, lower social support, the absence of a partner, lower physical activity (Schuch et al., 2016), and cannabis use (Kessler and Bromet, 2013; Lev-Ran et al., 2014; Otte et al., 2016). Emerging evidence also suggests that gene-environment interactions may contribute to the development of depressive episodes (Januar et al., 2015), and accumulating evidence also indicates that epigenetic mechanisms may play a role in the psychopathophysiology of this illness. Although some evidence suggests that the onset of depression can be prevented, the field awaits more conclusive data (Munoz et al., 2010; van Zoonen et al., 2014).

Fine mapping of environmental risk factors, some of which could be potentially modifiable, may be useful towards designing more targeted and effective preventative strategies for depression. Notwithstanding several systematic reviews and meta-analyses

of environmental risk factors for depression have been published, no systematic effort has been made to our knowledge to synthesize evidence from all these meta-analyses. Furthermore, Mendelian randomization (MR) studies may provide a methodologically sound alternative to infer causation. In those studies, an instrumental variable (often a genetic risk score) associated with the exposure is employed, and associations between the instrumental variable and outcome (here, depression) are considered to reflect a causal effect of the exposure on the outcome, if certain assumptions are met. MR experiments have been increasingly employed as an attempt to elucidate potential risk factors for depression (refer to Gage et al. (2013) for a review).

Thus, an umbrella review of evidence from meta-analyses and MR studies that investigated environmental risk factors for depression was performed. In addition, the prevalence of various biases in this literature was assessed. Finally, associations supported by strongest epidemiologic evidence were identified.

2. Material and methods

2.1. Search strategy and eligibility criteria

An umbrella review (i.e. a systematic review of multiple meta-analyses on a specific research topic) was conducted (Ioannidis, 2009). An *a priori* defined protocol was followed (available upon request to the authors). We systematically searched the PubMed and PsycInfo databases from inception to August 3rd, 2017 to identify meta-analyses of observational (i.e., cross-sectional, case-control, and prospective) studies investigating associations of environmental (non-genetic) risk factors for depression. Furthermore, MR studies that examined putative environmental risk factors for depression were also included. For the purposes of this umbrella review, meta-analyses and MR studies that specifically assessed environmental risk factors for perinatal depression were excluded. Detailed search strings are provided in the supplement that accompanies the online

version of this article. We included meta-analyses published in English that assessed one or more environmental factors (including other disease conditions) for associations with depression, and that included at least 3 datasets (i.e., component studies) and provided some measure of association. A diagnosis of depression had to be established through validated structured or semi-structured interviews based on *Diagnostic and Statistical Manual of Mental Disorders* (DSM) (American Psychiatric Association, 2013), *International Classification of Diseases* (ICD) (World Health Organization, 1993), or other acceptable diagnostic criteria. We also considered studies in which case ascertainment for depression was established by means of a validated screening instrument with a cutoff score.

2.2. Data extraction

Data extraction was independently conducted by two investigators, and in case of discrepancies, a consensus was reached through mutual discussion. From each eligible article, we recorded the first author, journal, year of publication, investigated risk factors and the number of included studies. We also extracted study-specific risk estimates (relative risk [RR], odds ratio [OR], hazard ratio [HR], or incident risk ratio) with their corresponding 95% CIs (confidence intervals), and number of cases and controls. Whenever the studies included several control groups, we considered associations with the healthy control group. We also annotated whether included meta-analyses primarily synthesized evidence for depression in youths or older adults. When 2 or more meta-analyses were available on the same exposure, we considered the one with the largest number of component datasets. Finally, we extracted data from forest plots of meaningful subgroup analyses from the original reports when at least 3 datasets were included (e.g. analyses limited to prospective studies). For MR studies, we extracted data on study population, sample size, genetic instruments, the variance of the environmental risk factor

explained by the genetic instruments (R^2) and MR effect estimates (odds ratio, hazard ratio, or regression coefficient β).

2.3. Methodological quality assessment

Two authors (CAK and AFC) independently rated the methodological quality of included systematic reviews and meta-analyses with the Assessment of Multiple Systematic Reviews (AMSTAR) instrument, which has been validated for this purpose (Pieper et al., 2015; Shea et al., 2007). Scores range from 0 to 11 with higher scores indicating greater quality. The AMSTAR tool involves dichotomous scoring (i.e. 0 or 1) of 11 items related to assess methodological rigor of systematic reviews and meta-analyses (e.g., comprehensive search strategy, publication bias assessment). AMSTAR scores are graded as high (8–11), medium (4–7) and low quality (0–3) (Shea et al., 2007).

2.4. Statistical analysis

We followed established procedures to assess the epidemiological credibility of possible risk factors for depression (Belbasis et al., 2015; Bellou et al., 2016; Carvalho et al., 2016a). First, for each meta-analysis we estimated the summary effect size (ES) with 95% CIs using random-effects modelling (Higgins et al., 2009; Lau et al., 1997). We also estimated the prediction interval and its 95% CI, which assesses heterogeneity, and evaluates the uncertainty of the ES in a new study investigating the same association (Higgins et al., 2009; IntHout et al., 2016). For the largest dataset of each meta-analysis, the standard error (SE) of the effect size was estimated and we examined whether the SE was less than 0.10. In a study with SE < 0.10, the difference between the effect size estimate and the upper or lower 95% CI is less than 0.20 which is regarded as a small effect size.

Between-study heterogeneity was assessed with the I^2 statistic (Patsopoulos et al., 2009). I^2 values may range from 0 to 100% and quantifies the variability in effect size

estimates that is due to heterogeneity rather than sampling error (Higgins and Thompson, 2002). Values exceeding 50% and 75% indicate large and very large heterogeneity, respectively.

We also assessed small-study effects (i.e. whether smaller studies had a tendency to provide more robust effect sizes compared to larger ones) using the Egger's regression asymmetry test (Egger et al., 1997). A $p < 0.10$ combined with a more conservative effect size in the largest study than in the summary effect size of random-effects meta-analysis was judged to provide evidence for small-study effects (Carvalho et al., 2016b).

We also applied the excess of significance test, which determines whether there is a relative excess of formally significant findings in the published literature due to publication bias, selective reporting of outcomes or analyses, or other reasons. This is a χ^2 test that assesses whether the observed (O) number of nominally significant findings is larger than their expected (E) number (Ioannidis and Trikalinos, 2007). We used the effect size of the largest study in each meta-analysis to calculate the power of each study using a non-central *t* distribution (Ioannidis, 2013; Lubin and Gail, 1990). Evidence of excess of significance was claimed when $E > O$ and two-tailed $p < 0.10$ as previously proposed (Ioannidis and Trikalinos, 2007).

For meta-analyses with Level I or II evidence (see below) sensitivity analyses were conducted: (1) considering only prospective studies; and (2) studies in which depression was diagnosed with a structured or semi-structured diagnostic interview as opposed to studies in which the ascertainment of cases was established through a screening instrument. Data from at least 3 datasets were required to perform these analyses.

Finally, for associations supported by type I or type II evidence, we used credibility ceilings, a sensitivity analysis tool, to account for potential methodological

limitations of included studies, which might lead to spurious precision of combined effect size estimates (Salanti and Ioannidis, 2009). This method assumes that every observational study has a probability *c* (credibility ceiling) that the true effect size is in a different direction from the one indicated by its point estimate. The pooled effect sizes were estimated for a wide range of credibility ceilings (Kyrgiou et al., 2017; Salanti and Ioannidis, 2009).

For MR studies, we performed and present a descriptive analysis of eligible studies. If all of the information required for calculation was provided (ie, sample size, number of cases, R^2, estimates of association, and measure of association), we performed a power calculation for the largest MR studies by using the non-centrality parameter based approach (Li, X. et al., 2017). For MR studies with missing R^2 values, we performed a crude power estimation by using the R^2 values from other MR studies that used the same genetic variants as instrumental variables. Statistical analysis and power calculations were performed in STATA, version 12.0.

2.5. Assessment of epidemiologic credibility

The level of epidemiologic credibility for each environmental risk factor was rated in accordance to pre-established criteria: I (convincing evidence), II (highly suggestive evidence), III (suggestive evidence), IV (weak evidence), and NS (all associations with non-significant findings). This set of criteria were used in similar efforts to synthesize evidence of environmental risk factors for other neuropsychiatric disorders (Belbasis et al., 2015; Bellou et al., 2016; Bortolato et al., 2017). In brief, evidence was rated as convincing evidence when all the following criteria are met: statistical significance according to random-effects models at $P < 10^{-6}$; included more than 1000 cases; without large between-study heterogeneity ($I^2 < 50\%$); 95% PI excluding the null value; and no evidence of small-study effects and excess significance. Other associations with more

than 1000 cases, $P < 10^{-6}$, and largest study presenting a statistically significant effect were graded as highly suggestive evidence. Other associations supported by > 1000 cases and a significant effect at $P < 10^{-3}$ were rated as suggestive evidence, while all remaining nominally significant associations (at $P < 0.05$) were considered as having weak evidence.

3. Results

3.1. Overall assessment of meta-analyses

Overall, the title and abstracts of 5,846 unique references were evaluated for eligibility (Figure 1), while full texts of 458 references were scrutinized for eligibility. Finally, 382 references were excluded after full-text review with reasons (Supplementary Table S1, available online) leaving 76 unique references that met eligibility criteria (70 references were meta-analytic reviews and the remaining 6 references were MR studies).

Data from 1283 original studies that assessed 89 different associations of environmental risk factors for depression (Table 1), 6 unique associations with pediatric depression (Table S2, available online), and 39 putative associations with late-life depression (Table 2) were obtained for eligible meta-analyses. Therefore, 134 unique associations were identified. The median number of datasets included in meta-analyses was 7.5 (IQR: 5–11), and the number of cases was > 1000 in 56 (41.8%) meta-analyses. The heterogeneity was large ($I^2 > 50\%$) in 76 (56.7%) meta-analyses, while 24 (17.9%) meta-analyses exhibited evidence of small-study effects. Excess of statistical significance could not be calculated for 29 (21.6%) meta-analyses (Chang-Quan et al., 2010a; Chang-Quan et al., 2010b; Grosso et al., 2016; Hasan et al., 2015; Huang et al., 2010; Lorant et al., 2003; Luger et al., 2014; Ttofi et al., 2011; Wang et al., 2016; Xiu-Ying et al., 2012; Yan et al., 2011; Zhai, Long et al., 2015), and this type of bias was observed in 17 (12.7%) meta-analyses (Bonde et al., 2016; Cole and Dendukuri, 2003; Huang et al., 2010; Osborn et al., 2014; Pan et al., 2012; Parsaik et al., 2014; Rahe et al., 2014; Zhao et al., 2012).

The overall methodological quality of included meta-analyses was medium (median AMSTAR score: 6; IQR: 5–7); Table S2 (available online) provides scores for each included meta-analysis.

<Please insert Figure 1 here>

<Please insert Table 1 here>

<Please insert Table 2 here>

3.2. Environmental risk factors for depression

Of 89 associations of environmental risk factors for depression in adults, 68 (77.3%) were nominally significant ($P < 0.05$). These associations pertained to dietary factors (k=18); drugs/substances (k=3); family factors (k=2); habits/lifestyle factors (k=6); infections (k=6); medical history and comorbid diseases (k=14); obesity and metabolic abnormalities (k=17); pregnancy and birth-related factors (k=3); socio-demographic factors (k=10); and exposure to trauma/disasters (k=10) (Table 1). Only 8 associations were supported by type I evidence namely obesity, exposure to physical abuse in childhood, the presence of 4-5 metabolic risk factors, tea intake, sexual dysfunction, job strain, widowhood, and dietary zinc (Figure 2). However, in sensitivity analysis in which only prospective studies were considered obesity, exposure to physical abuse in childhood, the presence of 4-5 metabolic risk factors, sexual dysfunction, job strain, and widowhood remained supported by type I evidence, whilst the association with tea intake was no longer statistically significant, and only 2 studies were available for the association with zinc intake (Table S4, available online). In sensitivity analysis where only studies in which a case definition of depression was established by means of a structured diagnostic interview as opposed to studies that used screening instruments were considered only job strain and widowhood remained supported by type I evidence (Table S5, available online), whilst sexual dysfunction dropped to type II evidence, and

exposure to physical abuse was now supported by weak evidence (Table S5, available online). Fewer than 3 independent studies in which a structured diagnostic interview was used to assess depression were available for obesity (n=2 studies), the presence of 4-5 metabolic risk factors (n=2 studies), tea intake (n=1 study), and dietary zinc (n=1 study). Thus, sensitivity analyses were not performed.

Finally, all environmental risk factors with the exception of job strain survived 10% credibility ceilings, while obesity, exposure to physical abuse in childhood, and the presence of 4-5 metabolic risk factors remained nominally significant when 20% credibility ceilings were considered (Table S6, available online).

<Please insert Figure 2 here>

Furthermore, 9 associations were supported by type II evidence namely intimate partner violence against women, co-occurring psoriasis, the metabolic syndrome, the presence of 3 metabolic risk factors, sedentary behavior, being a Gulf-war veteran, exposure to sexual abuse during childhood, exposure to emotional abuse during childhood and dry eye disease with Sjögren syndrome (Table 1). When only prospective studies were considered, associations with the presence of 3 metabolic risk factors and exposure to physical abuse during childhood remained supported by type II evidence, whilst associations with the metabolic syndrome and exposure to emotional abuse during childhood dropped to type III evidence (Table S4, available online). When sensitivity analysis considered only studies in which a diagnosis of depression was established through a structured diagnostic interview, then associations with being a Gulf war veteran was supported by type I evidence whereas associations with intimate partner violence against women, co-occurring psoriasis, and the presence of 3 metabolic risk factors remained supported by type II evidence (Table S5, available online). Associations with exposure to sexual abuse during childhood dropped to type III evidence, whilst

associations with exposure to emotional abuse during childhood became supported by type III evidence when only studies in which depression was assessed through a structured diagnostic interview were considered (Table S5, available online). In addition, associations with the metabolic syndrome were no longer nominally significant, while sensitivity analysis could not be performed for the presence of 3 metabolic risk factors, sedentary behavior, and the presence of dry eye in Sjögren syndrome because fewer than 3 independent studies were available (Table S5, available online). Finally, all association supported by type II evidence remained nominally significant when 10% credibility ceilings were considered (Table S6, available online).

3.3. Environmental risk factors for pediatric depression

Six environmental risk factors for depression in youths have been examined through meta-analyses. The presence of co-occurring asthma was supported by type II evidence (Lu et al., 2012). No sensitivity analysis considering only prospective studies or studies in which depression was assessed by means of a semi-structured diagnostic interview could be performed for this association because fewer than 3 independent studies were available. Any childhood maltreatment and obesity were supported by type III evidence. The remaining associations, namely smoking, and childhood chronic illnesses (cancer or diabetes) were not statistically significant (Table S3, available online). The association with co-occurring asthma remained nominally significant at 10% credibility ceilings, but was no longer significant at 20% credibility ceilings (Table S6, available online).

3.4. Environmental risk factors for late-life depression

Thirty-nine associations of environmental risk factors for late-life depression have been examined across 10 included articles (Table 2) pertaining to dietary factors (k=2); family factors (k=1); medical history and co-morbid diseases (k=20); obesity and metabolic factors (k=2); socio-demographic factors (k=13), and exposure to trauma and disasters

(k=1). No association was supported by type I evidence, though poor health status, presence of a chronic disease, poor vision, low-educational level and sleep disturbances were supported by type II evidence (Figure 2). However, when only prospective studies were considered, associations with the presence of a chronic disease were then supported by type III evidence, whilst the association with low educational level was supported by weak evidence (Table S4, available online). Only the association with sleep disturbances remained supported by type II evidence whereas sensitivity analysis limited to prospective studies could not be conducted for associations with poor health status or poor vision because fewer than 3 component datasets were available (Table S4, available online). Furthermore, when only studies in which a diagnosis of depression was established through a structured diagnostic interview were considered, associations with educational level and poor health status were then supported by weak evidence, whilst the association with sleep disturbances was now supported by type III evidence (Table S5, available online). Furthermore, all associations which were supported by type II evidence remained nominally significant when 20% credibility ceilings were considered (Table S6, available online).

3.5. Mendelian randomisation studies

Eight MR analyses were identified from 6 publications (Table 3). The median number of participants was 3,222 (range: 2,404–78,154) and median number of cases was 1,768 (range: 610–2,430). Four unique environmental risk factors (Table 3) were investigated in individual MR studies namely tobacco smoking, alcohol consumption, obesity and age of menarche. The proportion of variance in risk factors (R^2) explained by genetic instruments was 0.1–0.4%. More than one MR study was identified for 2 outcomes (tobacco smoking and alcohol consumption), and two studies used more than one instrumental (i.e. genetic) variable (Table 3). Discordance in either the direction and/or

statistical significance of associations among overlapping MR investigations existed for alcohol consumption. Only age of menarche presented significant associations at P<0.05 for one among multiple analyses, for only some quartiles (Sequeira et al., 2017).

4. Discussion

We systematically collected and appraised putative environmental risk factors for depression which have been examined in meta-analyses of observational studies. While this effort identified 139 associations with depression, including 6 specific associations with pediatric depression and 39 associations with late-life depression, only nine associations met criteria for convincing evidence either in the main or in sensitivity analyses. These risk factors included obesity (Jokela et al., 2014), the presence of 4-5 metabolic risk factors (Jokela et al., 2014), widowhood (Onrust and Cuijpers, 2006), sexual dysfunction (Atlantis and Sullivan, 2012), tea consumption (Dong et al., 2015), exposure to physical abuse during childhood (Mandelli et al., 2015), job strain (Madsen et al., 2017), dietary zinc intake (Li, Z. et al., 2017), and being a Gulf war veteran (Blore et al., 2015). The latter association was supported by convincing evidence only when the analysis was limited to studies in which the case definition of depression was performed through a structured diagnostic interview. Fifteen environmental risk factors were supported by highly suggestive evidence in the main analysis. These included intimate partner violence against women (Beydoun et al., 2012), the metabolic syndrome (Pan et al., 2012), being a Gulf War veteran (Blore et al., 2015), the presence of 3 metabolic risk factors (Jokela et al., 2014), sedentary behavior (Zhai, L. et al., 2015), co-occurring psoriasis (Dowlatshahi et al., 2014), exposure to emotional abuse during childhood (Mandelli et al., 2015), exposure to sexual abuse during childhood (Mandelli et al., 2015), and dry eye disease with Sjögren syndrome (Wan et al., 2016). In addition, co-occurring asthma was supported by type II evidence as a risk factor for pediatric depression (Lu et

al., 2012). Finally, associations with poor health status (Chang-Quan et al., 2010a), the presence of a chronic disease (Chang-Quan et al., 2010a), poor vision (Huang et al., 2010), lower educational attainment (Chang-Quan et al., 2010b), and sleep disturbances (Bao et al., 2017) were supported by highly suggestive evidence as risk factors for late-life depression.

Despite this substantial number of associations with type I or II in different analyses, very few factors maintained type I (convincing) evidence when the data were limited to prospective studies or studies with structured diagnostic interviews. Therefore, many of these associations still need to be seen with caution.

Evidence of high heterogeneity was observed in 56.4% of eligible meta-analyses. There are clear limitations when interpreting the Egger's test which provides an indication of small-study effects in the context of large heterogeneity (Sterne et al., 2011). A high degree of heterogeneity across studies in a meta-analysis may constitute an indication of bias, which may hinder interpretation of otherwise statistically significant associations. Nevertheless, it is possible that genuine heterogeneity across studies exists. Several lines of evidence indicate that genuine heterogeneity might operate in the field of depression research. First, significant cross-cultural differences have been reported in the prevalence of depression (Bromet et al., 2011; Kessler and Bromet, 2013), which may be partly due to varying levels of exposure to risk factors. For example, large variability of the conditional prevalence of a major depressive episode considering participants who screen positive for depression appeared when data from different countries in the WHM survey were compared (Bromet et al., 2011). In addition, depression is a heterogeneous phenotype with several subtypes (Hasler et al., 2004; Lichtenberg and Belmaker, 2010), which might have also complicated the search of significant genetic variants for depression (Direk et al., 2016; Flint and Kendler, 2014). In addition, our sensitivity

analyses suggest that study design and case definition through a screening tool compared to a structured diagnostic interview could influence the epidemiological credibility of some associations. This is a relevant methodological aspect because recent evidence suggests that the selective use of different cutoff points may bias accuracy estimates of screening instruments for depression, even if these instruments are regarded as validated measures (Levis et al., 2017), whilst this limitation is not evident in gold-standard structured diagnostic interviews for depression.

Most of the factors identified with convincing or highly suggestive evidence are stressors. It is speculated that stressors contribute to the emergence of depression by altering brain circuits, for example in the amygdala, which are involved in mood regulation (McEwen, 2003). However, it is unclear why some stressors, but not others, exhibited more convincing associations with depression. It is worthy to note that exposure to stressors may also contribute to the bidirectional associations between depression and several chronic disease states through the generation of allostatic overload (Fava et al., 2017; Fava et al., 2010; McEwen, 2017; Porcelli et al., 2012; Tomba and Offidani, 2012).

Particularly, we found convincing evidence that exposure to physical abuse during childhood is a risk factor for depression (Mandelli et al., 2015). These findings are consistent with the view that during critical periods of brain development, exposure to trauma may permanently impact brain function and response to stressors later in life via several mechanisms (Ignacio et al., 2016; Lopizzo et al., 2015; Post and Weiss, 1998). However, it should be noted that the epidemiological credibility of those findings decreased when sensitivity analyses were limited to studies in which depression was assessed through structured diagnostic interviews. Evidence indicates that the severity of depression occurs on a continuum, and there is no clear discontinuity in the latent structure of depression (Cuijpers and Smit, 2004; Prisciandaro and Roberts, 2009).

Notwithstanding structured diagnostic interviews are considered 'gold standards' to ascertain a diagnosis of a mental disorder for research purposes, there are also inherent limitations in this approach (see Nordgaard et al. (2013) for a wider discussion). Nevertheless, associations in which the epidemiological robustness of evidence does not survive sensitivity analysis restricted to studies in which the case definition of depression is established through structured diagnostic interviews should be interpreted with caution, and may require further elucidation.

Exposure to childhood maltreatment may also have deleterious systemic consequences. It has been argued that exposure to trauma early in life may be associated with several deleterious health outcomes later in life, some of which may be relevant to the emergence of depressive episodes (Nemeroff, 2016). We found highly suggestive evidence that women exposed to intimate partner violence may have a higher risk of developing depression. Notwithstanding variations across cultures should be acknowledged (Kessler and Bromet, 2013), most research points to a higher prevalence of depression among women. The mechanisms explaining a greater likelihood to develop depression among women appear complex (Kuehner, 2016), but a higher gender-specific exposure to certain stressors may play a role.

We also found convincing evidence that being a Gulf War veteran is associated with an increased risk of depression when studies that used a structured diagnostic interview for depression were considered. This is consistent with the stress-diathesis model of depression, which posits that exposure to more extreme stressors confers an elevated risk of depression (Booij et al., 2013). Finally, it is worth noting that depression could be conceived as a 'pathoplastic' syndrome (Keller et al., 2007), in which exposure to different types of stressors could shape symptom expression, further contributing to the heterogeneity of the phenotype (Hu et al., 2016; Keller et al., 2007). However, this

association was largely driven from cross-sectional studies, and thus prospective research is warranted.

We found evidence that obesity and the presence of 4-5 metabolic risk factors may increase the risk of developing depression. Several mechanisms may contribute to this association including speculated higher peripheral inflammation (de Melo et al., 2017; Kohler et al., 2017; Liu et al., 2014; Mansur et al., 2015). The concept of 'metabolic' depression has been proposed although epidemiological evidence remains modest (Liu et al., 2014; Mansur et al., 2015). The association of obesity and metabolic risk factors with depression may simply suggest that both conditions share common environmental risk factors. It is worthy to note that notwithstanding this analysis of 8 large-scale cohort studies provided convincing evidence for an association of obesity and depression, we observed that the association between obesity and depression was supported by weak evidence when data from a previous meta-analysis of 8 cohort studies were considered (Luppino et al., 2010). In addition, an included MR study did not provide evidence for a causal association of obesity as a risk factor for depression (Huang et al., 2010) although we could not estimate the statistical power of this study. Two meta-analyses of prospective studies indicated that adherence to either traditional healthy dietary patterns or to a Mediterranean-type diet could prevent the onset of depression (Rahe et al., 2014). These associations, however, were supported by weak evidence. Therefore, although several mechanisms may contribute to potential protective effects of a healthy diet for both depression and its associated metabolic comorbidities, including interactions with the microbiome (Slyepchenko et al., 2017). Clearly, more well-designed studies including randomized controlled trials are warranted.

The evidence that tea consumption and dietary zinc intake may protect against the development of depression needs to be seen cautiously. Several biological mechanisms

may at least in part explain the association of tea consumption and depression (Garcia-Blanco et al., 2016). For example, epigallocatechin gallate, a component of green tea may have antioxidant effects (Han et al., 2014), while certain flavanols derived from green tea were shown to increase hippocampal brain-derived neurotrophic factor (BDNF) and monoamine levels (Stringer et al., 2015). However, this association was no longer significant in sensitivity analysis restricted to prospective studies and there were not enough data assessing the association of zinc with depression in prospective studies. Furthermore, other lifestyle and dietary factors may confound this association as suggested by the authors of the included meta-analysis (Dong et al., 2015). Associations between single nutrients and outcomes in retrospective studies can be notoriously unreliable, even when the published data seem consistent (Schoenfeld and Ioannidis, 2013). Therefore, further prospective studies are warranted to assess this association.

The same caveats exist for the observed association between zinc and depression. Several lines of evidence have suggested an emerging role for zinc in the pathophysiology of depression (see Petrilli et al. (2017) for a recent review). For example, peripheral levels of zinc are lower in individuals with MDD compared to healthy controls (Swardfager et al., 2013), while zinc may exert pleiotropic effects including anti-inflammatory effects, modulatory effects on the glutamatergic N-methyl-D-aspartate (NMDA) receptor, and neurotrophic effects, which could be relevant for the pathophysiology of depression (Petrilli et al., 2017). However, a recent meta-analysis found that controlled trials that have tested adjunctive zinc for the treatment of depression have provided mixed results (Sarris et al., 2016). Furthermore, sensitivity analysis limited to prospective studies could not be performed only 2 studies were available from the included meta-analysis (Li, Z. et al., 2017). Therefore, further evidence from prospective studies and controlled trials are needed to provide more consistent causal inferences for this association.

Several putative sociodemographic risk factors for depression have been examined in meta-analyses. We found convincing evidence that widowhood as opposed to all other types of marital status was significantly associated with depression. However, prospective data does not necessarily rule out reverse causation or confounders. For example, it could be argued that depression prevents remarriage. In addition, type II evidence indicated that a lower educational attainment was associated with a higher risk of late-life depression. These findings are broadly consistent with large scale cross-cultural findings (Kessler and Bromet, 2013). However, there were variations across different countries (Bromet et al., 2011). In addition, a recent large-scale study confirmed that lower educational attainment was associated with depression (Peyrot et al., 2015). Furthermore, this study found no pleiotropic genetic effects mediating this association, thus suggesting that environmental factors are more likely involved.

Convincing evidence also indicates that sexual dysfunction is associated with depression, and this finding survived sensitivity analysis restricted to prospective studies. An accumulating body of evidence has pointed to reciprocal interactions between sexual dysfunction and depression (reviewed by de Abreu Barata (2017)). Hence sexual dysfunction may be either a manifestation of depression or a troublesome side effect associated with the use of antidepressant drugs (Carvalho et al., 2016c).

According to the job strain theory, work-related stress result from the additive effects of high job demands and low job control (Karasek et al., 1988). Convincing evidence indicates that job strain is a risk factor for depression, and the same level of evidence remained after sensitivity analysis restricted to either prospective studies or studies in which depression was assessed through structured diagnostic interviews. However, all included studies were conducted in Europe and Canada (Madsen et al., 2017), and thus the extent to which those findings apply to other countries (for example,

low- and middle-income countries) deserve further investigation. In addition, studies did not adjust for baseline depressive symptoms or a history of clinical depression, and the definition of job strain varied across studies. Considering that job strain across included studies was assessed through self-reported instruments, one cannot rule out the possible influence of participants' affective state on the reporting of work conditions. Furthermore, reverse causality is possible since evidence suggests that depression is associated with a significant detrimental impact on work productivity (Broadhead et al., 1990; McIntyre et al., 2015; Stewart et al., 2003).

4.1. Strengths and limitations

Some caveats should be considered when interpreting our analyses. First, our analyses included assessment of several hints of bias, but do not provide definitive proof thereof. Second, the methodological quality of meta-analyses varied; they were on average of medium quality. Third, some potential risk factors for depression which have not yet been subjected to meta-analyses might have been missed. These include some risk factors which have been classically related to depression namely childhood loss of a parent (Bifulco et al., 1987; Harris et al., 1986), poor parenting style (Parker et al., 1997), lack of social support, and social adversity (Bruce, 2002). Finally, evidence from MR studies that assessed putative environmental risk factors for depression remains largely negative or inconclusive. For example, two MR studies provided no evidence for a causal association of alcohol consumption and depression (Almeida et al., 2014; Wium-Andersen et al., 2015b). Nevertheless, our analyses indicate that both investigations were under-powered. Allowing for these caveats, the main strengths of this work rest on the topical comprehensiveness of the search, the inclusion of a large body of evidence, and the systematic quantitative and qualitative approaches used to rate the quality of available evidence.

4.2. Implications

Convincing associations with strong effect sizes may aid in the identification of high-risk populations, regardless of whether they are causal or not. Preventive efforts could be targeted to high-risk populations including children exposed to physical abuse, military personnel exposed to combat, recently widowed adults, obese individuals, and people with 4-5 five metabolic risk factors. This perspective is relevant considering recent evidence which suggests that depression can be prevented in youths (Merry et al., 2011), adults (van Zoonen et al., 2014), and the elderly (Cuijpers et al., 2015). In addition, a better organization and integration of services could enable the early recognition and management of depression in these populations. For example, a better integration of primary health care service and specialized mental health services could potentially aid in the recognition and treatment of depression among high-risk veterans (Ashrafioun et al., 2016). In addition, some evidence indicates that collaborative care interventions could improve outcomes among patients with depression and co-occurring obesity or with several metabolic risk factors (Katon et al., 2010; Panagioti et al., 2016). Moreover, this umbrella review indicates that several possible risk factors deserve proper meta-analytic assessment.

4.3. Conclusion

Our umbrella review mapped the status of 139 putative associations of environmental risk factors for depression across the life span. The adoption of reporting guidelines (e.g. Strengthening the Reporting of Observational Studies in Epidemiology [STROBE] (Vandenbroucke et al., 2007), and the pre-registration of the protocols of hypothesis testing observational studies might advance the availability of higher quality evidence in this field (Dal-Re et al., 2014), which could help to pave the way towards preventive efforts.

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Conflicts of interest

Authors report no direct competing interests for the present study.

FIGURES LEGENDS

***Fig. 1*. Flowchart of study selection.**

***Fig. 2.* Forest plot depicting effect sizes (relative risks or odds ratios) and 95% CIs of environmental risk factors for depression supported by type I or type II evidence.** Only primary analyses are presented. See text for sensitivity analyses. Underlined associations remained supported by type I or type II evidence in prospective studies, and associations in *italics* remained supported by type I or type II evidence in studies using a structured interview for depression diagnosis. #The association of being a Gulf-war veteran was supported by type I evidence in studies in which depression was assessed through a structured diagnostic interview. Other highlighted associations remained supported by the same type of evidence.

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Table 1. Characteristics, assessment of epidemiological credibility, and methodological quality assessment of the 88 eligible meta-analyses of environmental risk factors for depression.

Reference	Risk factor	Total number of cases/ controls	Number of primary studies	Effect size metric	Random effects summary effect size (95% CI)	P (by random effects)	95% PI	I^2	Small-study effects/ excess statistical significance	Level of evidence*	AMSTAR
Dietary factors											
Li, 2016	Dietary magnesium	NA	10	RR	0.83 (0.73–0.95)	0.006	0.59 – 1.16	68	Yes/NE	Weak	7
Li, 2016	Dietary calcium	NA	5	RR	0.64 (0.38–1.08)	0.097	0.12 – 3.32	61	No/NE	NS	7
Li, 2016	Fish consumption (highest vs. Lowest)	1113/496	17	RR	0.83 (0.69–1.01)	0.057	0.42 – 1.63	72	No/Yes	NS	7
Li, 2017	Iron intake	1045/5809	3	RR	0.40 (0.24–0.65)	< 0.001	0.00 – 87.36	63	No/No	III	6
Li, 2017	Dietary zinc	3708/16133	8	RR	0.65 (0.57–0.75)	$< 10^{-6}$	0.55 – 0.77	0	No/NE	I	6
Grosso, 2016	EPA + DHA intake	NA	4	RR	0.85 (0.70 – 1.04)	0.123	0.43 – 1.72	38	No/NE	NS	5
Grosso, 2016	Total n-3 PUFA intake	NA	9	RR	0.82 (0.69 – 0.97)	0.021	0.55 – 1.22	36	No/NE	Weak	5
Wang, 2016	Coffee intake	NA	4	RR	0.71 (0.52 – 0.99)	0.044	0.25 – 2.02	28	No/NE	Weak	4
Wang, 2016	Caffeine intake	NA	7	RR	0.65 (0.33 – 1.29)	0.219	0.05 – 7.75	97	No/NE	NS	4
Dong, 2015	Tea intake	4373/19174	13	RR	0.68 (0.61 – 0.77)	$< 10^{-6}$	0.49 – 0.95	42	No/No	I	7
Rahe, 2014	Traditional/healthy dietary patterns	NA	17	RR	0.76 (0.68 – 0.86)	$> 10^{-6}$ but $< 10^{-3}$	0.50 – 1.17	79	Yes/Yes	III	5
Rahe, 2014	Western/unhealthy dietary patterns	NA	9	RR	1.18 (0.97 – 1.43)	0.096	0.68 – 2.06	62	No/No	NS	5
Rahe, 2014	Mediterranean dietary patterns	NA	4	RR	0.76 (0.64 – 0.91)	0.002	0.39 – 1.51	57	No/No	Weak	5
Liu, 2016	Fruit intake	NA	8	RR	0.85 (0.77 – 0.93)	$> 10^{-6}$ but $< 10^{-3}$	0.67 – 1.07	42	Yes/No	III	5
Liu, 2016	Vegetable intake	NA	7	RR	0.87 (0.77 – 0.97)	0.014	0.68 – 1.11	26	Yes/No	Weak	5
Psaltopoulou, 2013	High adherence to Mediterranean diet	2951/15223	9	OR	0.68 (0.54 – 0.86)	0.001	0.37 – 1.26	50	No/No	Weak	7
Anglin, 2013	Vitamin D deficiency	NA	9	OR	1.31 (1.00 – 1.71)	0.047	0.63 – 2.73	53	No/No	Weak	9
Anglin, 2013	Vitamin D deficiency (prospective studies)	NA	3	HR	2.22 (1.42 – 3.47)	$> 10^{-6}$ but $< 10^{-3}$	0.05 – 92.87	19	No/No	III	9

Reference	Risk factor	Total number of cases/ controls	Number of primary studies	Effect size metric	Random effects summary effect size (95% CI)	P (by random effects)	95% PI	I^2	Small-study effects/ excess statistical significance	Level of evidence*	AMSTAR
Drugs/Substances											
Parsaik, 2014	Statins use	NA	7	OR	0.68 (0.52 – 0.89)	0.005	0.33 – 1.41	55	Yes/Yes	Weak	7
Lev-Ran, 2014	Cannabis use	NA	6	OR	1.17 (0.97 – 1.41)	0.102	0.73 – 1.86	37	No/No	NS	7
Lev-Ran, 2014	Heavy cannabis use	NA	5	OR	1.43 (1.00 – 2.04)	0.051	0.54 – 3.75	36	No/No	NS	7
Family factors											
Beydoun, 2012	Intimate partner violence against women	3003/21569	9	RR	2.57 (2.25 – 2.94)	$< 10^{-6}$	2.18 – 3.02	0	Yes/No	II	4
Onrust, 2006	Widowhood (vs. any other marital status)	2720/4868	5	RR	5.59 (3.79 – 8.23)	$< 10^{-6}$	2.09 – 14.97	30	No/No	I	3
Habits/Lifestyle											
Zhai, 2015	Short sleep duration	NA	6	RR	1.25 (0.96 – 1.62)	0.095	0.86 – 1.81	0	No/NE	NS	7
Zhai, 2015	Long sleep duration	NA	5	RR	1.41 (1.04 – 1.92)	0.026	0.86 – 2.32	0	No/NE	Weak	7
Zhai, 2015	Sedentary behaviour	60526/148346	24	RR	1.25 (1.16 – 1.35)	$< 10^{-6}$	0.97 – 1.60	51	No/No	II	7
Ho, 2014	Internet addiction	857/6842	5	OR	2.77 (2.04 – 3.75)	$< 10^{-6}$	1.09 – 7.02	56	No/No	Weak	5
Luger, 2014	Smoking	NA	77	OR	1.68 (1.55 – 1.82)	$< 10^{-6}$	0.91 – 3.10	95	Yes/NE	Weak	4
Zeng, 2016	Secondhand smoke exposure	NA	7	OR	1.60 (1.35–1.90)	$< 10^{-6}$	0.94 – 2.72	90	No/NE	Weak	7
Infections											
Wang, 2014	Borna disease virus (BDV) infection	205/2654	15	OR	3.25 (1.62 – 6.54)	$> 10^{-6}$ but $< 10^{-3}$	0.48 – 21.92	46	No/No	Weak	7
Wang, 2014	Varciella zoster virus	71/164	3	OR	2.10 (1.02 – 4.33)	0.045	0.02 – 229.62	0	No/No	Weak	7
Wang, 2014	Epstein-Barr virus	295/178	4	OR	1.98 (1.20 – 3.28)	0.008	0.66 – 5.99	0	No/No	Weak	7
Wang, 2014	Herpes simplex virus (HSV-1, HHV-1)	295/178	4	OR	1.98 (1.20 – 3.28)	0.008	0.66 – 5.99	0	No/No	Weak	7
Wang, 2014	Cytomegalovirus	100/135	3	OR	1.94 (0.91 – 4.14)	0.086	0.01 – 262.27	0	No/No	NS	7
Wang, 2014	Toxoplasma gondii	40/272	3	OR	1.46 (0.62 – 3.43)	0.381	0.01 – 367.24	0	No/No	NS	7

Reference	Risk factor	Total number of cases/ controls	Number of primary studies	Effect size metric	Random effects summary effect size (95% CI)	P (by random effects)	95% PI	I^2	Small-study effects/ excess statistical significance	Level of evidence*	AMSTAR
Medical history and comorbid diseases											
Fiest, 2013	Epilepsy (current depression)	1632/79762	3	OR	3.12 (1.70 – 5.73)	> 10^{-6} but < 10^{-3}	0.00 – 6085.29	91	No/No	III	8
Baglioni, 2011	Insomnia	NA	21	OR	2.60 (1.98 – 3.42)	< 10^{-6}	0.79 – 8.57	84	Yes/No	III	4
Dokras, 2011	Polycystic ovary syndrome	522/475	10	OR	4.03 (2.96 – 5.49)	< 10^{-6}	2.80 – 5.80	0	No/No	Weak	4
Cheungpasitporn, 2015	Hypomagnesaemia	NA	6	RR	1.34 (1.01 – 1.79)	0.046	0.68 – 2.64	33	Yes/No	Weak	7
Gilbody, 2007	Low folate	1769/13446	10	OR	1.54 (1.09 – 2.20)	0.016	0.59 – 4.05	50	No/No	Weak	7
Diprose, 2016	Psychogenic nonepileptic seizures (vs. epilepsy)	196/228	7	RR	1.15 (0.85 – 1.57)	0.365	0.65 – 2.04	14	No/No	NS	2
Osborn, 2014	Traumatic brain injury	NA	16	OR	3.41 (2.40 – 4.84)	< 10^{-6}	1.28 – 9.13	50	No/Yes	Weak	3
Hasan, 2015	Diabetes	NA	16	RR	1.25 (1.17 – 1.34)	< 10^{-6}	1.03 – 1.52	59	No/NE	Weak	4
Dowlatshahi, 2014	Psoriasis	86945/271291	9	OR	1.64 (1.41 – 1.90)	< 10^{-6}	1.07 – 2.51	84	No/No	II	6
Mitchell, 2013	Long-term cancer survivor (vs. HCs)	NA	13	RR	1.17 (0.95 – 1.45)	0.142	0.55 – 2.51	87	No/No	NS	8
Atlantis, 2013	COPD/chronic lung disease	297031/7128500	4	RR	2.38 (1.47 – 3.85)	> 10^{-6} but < 10^{-3}	0.31 – 18.45	78	Yes/No	III	6
Atlantis, 2012	Sexual dysfunction	5488/5683	6	OR	2.71 (1.93 – 3.79)	< 10^{-6}	1.13 – 6.50	41	No/No	I	8
Wan, 2016	Dry eye disease with Sjogren syndrome	3062/11178	7	OR	4.25 (2.67–6.76)	< 10^{-6}	1.02 – 2.77	72	No/NE	II	3
Wan, 2016	Dry eye disease without Sjogren syndrome	611517/2927706	6	OR	2.24 (1.50–3.34)	< 0.001	0.88 – 2.41	99	No/No	III	3
Obesity and metabolic abnormalities											
Pan, 2012	Metabolic syndrome	20924/132380	27	OR	1.42 (1.28 – 1.57)	< 10^{-6}	0.97 – 2.07	55	No/No	II	6
Pan, 2012	Metabolic syndrome (prospective studies)	2316/27034	9	OR	1.48 (1.17 – 1.87)	0.001	0.74 – 2.95	61	No/Yes	Weak	6
Tong, 2016	Newly diagnosed diabetes vs. normal glucose metabolism	NA	6	RR	1.07 (0.74–1.55)	0.726	1.02 – 2.77	42	No/NE	NS	9

Reference	Risk factor	Total number of cases/ controls	Number of primary studies	Effect size metric	Random effects summary effect size (95% CI)	P (by random effects)	95% PI	I^2	Small-study effects/ excess statistical significance	Level of evidence*	AMSTAR
Tong, 2016	Previously diagnosed diabetes vs. normal glucose metabolism	NA	6	RR	1.29 (1.03–1.62)	0.026	0.88 – 2.41	49	No/NE	Weak	9
Chen, 2016	Pre-diabetes vs. Normal glucose metabolism	63349/194031	19	OR	1.11 (1.03–1.19)	0.009	0.32 – 1.38	48	No/Yes	Weak	7
Chen, 2016	Undiagnosed diabetes vs. Normal glucose metabolism	27579/226971	14	OR	1.27 (1.02–1.59)	0.034	0.94 – 2.56	77	No/Yes	Weak	7
Nouwen, 2011	Impaired glucose metabolism	6236/26722	11	OR	0.95 (0.85 – 1.07)	0.441	0.74 – 1.23	25	No/No	NS	5
Nouwen, 2010	Type 2 diabetes mellitus	37964/131033	11	OR	1.24 (1.09 – 1.40)	$> 10^{-6}$ but $< 10^{-3}$	0.86 – 1.77	68	No/No	III	6
de Wit, 2010	Obesity (community samples)	NA	16	OR	1.21 (0.96 – 1.52)	0.103	0.47 – 3.12	95	Yes/No	NS	4
Luppino, 2010	Obesity (prospective studies)	NA	8	OR	1.55 (1.22 – 1.98)	$> 10^{-6}$ but $< 10^{-3}$	0.86 – 2.79	38	No/No	III	7
Luppino, 2010	Overweight (prospective studies)	NA	7	OR	1.27 (1.07 – 1.50)	0.007	0.92 – 1.74	14	No/No	Weak	7
Jokela, 2014	Overweight	11251/11413	8	OR	1.01 (0.91 – 1.11)	0.880	0.89 – 1.14	0	No/No	NS	3
Jokela, 2014	Obesity	7673/11413	8	OR	1.35 (1.21 – 1.50)	$< 10^{-6}$	1.18 – 1.54	0	No/No	I	3
Jokela, 2014	1 metabolic risk factor	9855/9586	8	OR	1.32 (1.06 – 1.65)	0.014	0.72 – 2.42	60	Yes/No	Weak	3
Jokela, 2014	2 metabolic risk factors	6691/9586	8	OR	1.45 (1.17 – 1.80)	$> 10^{-6}$ but $< 10^{-3}$	0.83 – 2.54	51	Yes/No	III	3
Jokela, 2014	3 metabolic risk factors	3014/9586	8	OR	1.99 (1.60 – 2.48)	$< 10^{-6}$	1.20 – 3.30	35	Yes/No	II	3
Jokela, 2014	4 or 5 metabolic risk factors	1191/9586	8	OR	2.06 (1.59 – 2.68)	$< 10^{-6}$	1.19 – 3.56	24	No/No	I	3
Pregnancy and birth related											
Loret de Mola, 2014	Low birth weight (≤ 2500 g)	NA	21	OR	1.38 (1.16 – 1.65)	$> 10^{-6}$ but $< 10^{-3}$	0.89 – 2.14	24	No/No	III	5
Loret de Mola, 2014	Premature birth	NA	8	OR	1.08 (0.77 – 1.52)	0.662	0.45 – 2.57	48	No/No	NS	5
Loret de Mola, 2014	Small for gestacional age	NA	5	OR	1.14 (0.64 – 2.03)	0.656	0.20 – 6.36	49	No/No	NS	5

Reference	Risk factor	Total number of cases/ controls	Number of primary studies	Effect size metric	Random effects summary effect size (95% CI)	P (by random effects)	95% PI	I^2	Small-study effects/ excess statistical significance	Level of evidence*	AMSTAR
Sociodemographic factors											
Tarricone, 2012	Ethnical group (minority vs. predominant)	13569/32646	19	RR	1.15 (1.00 – 1.33)	0.050	0.65 – 2.04	86	No/No	NS	4
Lorant, 2003	Low socioeconomic status	NA	56	OR	1.87 (1.62 – 2.16)	$< 10^{-6}$	0.73 – 4.78	87	Yes/NE	Weak	6
Richardson, 2015	Neighborhood socioeconomic conditions	NA	10	OR	1.12 (1.01 – 1.24)	0.029	0.82 – 1.53	80	No/No	Weak	7
Madsen, 2017	Job strain	1909/25552	7	OR	1.77 (1.46–2.13)	$< 10^{-6}$	1.02 – 14.01	24	No/No	I	7
Madsen, 2017	Job strain	982/119229	14	HR	1.27 (1.04–1.55)	0.019	0.88 – 7.32	25	No/Yes	Weak	7
Puthran, 2016	Medical students vs. non-medical students	NA	6	OR	0.96 (0.65–1.43)	0.857	0.25 – 3.67	85	Yes/NE	NS	7
Kim, 2016	Insecurely employed vs. Securely employed	795/3903	6	OR	1.29 (1.06–1.57)	0.010	0.66 – 2.53	89	No/Yes	Weak	6
Kim, 2016	Employed vs. Unemployed	40679/1836	13	OR	1.16 (1.09–1.23)	< 0.001	0.96 – 1.40	69	Yes/Yes	III	6
Kisely, 2017	Indigenous vs. Other populations	4843/57988	8	OR	1.24 (0.63–2.44)	0.536	0.11 – 13.60	97	No/NE	NS	8
Watanabe, 2016	Overtime work	432/12990	5	RR	1.05 (0.68–1.61)	0.829	0.28 – 3.95	54	No/No	NS	5
Trauma and disasters											
Chen, 2010	Lifetime exposure to sexual abuse	503/2768	11	OR	2.64 (1.92 – 3.65)	$< 10^{-6}$	1.03 – 6.77	68	No/No	Weak	9
Bonde, 2016	Natural disaster	NA	5	OR	2.14 (1.18 – 3.89)	0.013	0.23 – 19.66	94	No/Yes	Weak	8
Bonde, 2016	Terrorist act	NA	6	OR	2.02 (1.38 – 2.96)	$> 10^{-6}$ but $< 10^{-3}$	0.57 – 7.16	82	No/No	III	8
Bonde, 2016	Military deployment	NA	7	OR	1.66 (1.10 – 2.49)	0.015	0.38 – 7.29	99	No/No	Weak	8
Blore, 2015	Gulf-war veterans (vs. non-deployed personnel)	16826/13136	11	OR	2.37 (1.91 – 2.93)	$< 10^{-6}$	1.20 – 4.67	76	No/No	II	5
Ttofi, 2011	Bullying victimization during school	NA	20	OR	1.74 (1.54 – 1.97)	$< 10^{-6}$	1.13 – 2.68	58	No/NE	Weak	5
Mandelli, 2015	Emotional abuse in childhood	4112/12337	8	OR	2.78 (1.89 – 4.09)	$< 10^{-6}$	0.74 – 10.46	91	No/No	II	7
Mandelli, 2015	Neglect in childhood	1668/3620	6	OR	2.75 (1.59 – 4.74)	$> 10^{-6}$ but $< 10^{-3}$	0.40 – 19.06	92	Yes/No	III	7

Reference	Risk factor	Total number of cases/ controls	Number of primary studies	Effect size metric	Random effects summary effect size (95% CI)	P (by random effects)	95% PI	I^2	Small-study effects/ excess statistical significance	Level of evidence*	AMSTAR
Mandelli, 2015	Sexual abuse in childhood	4586/13915	14	OR	2.42 (1.94 – 3.02)	$< 10^{-6}$	1.19 – 4.94	69	No/No	II	7
Mandelli, 2015	Physical abuse in childhood	3886/12952	10	OR	1.98 (1.68 – 2.33)	$< 10^{-6}$	1.33 – 2.94	42	No/No	I	7

Abbreviations: CI = confidence interval; **NA** = not available; **NE** = not evaluated due to lack of datasets or individual study information for power calculations; **EPA =** Eicosapentaenoic acid; **DHA** = Docosahexaenoic acid; **HSV-1** = Herpes simplex virus type I; **HHV-1** = Human herpesvirus 1; **COPD** = Chronic pulmonary obstructive disease

* **Evidence classification: I** = convincing evidence criteria ($p < 10^{-6}$ per random-effects model; over than 1000 cases; no excess of significance bias; no small studies effects; small heterogeneity; prediction interval not including the null); **II** = highly-suggestive evidence criteria (not meeting type I criteria, but having $p < 10^{-6}$ per random-effects model; over than 1000 cases; effect size of the largest study not crossing the null); **III** = suggestive evidence criteria (not meeting type I or II criteria, but having $p < 10^{-3}$ per random-effects model; over than 1000 cases); **Weak** evidence criteria (all other associations with $p < 0.05$ per random-effects model); **NS** = non-significant associations; **NE** = not evaluated due to lack of individual study information.

Table 2. Characteristics, assessment of epidemiological credibility, and methodological quality assessment of 39 eligible meta-analyses of environmental risk factors for late-life depression.

Reference	Risk factor	Total number of cases/ controls	Number of primary studies	Effect size metric	Random effects summary effect size (95% CI)	P (by random effects)	95% PI	I^2	Small-study effects/ excess statistical significance	Level of evidence*	AMSTAR
Dietary factors											
Petridou, 2016	Low folate	NA	11	OR	1.21 (0.98 – 1.49)	0.079	0.71 – 2.05	37	No/No	NS	4
Petridou, 2016	Low B12	NA	8	OR	1.25 (0.97 – 1.59)	0.080	0.68 – 2.29	40	No/No	NS	4
Family factors											
Yan, 2011	Marital status (widowed, divorced, never married vs. married)	515/539	8	RR	1.36 (1.01 – 1.84)	0.042	0.51 – 3.61	81	No/NE	Weak	4
Medical history and comorbid diseases											
Chang-Quan, 2010	Poor health	8630/16552	11	OR	4.08 (3.25 – 5.12)	$< 10^{-6}$	1.88 – 8.84	83	No/No	II	4
Chang-Quan, 2010	Chronic disease	9090/15321	10	OR	2.59 (1.78 – 3.76)	$< 10^{-6}$	0.65 – 10.30	95	No/No	II	4
Chang-Quan, 2010	Chronic disease (prospective studies)	1056/8755	8	RR	1.53 (1.20 – 1.96)	$> 10^{-6}$ but $< 10^{-3}$	0.72 – 3.25	68	No/NE	III	4
Huang, 2010	Stroke	646/5283	9	OR	1.87 (1.33 – 2.62)	$> 10^{-6}$ but $< 10^{-3}$	0.66 – 5.31	66	No/No	Weak	6
Huang, 2010	Poor hearing	4448/13319	7	OR	1.71 (1.28 – 2.27)	$> 10^{-6}$ but $< 10^{-3}$	0.75 – 3.91	79	No/No	III	6
Huang, 2010	Poor vision	11066/20976	12	OR	1.94 (1.67 – 2.25)	$< 10^{-6}$	1.32 – 2.86	54	No/No	II	6
Huang, 2010	Arthritis	2269/6491	6	OR	2.27 (1.35 – 3.82)	0.002	0.37 – 13.85	88	No/No	Weak	6
Huang, 2010	Hypertension	762/3348	5	OR	1.28 (0.96 – 1.72)	0.096	0.53 – 3.08	57	No/No	NS	6
Huang, 2010	Cardiac disease	1911/10134	6	OR	1.81 (1.41 – 2.31)	$> 10^{-6}$ but $< 10^{-3}$	0.91 – 3.60	56	No/No	III	6
Huang, 2010	Chronic lung disease	646/2263	4	OR	2.14 (0.97 – 4.74)	0.060	0.06 – 78.35	84	No/No	NS	6
Huang, 2010	Gastrointestinal disease	166/2653	4	OR	1.95 (0.80 – 4.72)	0.140	0.04 – 84.40	75	No/No	NS	6
Huang, 2010	Cancer survivors	310/2371	5	OR	0.87 (0.61 – 1.25)	0.463	0.49 – 1.57	0	No/No	NS	6
Huang, 2010	Kidney disease	67/1872	3	OR	2.22 (1.31 – 3.76)	0.003	0.07 – 67.38	0	No/No	Weak	6
Huang, 2010	Urologic problems	150/1492	3	OR	2.19 (1.52 – 3.15)	$> 10^{-6}$ but $< 10^{-3}$	0.20 – 23.32	0	No/No	Weak	6

Reference	Risk factor	Total number of cases/ controls	Number of primary studies	Effect size metric	Random effects summary effect size (95% CI)	P (by random effects)	95% PI	I^2	Small-study effects/ excess statistical significance	Level of evidence*	AMSTAR
Cole, 2003	Prior depression	623/3667	3	OR	2.49 (1.55 – 4.01)	$> 10^{-6}$ but $< 10^{-3}$	0.01 – 510.34	71	Yes/No	Weak	5
Bao, 2017	Sleep disturbances	2610/3545	11	RR	1.92 (1.59–2.33)	$< 10^{-6}$	1.39 – 2.66	10	No/Yes	II	8
Bao, 2017	Persistent sleep disturbances	674/1872	4	RR	3.87 (2.45–6.12)	$< 10^{-6}$	0.90 – 16.61	27	No/Yes	Weak	8
Agtmaal, 2017	Cerebral microbleeds	NA	4	OR	1.18 (1.03–1.35)	0.015	0.88 – 1.58	0	No/NE	Weak	8
Agtmaal, 2017	Cerebral microinfarction	NA	4	OR	1.43 (1.22–1.68)	< 0.001	0.98 – 2.10	2	Yes/NE	Weak	8
Agtmaal, 2017	White Matter Hyperintensities	NA	30	OR	1.24 (1.15–1.34)	$< 10^{-6}$	1.39 – 2.66	67	Yes/NE	Weak	8
Obesity and metabolic factors											
Huang, 2010	Diabetes	1814/6804	9	OR	1.88 (1.31 – 2.70)	$> 10^{-6}$ but $< 10^{-3}$	0.57 – 6.25	82	No/Yes	III	6
Huang, 2010	Diabetes (prospective studies)	518/1009	3	RR	1.50 (0.92 – 2.44)	0.107	0.01 – 402.35	71	No/NE	NS	6
Sociodemographic factors											
Zhao, 2012	Age > 65	15017/7004	6	OR	1.63 (1.24 – 2.16)	$> 10^{-6}$ but $< 10^{-3}$	0.61 – 4.35	90	No/Yes	III	5
Zhao, 2012	Age > 70	11875/10650	6	OR	1.22 (0.97 – 1.52)	0.089	0.56 – 2.64	87	No/No	NS	5
Zhao, 2012	Age > 70 (prospective studies)	442/4980	4	RR	5.92 (0.52 – 66.86)	0.150	0.00 – 819558.94	99	No/No	NS	5
Zhao, 2012	Age > 75	11219/20534	19	OR	1.35 (1.17 – 1.56)	$> 10^{-6}$ but $< 10^{-3}$	0.76 – 2.39	76	No/No	III	5
Zhao, 2012	Age > 80	621/8326	3	RR	1.64 (1.36 – 1.98)	$< 10^{-6}$	0.47 – 5.67	1	No/No	Weak	5
Zhao, 2012	Age > 85	4559/19039	12	OR	1.52 (1.20 – 1.93)	$> 10^{-6}$ but $< 10^{-3}$	0.68 – 3.38	73	No/No	III	5
Xiu-Ying, 2012	Living alone	10478/23612	16	OR	1.55 (1.23 – 1.95)	$> 10^{-6}$ but $< 10^{-3}$	0.64 – 3.72	76	No/No	III	5
Xiu-Ying, 2012	Living alone (prospective studies)	591/754	4	RR	1.27 (0.90 – 1.79)	0.180	0.45 – 3.59	21	No/NE	NS	5
Xiu-Ying, 2012	Live in nursing home (prospective studies)	272/1927	3	RR	1.94 (1.18 – 3.18)	0.009	0.01 – 362.10	56	No/NE	Weak	5
Chang-Quan, 2010	Low educational level	16590/24067	24	OR	1.58 (1.38 – 1.82)	$< 10^{-6}$	0.89 – 2.83	74	No/No	II	4
Chang-Quan, 2010	Low educational level (prospective studies)	3957/6374	12	RR	1.49 (1.16 – 1.91)	0.002	0.64 – 3.49	75	No/NE	Weak	4
Cole, 2003	Lower education	619/5759	4	OR	1.79 (1.14 – 2.80)	0.011	0.27 – 12.02	73	Yes/Yes	Weak	5

Reference	Risk factor	Total number of cases/ controls	Number of primary studies	Effect size metric	Random effects summary effect size (95% CI)	P (by random effects)	95% PI	I^2	Small-study effects/ excess statistical significance	Level of evidence*	AMSTAR
Cole, 2003	Unmarried	782/6839	5	OR	1.01 (0.83 – 1.22)	0.950	0.64 – 1.59	22	No/No	NS	5
Trauma and disasters											
Cole, 2003	Recent bereavement	713/7507	3	OR	3.95 (3.06 – 5.08)	$< 10^{-6}$	0.61 – 25.51	10	No/No	Weak	5

Abbreviations: CI = confidence interval; **NA** = not available; **NE** = not evaluated due to lack of datasets or individual study information for power calculation

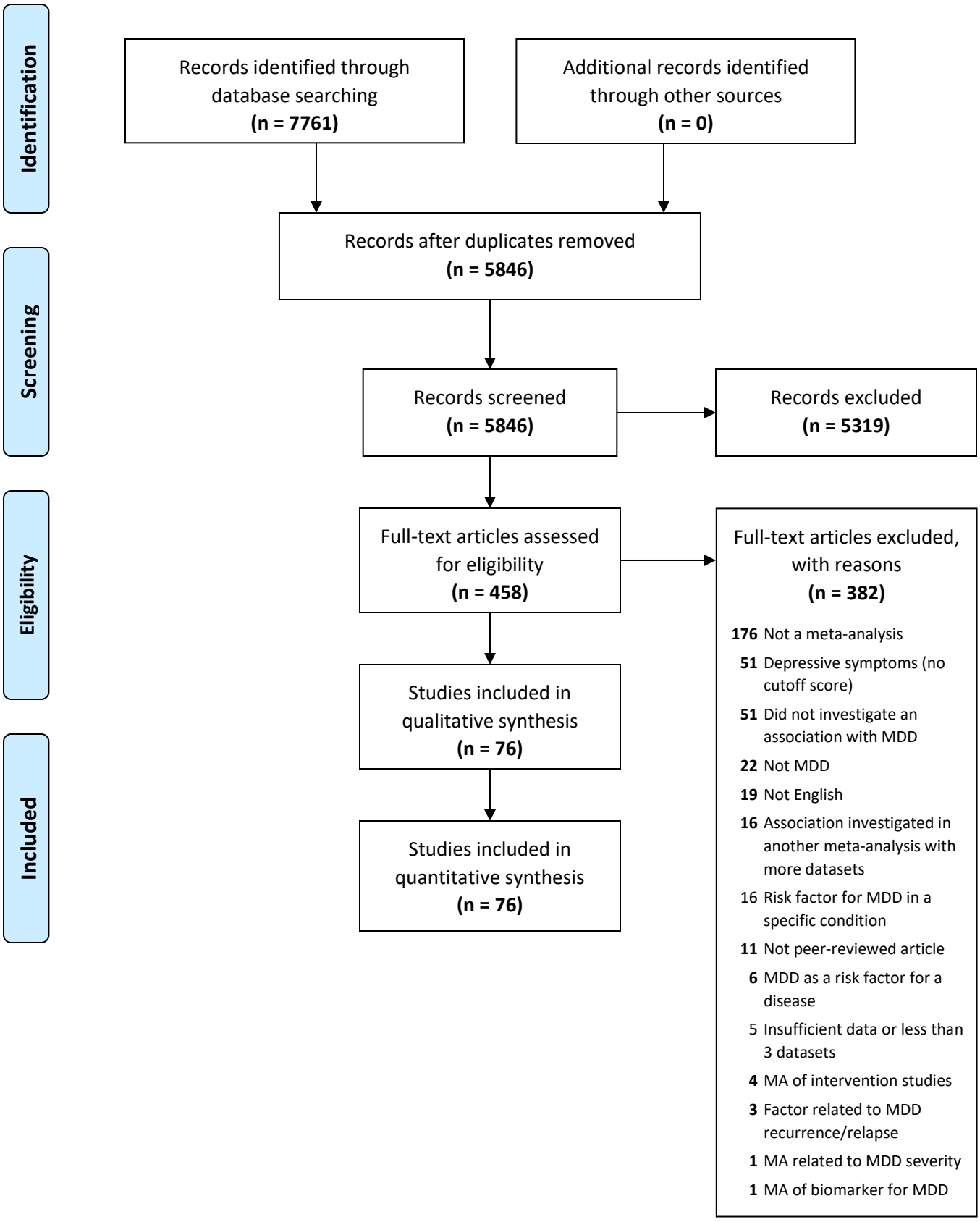
* **Evidence classification: I** = convincing evidence criteria ($p < 10^{-6}$ per random-effects model; over than 1000 cases; no excess of significance bias; no small studies effects; small heterogeneity; prediction interval not including the null); **II** = highly-suggestive evidence criteria (not meeting type I criteria, but $p < 10^{-6}$ per random-effects model; over than 1000 cases; effect size of the largest study not crossing the null); **III** = suggestive evidence criteria (not meeting type I or II criteria, but $p < 10^{-3}$ per random-effects model; over than 1000 cases); **Weak** evidence criteria (all other associations with $p < 0.05$ per random-effects model); **NS** = non-significant associations; **NE** = not evaluated due to lack of individual study information.

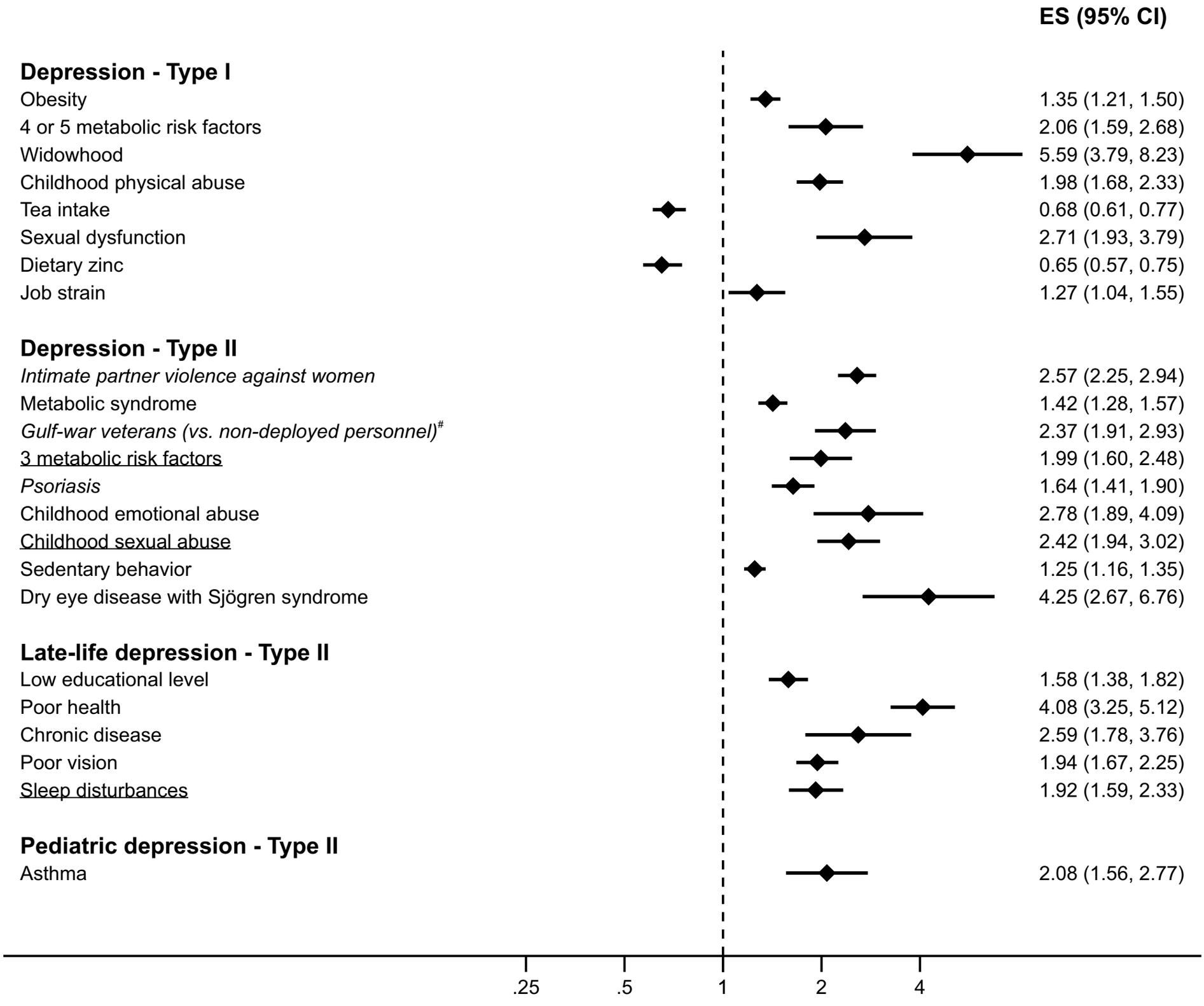
Table 3. Risk factors for depression reported in Mendelian randomization studies.

Risk factor (Reference)	Population	Sample size/ Number of events (Number of studies)*	Genetic instruments (GI)	Variance (R^2) explained by GI (%)	Type of metric	Estimate of effect (95% CI)	P value	Statistical power
Alcohol consumption (Almeida, 2014)	Australia	3,873/610 (DS)	*ADH1B* rs1229984	0.11	OR	GA = 0.99 (0.70, 1.40) AA = 1.65 (0.54, 5.08)	0.946	0.05 0.07
Smoking (Bjorngaard, 2013)	Norway	56,664/NA (DS)	*CHRNA5-CHRNA3-CHRNB4* rs1051730	0.4	OR	1.02 (0.98, 1.06)	0.3120	NA
Obesity (Hung, 2014)	USA/United Kingdom/Europe	3,222/2,430 (3) (DEP)	*FTO* rs3751812	NA	β	-0.03 (-0.18, 0.13)	0.730	NA
Obesity (Hung, 2014)	USA/United Kingdom/Europe	3,222/2,430 (3) (DEP)	wGRS from 32 SNPS associated to BMI in GWAS studies	NA	β	-0.02 (-0.11, 0.07)	0.620	NA
Age of menarche (continuous genetic instrument) (Sequeira, 2017)	United Kingdom	2,404 (DS at 14yo) 2,145 (DS at 17 yo) 1,570 (DS at 19 yo) 1,910 (DEP at 18 yo)	nwGRS from 123 SNPSs associated with menarche in GWAS	NA	OR	1.020 (1.005, 1.04) 1.002 (0.99, 1.02) 1.001 (0.98, 1.02) 1.004 (0.98, 1.03)	0.010 0.780 0.880 0.730	NA NA NA NA
Age of menarche (categorical genetic instrument) (Sequeira, 2017)	United Kingdom	2,404 (DS at 14yo) 2,145 (DS at 17 yo)	nwGRS from 123 SNPs associated with menarche in GWAS (2nd, 3rd and 4th quartiles vs. 1st	NA	OR	1.62 (1.18, 2.25) 1.38 (0.99, 1.92) 1.74 (1.26, 2.40) 0.95 (0.71, 1.26) 0.90 (0.68, 1.21) 1.08 (0.82, 1.43)	0.003 0.050 0.001 0.730 0.490 0.590	NA NA

		1,570 (DS at 19 yo)	quartile)			0.74 (0.52, 1.04) 0.81 (0.58, 1.12) 1.06 (0.76, 1.46)	0.080 0.200 0.740	NA
		1,910 (DEP at 18 yo)				0.84 (0.55, 1.28) 0.83 (0.54, 1.26) 1.14 (0.76, 1.70)	0.410 0.370 0.530	NA
Tobacco smoking (Wium-Andersen, 2015)	Denmark	40,014/771 (CDD)	*CHRNA3* gene cluster rs1051730	0.4	OR	CT = 1.02 (0.88, 1.18) TT = 0.85 (0.66, 1.10)	0.440	0.05 0.06
Alcohol consumption (Wium-Andersen, 2015)	Denmark	78,154/1,106 (CDD)	*ADH1B* rs1229984 and *ADH1C* rs698 genotype combinations (2 vs. 1, 3 vs. 1, 4 vs. 1)	0.12	OR	1.18 (0.85, 1.63) 1.10 (0.80, 1.53) 1.40 (1.00, 1.96)	0.060	0.05 0.05 0.07

Abbreviations: DS = depression assessed by a screening instrument with a cutoff score; **DEP** = depression diagnosed with structured interview; **CDD** = clinical diagnosis based on ICD criteria for depression; **nwGRS** = non-weighted genetic risk score; **wGRS** = weighted genetic risk score


Identification
Screening
Eligibility
Included
Records identified through database searching (n = 7761)
Additional records identified through other sources (n = 0)
Records after duplicates removed (n = 5846)
Records screened (n = 5846)
Records excluded (n = 5319)
Full-text articles assessed for eligibility (n = 458)
Full-text articles excluded, with reasons (n = 382)
176 Not a meta-analysis
51 Depressive symptoms (no cutoff score)
51 Did not investigate an association with MDD
22 Not MDD
19 Not English
16 Association investigated in another meta-analysis with more datasets
16 Risk factor for MDD in a specific condition
11 Not peer-reviewed article
6 MDD as a risk factor for a disease
5 Insufficient data or less than 3 datasets
4 MA of intervention studies
3 Factor related to MDD recurrence/relapse
1 MA related to MDD severity
1 MA of biomarker for MDD
Studies included in qualitative synthesis (n = 76)
Studies included in quantitative synthesis (n = 76)


ES (95% CI)
Depression - Type I
Obesity
1.35 (1.21, 1.50)
4 or 5 metabolic risk factors
2.06 (1.59, 2.68)
Widowhood
5.59 (3.79, 8.23)
Childhood physical abuse
1.98 (1.68, 2.33)
Tea intake
0.68 (0.61, 0.77)
Sexual dysfunction
2.71 (1.93, 3.79)
Dietary zinc
0.65 (0.57, 0.75)
Job strain
1.27 (1.04, 1.55)
Depression - Type II
Intimate partner violence against women
2.57 (2.25, 2.94)
Metabolic syndrome
1.42 (1.28, 1.57)
Gulf-war veterans (vs. non-deployed personnel)#
2.37 (1.91, 2.93)
3 metabolic risk factors
1.99 (1.60, 2.48)
Psoriasis
1.64 (1.41, 1.90)
Childhood emotional abuse
2.78 (1.89, 4.09)
Childhood sexual abuse
2.42 (1.94, 3.02)
Sedentary behavior
1.25 (1.16, 1.35)
Dry eye disease with Sjögren syndrome
4.25 (2.67, 6.76)
Late-life depression - Type II
Low educational level
1.58 (1.38, 1.82)
Poor health
4.08 (3.25, 5.12)
Chronic disease
2.59 (1.78, 3.76)
Poor vision
1.94 (1.67, 2.25)
Sleep disturbances
1.92 (1.59, 2.33)
Pediatric depression - Type II
Asthma
2.08 (1.56, 2.77)
.25
.5
1
2
4

Declarations of interests

Authors report no direct competing interests for the present study.

SUPPLEMENTARY ONLINE MATERIAL

Supplementary Text – Detailed search strategy

PubMed/MEDLINE (through August 3rd, 2017)

#1: depression[Mesh]
#2: (meta-analysis OR systematic review) [Field: Title/Abstract]
#3: (Mendelian Randomization Analysis/ or Mendelian randomi*)
#4: #2 or #3
#5: #1 and #4

PsycINFO via OVID (through August 3rd, 2017)

#1: depress*.ti,ab
#2: (meta-analysis or systematic review).ti,ab
#3: Mendelian Randomization Analysis/ or Mendelian randomi*.mp.
#4: #2 or #3
#5: #1 and #4

Table S1. References excluded after full-text review, with reasons.

Reference	Reason for exclusion
Abate, 2013	Risk factor for MDD in specific condition
Abou Abbas, 2015	Risk factor for MDD in specific condition
Accortt, 2015	Not a meta-analysis
Adida, 2014	Not English
Aggarwal, 2015	Not a meta-analysis
Agius, 2016	Risk factor for MDD in specific condition
Ahlbom, 2001	Depressive symptoms (no cutoff)
Alalu, 2000	Not peer-reviewed
Albers, 2011	Not a meta-analysis
Alhasanat, 2017	Risk factor for MDD in specific condition
Ali, 2006	Association investigated in another meta-analysis
Almeida, 2013	Not a meta-analysis
Al-Modallal, 2008	Not a meta-analysis
Al-Sharfi, 2016	Not a meta-analysis
Alvarez-Segura, 2014	Not a meta-analysis
Alvaro, 2013	Not a meta-analysis
Amado, 2015	Depressive symptoms (no cutoff)
Amanatkar, 2014	Meta-analysis of intervention studies
Ammerman, 2010	Not a meta-analysis
Amouroux, 2008	Not English
Anderson, 2001	Association investigated in another meta-analysis
Anderson, 2017	Risk factor for MDD in specific condition
Ando, 2017	Not a meta-analysis
Andrade, 2008	Not English
Angold, 1999	Not a meta-analysis
Apostolova, 2008	Not a meta-analysis
Araujo, 2010	Not a meta-analysis
Arbelaez Moreno, 2005	Not peer-reviewed
Arfken, 2014	MDD as a risk factor for a disease
Aschenbrenner, 2003	Not English
Atlantis, 2008	Not a meta-analysis
Ayerbe, 2013	Did not investigate an association with MDD
Ba, 2017	Not a meta-analysis
Baker, 2016	Not a meta-analysis
Bannai, 2014	Not a meta-analysis
Barcelos-Ferreira, 2010	Did not investigate an association with MDD
Barnard, 2006	Not a meta-analysis
Barnard, 2010	Not a meta-analysis
Barry, 2011	Depressive symptoms (no cutoff)
Barry, 2014	Not a meta-analysis
Barskova, 2009	Not a meta-analysis
Bartoli, 2017	Not MDD
Bas-Sarmiento, 2017	Not a meta-analysis
Bastidas-Bilbao, 2014	Not English
Baumeister, 2005	Not English
Baune, 2012	Not a meta-analysis
Baune, 2012	Not a meta-analysis
Bautovich, 2014	Not a meta-analysis
Becker, 2017	Depressive symptoms (no cutoff)
Beekman, 1999	Not a meta-analysis
Beekman, 1999	Not a meta-analysis
Behle, 2016	Not major depressive disorder
Belhadj Kouider, 2015	Not published in English
Bell, 2016	Risk factor for MDD in specific condition
Benca, 1992	Not major depressive disorder

Reference	Reason for exclusion
Bennett, 1994	Depressive symptoms (no cutoff)
Bennett, 2004	Risk factor for MDD in specific condition
Berger-Greenstein, 2007	Not a meta-analysis
Berkman, 2007	Not a meta-analysis
Black, 2015	Not a meta-analysis
Blay, 2016	Association investigated in another meta-analysis
Bolsover, 2014	Not a meta-analysis
Bonde, 2008	Not a meta-analysis
Bottino, 2015	Not a meta-analysis
Boylan, 2007	Not a meta-analysis
Brandt, 2009	Not a meta-analysis
Brayne, 2005	Not a meta-analysis
Breyer, 2013	Not a meta-analysis
Bronsard, 2016	Did not investigate an association with MDD
Bronstein, 2011	Not a meta-analysis
Brotto, 2016	Not a meta-analysis
Brown, 2000	Not a meta-analysis
Brown, 2009	Not a meta-analysis
Buchberger, 2016	Depressive symptoms (no cutoff)
Buchtemann, 2012	Not a meta-analysis
Buigues, 2015	Not a meta-analysis
Burke, 2015	Depressive symptoms (no cutoff)
Burnett, 2011	Not major depressive disorder
Burnett, 2011	Not major depressive disorder
Burt, 2009	Not a meta-analysis
Cabrera-Alvarez, 2004	Not peer-reviewed
Cairns, 2014	Depressive symptoms (no cutoff)
Camoes Barbosa, 2011	Not English
Camus, 2004	Not a meta-analysis
Carli, 2012	Not a meta-analysis
Carter, 2006	Not major depressive disorder
Carvalho, 2016	Not a meta-analysis
Castro-de-Araujo, 2013	Did not investigate an association with MDD
Cerda, 2010	Not a meta-analysis
Chakrabarty, 2015	Depressive symptoms (no cutoff)
Chatillon, 2010	Not English
Chen, 1999	Did not investigate an association with MDD
Chen, 2012	Not a meta-analysis
Cheng, 2015	Not a meta-analysis
Cheng, 2015	Not a meta-analysis
Chi, 2015	Did not investigate an association with MDD
Chmielowska, 2017	Not a meta-analysis
Ciesla, 2001	No individual study information available
Cizza, 2010	MDD as a risk factor for a disease
Cojocaru, 2015	Not a meta-analysis
Conner, 2008	Depressive symptoms (no cutoff)
Conner, 2008	Depressive symptoms (no cutoff)
Conner, 2009	Risk factor for MDD in specific condition
Copeland, 1999	Did not investigate an association with MDD
Corruble, 1996	Not a meta-analysis
Cuijpers, 2005	Not a meta-analysis
Davydow, 2008	Not a meta-analysis
Davydow, 2008	Not a meta-analysis
Davydow, 2009	Not a meta-analysis
Dawes, 2016	Factor related to MDD recurrence/relapse
Dawson, 2014	Not a meta-analysis
de Boer, 2011	Depressive symptoms (no cutoff score)

Reference	Reason for exclusion
de Kruif, 2015	Not English
Delahaije, 2013	Not a meta-analysis
Delville, 2008	Not a meta-analysis
Derom, 2013	Not a meta-analysis
Derom, 2013	Not a meta-analysis
Devenish, 2017	Not a meta-analysis
Devries, 2013	Depressive symptoms (no cutoff score)
Di Manno, 2015	Not a meta-analysis
Dias, 2015	Not a meta-analysis
Dias, 2015	Not major depressive disorder
Dickens, 2002	Depressive symptoms (no cutoff score)
Dong, 2013	Not a meta-analysis
Dorstyn, 2017	Not environmental risk factor association
Dowling, 2015	Did not investigate an association with MDD
Dunne, 2017	Not MDD
Dyrbye, 2006	Not a meta-analysis
Egede, 2010	Not a meta-analysis
Elhai, 2017	Not a meta-analysis
Elzubeir, 2010	Not a meta-analysis
Erskine, 2016	Not major depressive disorder
Even, 2004	Not published in English
Even, 2004	Not published in English
Fabricatore, 2011	Depressive symptoms (no cutoff score)
Falah-Hassani, 2017	Risk factor for MDD in specific condition
Faller, 2008	Not published in English
Fang, 2015	Not major depressive disorder
Faria-Schutzer, 2017	Risk factor for MDD in specific condition
Farrokhi, 2014	Meta-analysis related to MDD severity
Fazel, 2008	Did not investigate an association with MDD
Ferrari, 2013	Not a meta-analysis
Ferrari, 2013	Did not investigate an association with MDD
Flensborg-Madsen, 2011	Not a meta-analysis
Fond, 2014	Depressive symptoms (no cutoff score)
Freire, 2013	Not a meta-analysis
Gadermann, 2012	Did not investigate an association with MDD
Gage, 2013	Not a meta-analysis
Gagliese, 2007	Not a meta-analysis
Gaida, 2016	Not a meta-analysis
Gaitatzis, 2004	Not a meta-analysis
Gale, 2011	Association investigated in a larger meta-analysis
Galvao, 2014	Less than 3 datasets
Gao, 2015	Less than 3 datasets
Gavard, 1993	Not a meta-analysis
Gavin, 2005	Not major depressive disorder
Geocze, 2013	Not a meta-analysis
Geulayov, 2012	Factor related to MDD recurrence/relapse
Golding, 1997	Not major depressive disorder
Golding, 1999	Did not investigate an association with MDD
Goldner, 2014	Did not investigate an association with MDD
Grosso, 2016	Association investigated in another meta-analysis
Gu, 2013	Did not investigate an association with MDD
Hackett, 2005	Not a meta-analysis
Hackett, 2005	Did not investigate an association with MDD
Haisfield-Wolfe, 2009	Not a meta-analysis
Hakulinen, 2015	Depressive symptoms (no cutoff)
Harris, 2002	Not peer-reviewed
Harrison, 2015	Not a meta-analysis

Reference	Reason for exclusion
Hartka, 1991	Depressive symptoms (no cutoff)
Hasan, 2013	Association investigated in another meta-analysis
Hellmann-Regen, 2013	Not a meta-analysis
Hennegan, 2015	Not a meta-analysis
Hershcovis, 2010	Depressive symptoms (no cutoff)
Hibbeln, 2014	Not a meta-analysis
Hoare, 2014	Not a meta-analysis
Hoare, 2016	Not a meta-analysis
Holzel, 2011	Not a meta-analysis
Hope, 2014	Not a meta-analysis
Hotopf, 2002	Not a meta-analysis
Hu, 2015	Depressive symptoms (no cutoff)
Huang, 2010	Not major depressive disorder
Huang, 2015	Depressive symptoms (no cutoff)
Ibrahim, 2013	Did not investigate an association with MDD
Inder, 2016	Not a meta-analysis
Infurna, 2016	Depressive symptoms (no cutoff)
Jane Costello, 2006	Did not investigate an association with MDD
Johnson, 2013	Not a meta-analysis
Joiner, 1995	Depressive symptoms (no cutoff)
Jokela, 2012	Depressive symptoms (no cutoff)
Jokela, 2014	Depressive symptoms (no cutoff)
Jonassaint, 2016	Risk factor for MDD in specific condition
Jumper, 1995	Depressive symptoms (no cutoff)
Kalt, 2013	Not a meta-analysis
Kan, 2013	Depressive symptoms (no cutoff)
Kawada, 2014	Not a meta-analysis
Khalid, 2016	Investigated in another meta-analysis
Killian-Farrell, 2017	Not peer-reviewed
Kim, 2008	Not a meta-analysis
King-Wing Ma, 2016	Not a meta-analysis
Koenig, 2001	Not a meta-analysis
Koenig, 2009	Not a meta-analysis
Kohli, 2016	Depressive symptoms (no cutoff)
Kornischka, 2008	Not published in English
Kornischka, 2008	Not published in English
Kraaij, 2002	Depressive symptoms (no cutoff score)
Krebber, 2014	Did not investigate an association with MDD
Kuehner, 2003	Not a meta-analysis
Kuo, 2017	Not a meta-analysis
Lai, 2014	Association investigated in a larger meta-analysis
Langan, 2017	Depressive symptoms (no cutoff)
Lei, 2016	Did not investigate an association with MDD
Lejoyeux, 2011	Meta-analysis of intervention studies
Lewis, 2011	Risk factor for MDD in specific condition
Li, 2014	Did not investigate an association with MDD
Li, 2014	Depressive symptoms (no cutoff score)
Li, 2015	Association investigated in another meta-analysis
Li, 2015	Did not investigate an association with MDD
Li, 2016	Association investigated in another meta-analysis
Limburg, 2016	Not MDD
Lindert, 2009	Did not investigate an association with MDD
Lindert, 2014	Association investigated in another meta-analysis
Maggi, 2015	Not a meta-analysis
Magyar-Russell, 2011	Not a meta-analysis
Maniglio, 2010	Not a meta-analysis
Mannan, 2016	Depressive symptoms (no cutoff score)

Reference	Reason for exclusion
Marqueling, 2005	Not a meta-analysis
Marqueling, 2007	Not a meta-analysis
Martinez-Gonzalez, 2016	Not a meta-analysis
Martins, 2016	Depressive symptoms (no cutoff)
Mata, 2015	Depressive symptoms (no cutoff score)
Matcham, 2013	Did not investigate an association with MDD
Matcham, 2014	Not a meta-analysis
Mathewson, 2017	Not environmental risk factor association
Mausbach, 2011	Depressive symptoms (no cutoff score)
McCarten, 2004	Not peer-reviewed
McDougall, 2007	Not a meta-analysis
McKechnie, 2014	Not a meta-analysis
McLeod, 2007	Depressive symptoms (no cutoff score)
Medisauskaite, 2017	Not MDD
Medlow, 2014	Not a meta-analysis
Melero, 2017	Not a meta-analysis
Mendenhall, 2014	Not a meta-analysis
Mendes, 2012	Not a meta-analysis
Menzel, 2008	Not a meta-analysis
Meszaros, 2012	Not a meta-analysis
Mezuk, 2008	Association investigated in another meta-analysis
Milan de Souza, 2006	Not published in English
Milazzo, 2016	Depressive symptoms (no cutoff)
Mirza, 2004	Not a meta-analysis
Mitchell, 2011	Did not investigate an association with MDD
Mitchell, 2012	Depressive symptoms (no cutoff score)
Mohamed, 2014	Not a meta-analysis
Moll, 2011	Not published English
Moran, 2014	Did not investigate an association with MDD
Moreira-Almeida, 2006	Not a meta-analysis
Morris, 2012	MDD as a risk factor for a disease
Murakami, 2010	Not a meta-analysis
Murtagh, 2007	Not a meta-analysis
Musisi, 2014	Book chapter
Nakimuli-Mpungu, 2012	Did not investigate an association with MDD
Nelson, 2016	Not a meta-analysis
Nelson, 2017	Factor related to MDD recurrence/relapse
Neuendorf, 2016	Did not investigate an association with MDD
Nie, 2013	Not published in English
Niu, 2016	Not a meta-analysis
Norwood, 2000	Not peer-reviewed
Oddone Paolucci, 2001	Depressive symptoms (no cutoff score)
Oliver, 201 3	Not a meta-analysis
Osborn, 2007	Not a meta-analysis
Palagini, 2013	Not a meta-analysis
Palmer, 2013	Did not investigate an association with MDD
Parker, 2016	Risk factor for MDD in specific condition
Pemberton, 2016	Not a meta-analysis
Persons, 2016	Meta-analysis of a biomarker
Pinquart, 2003	Depressive symptoms (no cutoff score)
Preiss, 2013	Not a meta-analysis
Priest, 2013	Not a meta-analysis
Puccio, 2016	Depressive symptoms (no cutoff score)
Purewal, 2017	Not a meta-analysis
Quinones-Munoz, 2001	Not peer-reviewed
Quirk, 2013	Not a meta-analysis
Reagon, 2016	Not MDD

Reference	Reason for exclusion
Reijnders, 2008	Not a meta-analysis
Ren, 2014	Did not investigate an association with MDD
Renn, 2011	Not a meta-analysis
Reppermund, 2016	Book chapter
Rhyner, 2016	MA of intervention studies
Ribeiro, 2015	Not a meta-analysis
Richardson, 2013	No individual study information available
Rona, 2016	Not major depressive disorder
Rooke, 2008	MDD as a risk factor for a disease
Rooney, 2011	Not a meta-analysis
Rotella, 2013	Association investigated in a larger meta-analysis
Roy, 2012	Not a meta-analysis
Rueger, 2016	Depressive symptoms (no cutoff score)
Rutledge, 2006	Did not investigate an association with MDD
Rytwinski, 2013	Did not investigate an association with MDD
Sajjadi, 2013	Not a meta-analysis
Salguero, 2011	Not a meta-analysis
Sanhueza, 2013	Not a meta-analysis
Sanhueza, 2013	Not a meta-analysis
Santini, 2015	Not a meta-analysis
Schlosser, 2016	Not a meta-analysis
Scholten, 2016	Not major depressive disorder
Schubert, 1993	Depressive symptoms (no cutoff score)
Schwarzbach, 2014	Not a meta-analysis
Schwarzbach, 2014	Not a meta-analysis
Scott, 2016	Did not investigate an association with MDD
Seitz, 2010	Not a meta-analysis
Sherr, 2011	Meta-analysis of intervention studies
Shore, 2017	Not a meta-analysis
Siegrist, 2008	Not a meta-analysis
Silva, 2014	Did not investigate an association with MDD
Singer, 2006	Depressive symptoms (no cutoff score)
Siskind, 2016	Risk factor for MDD in a specific condition
Slewa-Younan, 2015	Not a meta-analysis
Slewa-Younan, 2015	Not a meta-analysis
Smith, 2012	Depressive symptoms (no cutoff)
Spoletini, 2008	Not a meta-analysis
Stirling, 2015	Not a meta-analysis
Storm, 2013	Not a meta-analysis
Stubbs, 2016	Did not investigate an association with MDD
Suchert, 2015	Not a meta-analysis
Sutterland, 2015	Association investigated in a larger meta-analysis
Swinnen, 2007	Not major depressive disorder
Tang, 2014	Risk factor for MDD in a specific condition
Tesser, 1998	Not a meta-analysis
Thomas, 2004	Not a meta-analysis
Thombs, 2007	Not a meta-analysis
Trevillion, 2015	Not a meta-analysis
Tsai, 2014	Did not investigate an association with MDD
Valkanova, 2013	Association investigated in a larger meta-analysis
van Aalst, 2017	Not a meta-analysis
van den Akker, 2016	Risk factor for MDD in specific condition
van Ede, 1999	Not a meta-analysis
Vancampfort, 2014	MDD as a risk factor for a disease
Vancampfort, 2015	MDD as a risk factor for a disease
Vasquez-Vera, 2017	Not a meta-analysis
Verkaik, 2007	Not a meta-analysis

Reference	Reason for exclusion
Villegas, 2011	Not major depressive disorder
Vink, 2008	Not a meta-analysis
Wagner, 2012	Not published in English
Walker, 2013	Not a meta-analysis
Wallace, 2017	Not a meta-analysis
Walton, 2015	Not a meta-analysis
Wang, 2013	Not a meta-analysis
Wang, 2013	Not a meta-analysis
Ward, 2013	Not a meta-analysis
Washington, 2017	Not a meta-analysis
Watts, 2014	Did not investigate an association with MDD
Watts, 2015	Did not investigate an association with MDD
Weich, 2009	Not a meta-analysis
Weinstein, 2017	Not a meta-analysis
Weobong, 2015	Not a meta-analysis
Wesselhoft, 2016	Not peer-reviewed
Wilde, 2014	Not major depressive disorder
Wilder Schaaf, 2013	Not a meta-analysis
Williams, 2015	Did not investigate an association with MDD
Wojcik, 2013	Association investigated in a larger meta-analysis
Yang, 2013	Did not investigate an association with MDD
Yap, 2014	Depressive symptoms (no cutoff score)
Yap, 2015	Depressive symptoms (no cutoff score)
Yohannes, 2000	Not a meta-analysis
Yoon, 2013	Not major depressive disorder
Zaninotto, 2016	Not a meta-analysis
Zhang, 2012	Depressive symptoms (no cutoff score)
Zieger, 2010	Not a meta-analysis

Table S2. Methodological quality assessment of eligible meta-analyses with AMSTAR.

Reference	AMSTAR Question											Total
	1	2	3	4	5	6	7	8	9	10	11	
Agtmaal, 2017	1	1	1	0		1	1		1	1	1	**8**
Anglin, 2013	0	1	1	1	1	1	1	1	1	1	0	**9**
Atlantis, 2012	0	0	1	1	1	1	1	1	1	1	0	**8**
Atlantis, 2013	0	1	1	0	0	0	1	1	1	1	0	**6**
Baglioni, 2011	0	1	1	0	0	1	0	0	0	1	0	**4**
Bao, 2017	1	1	1	0	0	1	1	1	1	1	0	**8**
Beydoun, 2012	0	0	1	0	0	1	0	0	1	1	0	**4**
Blore, 2015	1	0	0	0	0	1	1	1	1	0	0	**5**
Bonde, 2016	1	1	1	0	0	1	1	1	1	1	0	**8**
Chaiton, 2009	0	0	1	0	0	1	1	0	1	1	0	**5**
Chang-Quan, 2010	0	1	0	0	0	1	0	0	1	1	0	**4**
Chang-Quan, 2010	0	1	0	0	0	1	0	0	1	1	0	**4**
Chen, 2010	1	1	1	1	0	1	1	1	1	1	0	**9**
Chen, 2016	1	1	1	0	0	1	1	0	1	1	0	**7**
Cheungpasitporn, 2015	1	0	1	0	0	1	1	1	1	1	0	**7**
Cole, 2003	0	0	1	0	0	1	1	1	1	0	0	**5**
de Wit, 2010	0	0	1	0	0	1	0	0	1	1	0	**4**
Diprose, 2016	0	0	1	0	0	1	0	0	0	0	0	**2**
Dokras, 2011	0	1	0	0	0	1	0	0	1	1	0	**4**
Dong, 2015	1	1	1	0	0	1	1	0	1	1	0	**7**
Dowlatshahi, 2014	0	1	0	0	0	1	1	1	1	1	0	**6**
Fiest, 2013	0	1	1	1	0	1	1	1	1	1	0	**8**
Gilbody, 2007	1	1	1	1	0	1	0	0	1	1	0	**7**
Grosso, 2016	0	0	1	0	0	1	1	0	1	1	0	**5**
Hasan, 2015	1	0	1	0	0	1	0	0	1	0	0	**4**
Ho, 2014	1	1	1	0	0	1	0	0	1	0	0	**5**
Huang, 2010	0	1	1	1	0	1	0	0	1	1	0	**6**
Jokela, 2014	0	0	1	0	0	1	0	0	1	0	0	**3**
Kim, 2016	1	1	1	0	0	1	0	0	1	1	0	**6**
Kisely, 2017	1	1	1	0	0	1	1	1	1	1	0	**8**
Lev-Ran, 2014	1	1	0	0	0	1	1	1	1	1	0	**7**
Li, 2016	1	1	1	0	0	0	1	1	1	1	0	**7**
Li, 2016	1	1	1	1	0	1	1	0	1	1	0	**8**
Li, 2017	1	1	1	0	0	1	0	0	1	1	0	**6**
Liu, 2016	0	1	1	0	0	1	0	0	1	1	0	**5**
Lorant, 2003	0	0	1	1	0	1	1	0	1	1	0	**6**
Loret de Mola, 2014	1	1	1	0	0	0	0	0	1	1	0	**5**
Lu, 2012	0	1	1	0	0	1	0	0	1	1	0	**5**
Luger, 2014	1	0	1	0	0	1	0	0	1	0	0	**4**
Luppino, 2010	0	1	1	0	0	1	1	1	1	1	0	**7**
Madsen, 2017	1	1	1	0	0	1	1	1	1	0	0	**7**
Mandelli, 2015	1	0	1	0	0	1	1	1	1	1	0	**7**
Mannan, 2016	1	1	1	0	0	1	1	1	1	1	0	**8**
Mitchell, 2013	1	0	1	1	0	1	1	1	1	1	0	**8**
Nouwen, 2010	0	1	1	0	0	1	1	0	1	1	0	**6**
Nouwen, 2011	0	1	1	0	0	1	0	0	1	1	0	**5**
Onrust, 2006	0	0	1	1	0	1	0	0	0	0	0	**3**
Osborn, 2014	0	0	1	0	0	0	0	0	1	1	0	**3**
Pan, 2012	0	1	1	1	0	1	0	0	1	1	0	**6**
Parsaik, 2014	1	1	1	1	0	0	1	1	1	0	0	**7**
Petridou, 2016	1	1	0	0	0	1	0	0	0	1	0	**4**
Psaltopoulou, 2013	0	1	0	1	1	0	1	1	1	1	0	**7**
Puthran, 2016	1	1	1	0	0	0	1	1	1	1	0	**7**
Rahe, 2014	1	1	1	0	0	1	0	1	0	0	0	**5**

Reference	AMSTAR Question											Total
Richardson, 2015	1	1	1	0	0	1	1	1	1	0	0	**7**
Secinti, 2017	1	0	1	0	0	1	1	1	1	1	1	**8**
Tarricone, 2012	0	1	1	0	0	1	0	0	1	0	0	**4**
Tong, 2016	1	1	1	0	0	1	1	1	1	1	1	**9**
Ttofi, 2011	0	0	1	1	1	1	0	0	1	0	0	**5**
Wan, 2016	1	1	1	0								**3**
Wang, 2014	1	1	1	0	0	1	1	1	1	0	0	**7**
Wang, 2016	0	1	0	0	0	1	0	0	1	1	0	**4**
Watanabe, 2016	1	1	0	0	0	1	0	0	1	1	0	**5**
Xiu-Ying, 2012	0	1	1	0	0	1	0	0	1	1	0	**5**
Yan, 2011	0	1	0	0	0	1	0	0	1	1	0	**4**
Younossi, 2016	1	1	1	0	0	1	1	1	1	1	1	**9**
Zeng, 2016	1	1	1	0	0	1	0	0	1	1	1	**7**
Zhai, 2015	1	1	1	0	0	1	1	0	1	1	0	**7**
Zhai, 2015	1	1	1	0	0	1	1	0	1	1	0	**7**
Zhao, 2012	0	1	1	0	0	1	0	0	1	1	0	**5**

Table S3. Characteristics, quantitative synthesis, and assessments of epidemiologic credibility and methodological quality of the 6 eligible meta-analyses investigating environmental risk factors for depression in youths.

Reference	Risk factor	Total number of cases/ controls	Number of primary studies	Effect size metric	Random effects summary effect size (95% CI)	P random	95% PI	I^2	Small-study effects/ excess statistical significance	Level of evidence	AMSTAR
Lu, 2012	Asthma	2828/11736	7	OR	2.08 (1.56 − 2.77)	< 10-6	0.88 − 4.91	76	No/No	II	5
Chaiton, 2009	Smoking	NA	3	OR	1.91 (0.98 − 3.73)	0.058	0.00 − 5591.77	82	No/NE	NS	5
Li, 2016	Any childhood maltreatment	1400/2142	5	OR	2.03 (1.37–3.01)	< 0.001	-0.57 – 1.99	63.2	No/No	III	8
Mannan, 2016	Obesity	2051/20738	9	RR	1.43 (1.20–1.72)	< 0.001	-0.03 – 0.75	25.0	No/NE	III	8
Secinti, 2017	Childhood chronic physical illness - Cancer	3907/4271	18	OR	1.19 (0.99–1.44)	0.060	-0.42 – 0.77	60.2	No/No	NS	8
Secinti, 2017	Childhood chronic physical illness - Diabetes	616/407	6	OR	1.37 (0.85–2.19)	0.196	-1.16 – 1.79	67.6	No/Yes	NS	8

Table S4. Sensitivity analysis limited to prospective studies for associations supported by convincing or highly suggestive evidence in the main meta-analyses.

Reference	Risk factor	Total number of cases/ controls	Number of primary studies	Effect size metric	Random effects summary effect size (95% CI)	P random	95% PI	I^2	Small-study effects/ excess statistical significance	Level of evidence*
Major depressive disorder										
Dong, 2015	Tea consumption	1166/7834	5	RR	0.85 (0.69 – 1.04)	0.108	0.50 – 1.44	32	No/No	NS
Beydoun, 2012	Intimate partner violence against women[a]	NE	NE	NE	NE	NE	NE	NE	NE	NE
Atlantis, 2012	Sexual dysfunction	5488/5683	6	OR	2.71 (1.93 – 3.79)	$< 10^{-6}$	1.13 – 6.50	41	No/No	I
Pan, 2012	Metabolic syndrome	2316/27034	9	OR	1.48 (1.17 – 1.87)	0.001	0.74 – 2.95	61	No/Yes	Weak
Blore, 2015	Gulf-war veterans (vs. non-deployed personnel)†	NE	NE	NE	NE	NE	NE	NE	NE	NE
Li, 2017	Dietary zinc†	NE	NE	NE	NE	NE	NE	NE	NE	NE
Jokela, 2014	Obesity	7673/11413	8	OR	1.35 (1.21 – 1.50)	$< 10^{-6}$	1.18 – 1.54	0	No/No	I
Jokela, 2014	3 metabolic risk factors	3014/9586	8	OR	1.99 (1.60 – 2.48)	$< 10^{-6}$	1.20 – 3.30	35	Yes/No	II
Jokela, 2014	4 or 5 metabolic risk factors	1191/9586	8	OR	2.06 (1.59 – 2.68)	$< 10^{-6}$	1.19 – 3.56	24	No/No	I
Onrust, 2006	Widowhood (vs. any other marital status)	2720/4868	5	RR	5.59 (3.79 – 8.23)	$< 10^{-6}$	2.09 – 14.97	30	No/No	I
Dowlatshahi, 2014	Psoriasis†	NE	NE	NE	NE	NE	NE	NE	NE	NE
Zhai, 2015	Sedentary behavior†	NE	NE	NE	NE	NE	NE	NE	NE	NE
Mandelli, 2015	Emotional abuse in childhood	3329/9644	4	OR	2.22 (1.41 – 3.47)	$> 10^{-6}$ but $< 10^{-3}$	0.27 – 17.88	92	No/No	III
Mandelli, 2015	Sexual abuse in childhood	3621/10991	7	OR	2.42 (1.77 – 3.31)	$< 10^{-6}$	0.89 – 6.57	83	No/No	II
Mandelli, 2015	Physical abuse in childhood	3054/10098	4	OR	1.95 (1.74 – 2.17)	$< 10^{-6}$	1.42 – 2.67	14	No/No	I
Madsen, 2017	Job strain	1909/25552	7	OR	1.77 (1.46–2.13)	$< 10^{-6}$	1.02 – 14.01	24	No/No	I
Wan, 2016	Dry eye with Sjögren syndrome†	NE	NE	NE	NE	NE	NE	NE	NE	NE
Late-life depression										
Chang-Quan, 2010	Low educational level	3957/6374	12	RR	1.49 (1.16 – 1.91)	0.002	0.64 – 3.49	75	No/NE	Weak
Chang-Quan, 2010	Poor health†	NE	NE	NE	NE	NE	NE	NE	NE	NE
Chang-Quan, 2010	Chronic disease (prospective studies)	NA	8	RR	1.53 (1.20 – 1.96)	$> 10^{-6}$ but $< 10^{-3}$	0.72 – 3.25	68	No/NE	III

Huang, 2010	Poor vision†	NE	NE	NE	NE	NE	NE	NE	NE	NE
Bao, 2017	Sleep disturbances	2610/3545	11	RR	1.92 (1.59–2.33)	$< 10^{-6}$	1.39 – 2.66	10	No/Yes	II

† Less than 3 prospective studies available. Not evaluated (NE).

* **Evidence classification: I** = convincing evidence criteria ($p < 10^{-6}$ per random-effects model; over than 1000 cases; no excess of significance bias; no small studies effects; small heterogeneity; prediction interval not including the null); **II** = highly-suggestive evidence criteria (not meeting type I criteria, but $p < 10^{-6}$ per random-effects model; over than 1000 cases; effect size of the largest study not crossing the null); **III** = suggestive evidence criteria (not meeting type I or II criteria, but $p < 10^{-3}$ per random-effects model; over than 1000 cases); **Weak** evidence criteria (all other associations with $p < 0.05$ per random-effects model); **NS** = non-significant associations; **NE** = not evaluated due to lack of individual study information.

Table S5. Sensitivity analysis limited to studies in which case ascertainment of depression was established by a structured diagnostic interview for association supported by convincing or highly suggestive evidence in the primary meta-analysis.

Reference	Risk factor	Total number of cases/ controls	Number of primary studies	Effect size metric	Random effects summary effect size (95% CI)	P random	95% PI	I^2	Small-study effects/ excess statistical significance	Level of evidence[†]
Major depressive disorder										
Dong, 2015	Tea consumption[*]	NE	NE	NE	NE	NE	NE	NE	NE	NE
Beydoun, 2012	Intimate partner violence against women	2316/18140	8	RR	2.81 (2.30 – 3.42)	$< 10^{-6}$	2.19 – 3.59	0	Yes/No	II
Atlantis, 2012	Sexual dysfunction	4803/5167	5	OR	2.98 (2.02 – 4.40)	$< 10^{-6}$	0.99 – 8.99	43	Yes/No	II
Pan, 2012	Metabolic syndrome (cross-sectional)	2202/7425	5	OR	1.12 (0.90 – 1.39)	0.325	0.61 – 2.03	36	Yes/No	NS
Blore, 2015	Gulf-war veterans (vs. non-deployed personnel)	3713/3337	4	OR	1.75 (1.47 – 2.07)	$< 10^{-6}$	1.20 – 2.54	0	No/No	I
Li, 2017	Dietary zinc[*]	NE	NE	NE	NE	NE	NE	NE	NE	NE
Jokela, 2014	Obesity[*]	NE	NE	NE	NE	NE	NE	NE	NE	NE
Jokela, 2014	3 metabolic risk factors[*]	NE	NE	NE	NE	NE	NE	NE	NE	NE
Jokela, 2014	4 or 5 metabolic risk factors[*]	NE	NE	NE	NE	NE	NE	NE	NE	NE
Onrust, 2006	Widowhood (vs. any other marital status)	2720/4868	5	RR	5.59 (3.79 – 8.23)	$< 10^{-6}$	2.09 – 14.97	30	No/No	I
Dowlatshahi, 2014	Psoriasis	86739/271030	5	OR	1.57 (1.40 – 1.76)	$< 10^{-6}$	1.04 – 2.37	84	No/No	II
Zhai, 2015	Sedentary behavior[*]	NE	NE	NE	NE	NE	NE	NE	NE	NE
Mandelli, 2015	Emotional abuse in childhood	1087/3128	4	OR	3.20 (1.44 – 7.10)	0.004	0.07 – 138.21	91	No/No	Weak
Mandelli, 2015	Sexual abuse in childhood	1295/2431	8	OR	2.64 (1.57 – 4.44)	$> 10^{-6}$ but $< 10^{-3}$	0.50 – 13.92	80	No/Yes	III
Mandelli, 2015	Physical abuse in childhood	721/1575	5	OR	1.74 (1.24 – 2.44)	0.001	0.74 – 4.11	29	No/No	Weak
Madsen, 2017	Job strain	1909/25552	7	OR	1.77 (1.46–2.13)	$< 10^{-6}$	1.02 – 14.01	24	No/No	I
Wan, 2016	Dry eye disease with Sjögren disease[*]	NE	NE	NE	NE	NE	NE	NE	NE	NE
Late-life depression										
Chang-Quan, 2010	Low educational level	NA	7	OR	1.36 (1.16 – 1.61)	$> 10^{-6}$ but $< 10^{-3}$	1.07 – 1.74	4	No/NE	Weak

Chang-Quan, 2010	Poor health	NA	3	OR	3.61 (2.69 – 4.86)	$< 10^{-6}$	0.53 – 24.54	0	No/NE	Weak
Chang-Quan, 2010	Chronic disease[a]	NE	NE	NE	NE	NE	NE	NE	NE	NE
Huang, 2010	Poor vision[a]	NE	NE	NE	NE	NE	NE	NE	NE	NE
Bao, 2017	Sleep disturbances	1179/1649	3	RR	1.88 (1.36–2.58)	< 0.001	0.24 – 14.88	0	No/Yes	III

[*] Less than 3 studies available. Not evaluated (NE).

[†] **Evidence classification: I** = convincing evidence criteria ($p < 10^{-6}$ per random-effects model; over than 1000 cases; no excess of significance bias; no small studies effects; small heterogeneity; prediction interval not including the null); **II** = highly-suggestive evidence criteria (not meeting type I criteria, but $p < 10^{-6}$ per random-effects model; over than 1000 cases; effect size of the largest study not crossing the null); **III** = suggestive evidence criteria (not meeting type I or II criteria, but $p < 10^{-3}$ per random-effects model; over than 1000 cases); **Weak** evidence criteria (all other associations with $p < 0.05$ per random-effects model); **NS** = non-significant associations; **NE** = not evaluated due to lack of individual study information.

Table S5. Sensitivity analysis using credibility ceilings for the meta-analyses investigating environmental risk factors for adult, late-life, or pediatric depression that were supported by convincing or highly-suggestive evidence.

Reference	Factor	0% Credibility ceiling ES (95% CI)	10% Credibility ceiling ES (95% CI)	20% Credibility ceiling ES (95% CI)	30% Credibility ceiling ES (95% CI)
Adult depression					
Dong, 2015	Tea consumption	0.68 (0.61 – 0.77)	0.79 (0.68 – 0.92)	0.84 (0.69 – 1.01)	0.88 (0.69 – 1.12)
Beydoun, 2012	Intimate partner violence against women	2.57 (2.25 – 2.94)	2.62 (1.57 – 4.37)	2.62 (1.20 – 5.71)	2.62 (0.75 – 9.15)
Onrust, 2006	Widowhood (vs. any other marital status)	5.59 (3.79 – 8.23)	4.76 (1.47 – 15.34)	4.47 (0.86 – 23.13)	4.32 (0.35 – 52.98)
Zhai, 2015	Sedentary behaviour	1.25 (1.16 – 1.35)	1.15 (1.08 – 1.23)	1.14 (1.04 – 1.24)	1.13 (0.99 – 1.29)
Dowlatshahi, 2014	Psoriasis	1.64 (1.41 – 1.90)	1.47 (1.12 – 1.92)	1.39 (0.94 – 2.06)	1.24 (0.71 – 2.19)
Atlantis, 2012	Sexual dysfunction	2.71 (1.93 – 3.79)	2.33 (1.32 – 4.12)	2.33 (0.98 – 5.54)	2.33 (0.58 – 9.34)
Pan, 2012	Metabolic syndrome	1.42 (1.28 – 1.57)	1.20 (1.08 – 1.33)	1.15 (1.01 – 1.31)	1.12 (0.93 – 1.34)
Jokela, 2014	Obesity	1.35 (1.21 – 1.50)	1.29 (1.09 – 1.52)	1.27 (1.02 – 1.59)	1.26 (0.91 – 1.76)
Jokela, 2014	4 or 5 metabolic risk factors	2.06 (1.59 – 2.68)	1.87 (1.27 – 2.76)	1.84 (1.05 – 3.21)	1.81 (0.77 – 4.27)
Blore, 2015	Gulf-war veterans (vs. non-deployed personnel)	2.37 (1.91 – 2.93)	2.06 (1.44 – 2.94)	2.06 (1.20 – 3.53)	2.06 (0.87 – 4.90)
Mandelli, 2015	Emotional abuse in childhood	2.78 (1.89 – 4.09)	1.54 (1.11 – 2.12)	1.38 (0.95 – 2.02)	1.36 (0.77 – 2.39)
Mandelli, 2015	Sexual abuse in childhood	2.42 (1.94 – 3.02)	1.48 (1.19 – 1.84)	1.46 (1.06 – 2.00)	1.46 (0.88 – 2.41)
Mandelli, 2015	Physical abuse in childhood	1.98 (1.68 – 2.33)	1.73 (1.28 – 2.32)	1.70 (1.10 – 2.64)	1.70 (0.84 – 3.44)
Madsen, 2017	Job strain	1.27 (1.04 – 1.55)	1.13 (0.95 – 1.36)	1.11 (0.91 – 1.34)	1.09 (0.88 – 1.35)
Wan, 2016	Dry eye disease with Sjögren syndrome	4.25 (2.67 – 6.76)	3.76 (1.64 – 8.65)	3.76 (1.06 – 13.36)	3.76 (0.49 – 28.75)
Li, 2017	Dietary zinc	0.65 (0.57 – 0.75)	0.71 (0.57 – 0.89)	0.76 (0.56 – 1.02)	0.82 (0.56 – 1.20)
Late-life depression					
Chang-Quan, 2010	Poor health	4.08 (3.25 – 5.12)	2.86 (1.70 – 4.81)	2.86 (1.30 – 6.31)	2.86 (0.80 – 10.19)
Chang-Quan, 2010	Chronic disease	2.59 (1.78 – 3.76)	1.45 (1.16 – 1.81)	1.45 (1.03 – 2.03)	1.45 (0.84 – 2.48)
Huang, 2010	Poor vision	1.94 (1.67 – 2.25)	1.65 (1.26 – 2.16)	1.53 (1.05 – 2.23)	1.35 (0.79 – 2.30)
Chang-Quan, 2010	Low educational level	1.58 (1.38 – 1.82)	1.24 (1.11 – 1.37)	1.16 (1.03 – 1.32)	1.10 (0.96 – 1.27)
Bao, 2017	Sleep disturbances	1.92 (1.59 – 2.33)	1.61 (1.21 – 2.13)	1.49 (1.07 – 2.07)	1.45 (0.90 – 2.34)
Pediatric depression					
Lu, 2012	Asthma	2.08 (1.56 – 2.77)	1.50 (1.08 – 2.08)	1.34 (0.92 – 1.97)	1.26 (0.78 – 2.03)

Abbreviations: CI = confidence interval; **ES** = effect size

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