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# Predictors of new-onset depressive disorders – Results from the longitudinal Finnish Health 2011 Study

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ABSTRACT

*Background:* Identifying risk factors for depression is important for understanding etiological mechanisms and targeting preventive efforts. No prior studies have compared risk factors of dysthymia and major depressive disorder (MDD) in a longitudinal setting.

*Methods:* Predictors of new-onset MDD and dysthymia were examined in a longitudinal general population study (Health 2000 and 2011 Surveys, BRIF8901). 4057 persons free of depressive disorders at baseline were followed up for 11 years. DSM-IV MDD and dysthymia were diagnosed with the Composite International Diagnostic Interview.

*Results:* 126 persons (4.4%, 95%CI 3.6–5.2) were diagnosed with MDD or dysthymia at follow-up. Predictors of new-onset depressive disorders were younger age (adjusted OR 0.97, 95%CI 0.95–0.99 per year), female gender (aOR 1.46, 95%CI 1.01–2.12), multiple childhood adversities (aOR 1.76, 95%CI 1.10–2.83), low trust dimension of social capital (aOR 0.58, 95%CI 0.36–0.96 for high trust), baseline anxiety disorder (aOR 2.75, 95%CI 1.36–5.56), and baseline depressive symptoms (aOR 1.65, 95%CI 1.04–2.61 for moderate and aOR 2.49, 95%CI 1.20–5.17 for severe symptoms). Risk factors for MDD were younger age, female gender, anxiety disorder and depressive symptoms, whereas younger age, multiple childhood adversities, low trust, and having 1–2 somatic diseases predicted dysthymia.

*Limitations:* We only had one follow-up point at eleven years, and did not collect information on the subjects' health during the follow-up period.

*Conclusions:* Persons with subclinical depressive symptoms, anxiety disorders, low trust, and multiple childhood adversities have a higher risk of depressive disorders. Predictors of MDD and dysthymia appear to differ. This information can be used to target preventive efforts and guide social policies.

## 1. Introduction

In light of the enormous burden of depressive disorders, there is increasing interest in interventions aimed at preventing onset of depression (Dennis and Dowswell, 2013; Muñoz et al., 2012; van der Waerden et al., 2011; van Zoonen et al., 2014). Knowledge on risk factors of depression would be useful to target interventions to populations at higher risk.

Correlates of depressive disorders in cross-sectional studies have been examined extensively, but less is known about predictors in longitudinal study settings. Yet, it is crucial to assess predictors in longitudinal settings to distinguish the complex patterns of causation (Kendler et al., 2002, 2006). Some perceived risk factors might in fact be a consequence of illness, or contribute to longer duration, thereby increasing prevalence but not incidence in that group (Lorant et al., 2003).

Some predictors have been identified consistently across several longitudinal studies, while findings on others are more contradictory. Female gender (Anthony and Petronis, 1991; De Graaf et al., 2002; Eaton et al., 2001, 2008; Klein et al., 2013; Wang et al., 2010a),

*Abbreviations:* MI, Multiple imputation; CIDI, Composite International Diagnostic Interview

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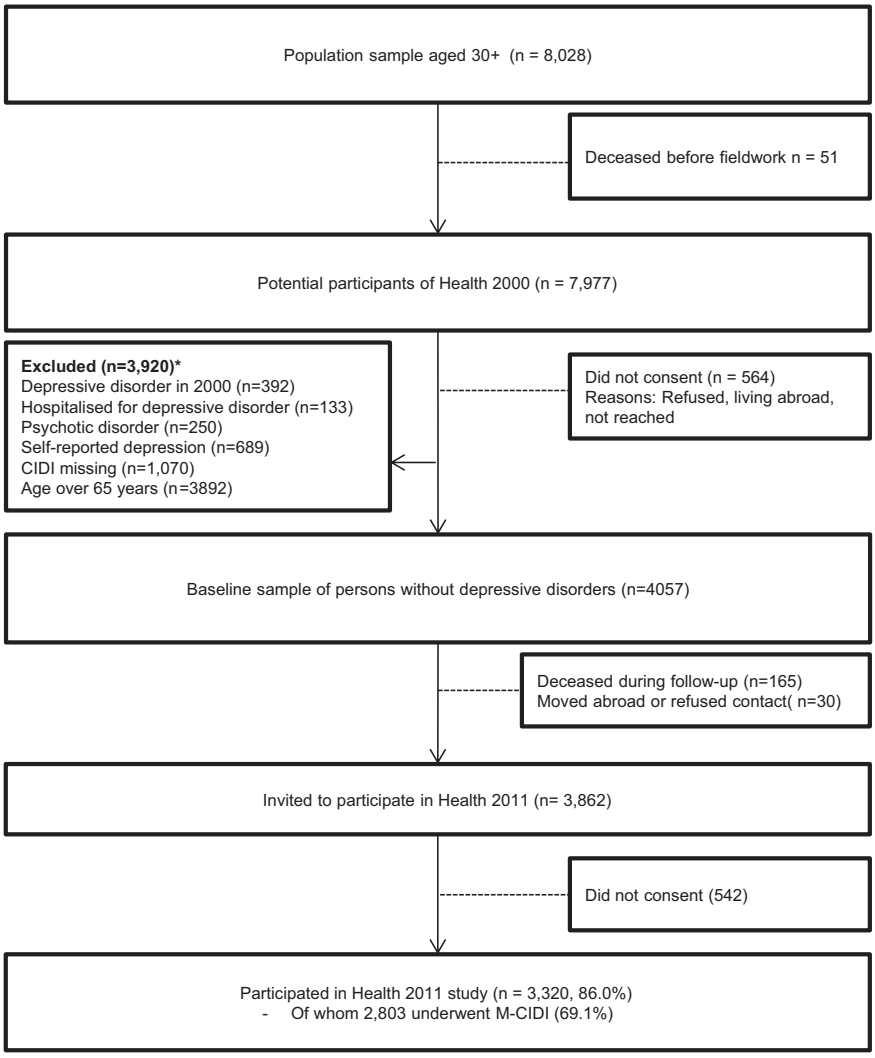


**Fig. 1. Participation in the baseline and follow-up study**. *Persons may belong to more than one exclusion category and therefore the numbers add up to more than the number of total exclusions.

younger age (Eaton et al., 2008; Stegenga et al., 2013; Wang et al., 2010a), being unmarried or unemployed (Anthony and Petronis, 1991; Stegenga et al., 2013) have appeared as risk factors for depression in many studies, even though not all of them.

The relationship between socioeconomic position (SEP) and depression is complex. Higher prevalence of depression is frequently found among socially disadvantaged groups (Kessler and Bromet, 2013; Pulkki-Råbäck et al., 2012), but causality of the association is not entirely clear. In a meta-analysis of longitudinal studies, the lowest SEP group had only slightly elevated risk of developing new episode of depression, while the odds for prevalence and persistence of disorder were higher (Lorant et al., 2003). Many longitudinal studies have not found any objective measures of lower SEP to predict onset of depression (De Graaf et al., 2002; Eaton et al., 2001; Kaplan et al., 1987; Skapinakis et al., 2006; Wang et al., 2010a; Weich and Lewis, 1998), but subjective measures, such as financial strain, have been significant predictors in various studies (Lorant et al., 2007; Skapinakis et al., 2006; Wang et al., 2010b; Weich and Lewis, 1998).

Many family and childhood factors may predispose a person to depression. Family history of depression appears a strong predictor (Eaton et al., 2001; Klein et al., 2013; Stegenga et al., 2013; Wang et al., 2010a), but also different childhood social adversities and maltreatment increase risk of depression in adulthood (Park et al., 2013; Ritsher et al., 2001; Stegenga et al., 2013).

Social capital, or the collective value of the social networks that an individual has, is strongly associated with current depressive symptoms (Almedom, 2005; Nieminen et al., 2010; Nyqvist et al., 2013), but less is known about it as a risk factor for new-onset depression. Two studies have found no association (Fujiwara and Kawachi, 2008; Noteboom et al., 2015), but others have found components of social capital, such as social isolation (Kaplan et al., 1987), lack of social support (Stegenga et al., 2013), and decreased social participation (Kivelä et al., 1996) to predict onset of depression.

Most of the above-mentioned determinants are from studies examining the risk of onset of major depressive disorder (MDD). Risk factors for dysthymia are much less known than those for MDD. They include family history of mood disorders, particularly dysthymia, and childhood adversity (Klein and Santiago, 2003). However, few studies have been carried out in a longitudinal setting.

This study examines predictors of new-onset depressive disorders, MDD and dysthymia, in an 11-year follow-up of a general population sample. We evaluate the impact of sociodemographic characteristics, childhood adversity, somatic and mental health, and social capital measured at baseline on the risk of developing a depressive disorder.

## 2. Methods

### 2.1. Study sample and participants

The Health 2000 Survey (http://www.terveys2000.fi/), conducted

in years 2000–2001 was based on a nationally representative sample of 8028 adults aged 30 years and over (Heistaro, 2008). The study used a two-stage clustered sampling of 15 largest Finnish towns and 65 health districts. Persons over 80 years were oversampled (2:1). The study consisted of a home interview, self-administered questionnaires and a comprehensive health examination. In total, 7112 persons (89%) participated in the home interview and/or the health examination, and 6005 persons (75%) participated in the Composite International Diagnostic Interview (CIDI) that was conducted as a part of the health examination.

Of persons older than 65, only 59% participated in the CIDI interview at baseline, while the overall participation rate was 75%. During the follow-up, 59% of persons over 65 years died. These issues may limit the generalizability of the results in this age group. Therefore, the analyses in this study are limited to participants aged 30–65 years at baseline.

To only analyse risk factors for new-onset cases of depression, we excluded three groups from the analyses: those with a 12-month depressive disorder (MDD or dysthymia) at the baseline CIDI interview; those who reported to have been diagnosed by a doctor as suffering from a depressive disorder during their lifetime, and those with a hospitalisation due to depressive disorders at any point in their life prior to the baseline. In addition, those with psychotic disorders at baseline were excluded from the analyses, as their risk factors were considered to differ from the general population. We also excluded those whose who did not participate in the baseline CIDI interview, because we could not exclude baseline depression. Consequently, the study population consisted of 4057 persons (Fig. 1).

The Health 2011 Survey (www.terveys2011.info) was a follow-up study of the Health 2000 Survey (Koskinen, 2012). All members of the Health 2000 Survey sample alive and living in Finland who had not refused to participate were invited to take part. Of the study population of 4057persons, 195 were lost during follow-up (165 persons died and 30 moved abroad or had refused contact). Therefore, 3862 persons were invited to participate in the Health 2011 Survey. Out of them, 3320 (86.0%) participated in at least one part of the study and 2803 (69.1%) participated in the CIDI interview. Participation in the whole Health 2011 has been reported elsewhere (Markkula et al., 2015b).

The Health 2000 and Health 2011 studies had approval of the Ethics Committee of the Hospital District of Helsinki and Uusimaa. Participants provided written informed consent.

### 2.2. Psychiatric assessment

The Munich version of the Composite International Diagnostic Interview (*m*-CIDI) was used to diagnose psychiatric disorders at baseline and follow-up. (Andrews and Peters, 1998; Wittchen et al., 1998) The process has been described in more detail elsewhere (Heistaro, 2008; Koskinen, 2012; Markkula et al., 2015a; Pirkola et al., 2005b). The presence of eight diagnoses (panic disorder, agoraphobia, social phobia, generalized anxiety disorder, dysthymia, major depressive disorder, alcohol abuse and alcohol dependence) during the past 12 months was assessed using diagnostic criteria of the DSM-IV. In addition, participants were inquired whether they had been diagnosed with depression by a doctor at any point in their life, and those who reported lifetime diagnosis at baseline were excluded, as described above.

Depressive symptoms at baseline were assessed with the 21-item Beck Depression Inventory (BDI) (Beck et al., 1961).

Psychotic disorders at baseline were screened and further examined with the Research Version of the Structured Clinical Interview for DSM-IV (SCID-1) (First, 1997), and together with a review of medical records, lifetime diagnosis of psychotic disorder was established (Perälä et al., 2007).

### 2.3. Sociodemographic variables

Sociodemographic information was obtained in the baseline interview. Marital status was categorized into married or cohabiting and unmarried (consisting of separated, never-married or widowed). Educational level was divided into three categories based on the level of education completed: basic (no high school or vocational training), secondary (high school or completed vocational school) and higher (degree from a higher vocational institution, polytechnic or university). Family income was obtained from registers of the Finnish Tax Administration, adjusted for family size and divided into quintiles.

### 2.4. Childhood adversities

Childhood adversities were inquired using an 11-item questionnaire (Pirkola et al., 2005a), which probed parental mental disorders, long-term financial difficulties, parental unemployment, parental serious diseases or disabilities, parental alcohol use, serious conflicts in the family and divorce, own serious illness during childhood and being a victim of bullying. Cronbach's alpha of the questionnaire was 0.6, indicating relatively low degree of correlation between the questions (Kananen et al., 2010). Parental (maternal or paternal) mental disorder was included as a separate risk factor to take into account the possibly genetic nature of the risk, and the remaining nine items were categorized into 0, 1–2 and 3 or more reported adversities.

### 2.5. Social capital

Social capital was assessed using a measure that included 39 variables divided into three dimensions: social support, social participation and networks, and trust and reciprocity (Nieminen et al., 2008). These three dimensions correspond to those proposed by a large international consensus meeting (Zukewich and Norris, 2005). The trust dimension included a shortened version of the Cook-Medley hostility scale consisting of eight items, in addition to questions about feeling safe in the neighbourhood and disappointments in close relationships (Nieminen et al., 2008).

### 2.6. Somatic health status

Somatic health status was assessed by the presence of 24 somatic conditions that were chosen for their chronic nature and reliability of self-report. Presence of each diagnosis was based on self-report, inquiring whether the person had been diagnosed by a physician for the condition (Saarni et al., 2006). Diseases were categorised into eight groups: pulmonary, cardiovascular, neurological, musculoskeletal, vision and hearing, cancer, diabetes and other chronic diseases, and then further into three categories of 0, 1–2 or 3 diseases.

### 2.7. Register data

Register data was used for two purposes: exclusion of persons with lifetime hospitalisations for depressive disorders prior to the baseline, and in multiple imputation (MI) to account for missing data at follow-up. Data was obtained from the Care Register for Health Care, which covers all public and private hospitals in Finland. For MI, binary variables on hospitalisation for depressive disorders and for any psychiatric disorder were constructed. The list of variables used and corresponding ICD-codes is presented in Supplementary Table 1. In the case of hospitalisation for any psychiatric disorder, the variable included lifetime hospitalisations (since 1969) until 31.12.2011. In the case of hospitalisations for depressive disorders, persons with hospitalisation by 31.12.2000 were excluded, and only hospitalisations between 2001 and 2011 were included.

### 2.8. Statistical analysis

Multiple imputation (MI) is an effective way to account for missing data (Härkänen et al., 2016; Li et al., 2015; Mackinnon, 2010; Rubin, 1987). As opposed to single imputations it, in particular, allows for the statistical uncertainty in the imputations. We used MI based on the chained equations approach (MICE) (van Buuren and Groothuis-Oudshoorn, 2011) and classification and regression trees (Therneau et al., 2015) to handle missing data. This approach is suitable for imputing continuous, categorical or binary variables. MICE is based on the assumption that given the variables used in the imputation procedure, the missing data are Missing At Random (MAR), which means that the probability that a value is missing depends only on observed values and not on unobserved values (Azur et al., 2011). In the current study, it has been shown to be the best method to take missing data into account (Härkänen et al., 2016). A list of the variables used in imputation is provided as Supplementary material. Altogether 35 imputed data sets were constructed for incomplete data by Gibbs sampling. In addition, poststratification weights were used to handle the unit nonresponse of the Health 2000 Survey. All analyses accounted for the two-stage cluster sampling as well as the MI.

Possible predictors were analysed in logistic regression models, where the outcomes were 1) MDD, 2) dysthymia or 3) any depressive disorder at follow-up. In bivariate analyses (data not shown), different distributions of age and sex appeared to modify the impact substantially. Unfortunately, the number of observations limited the possibility to carry out analyses separately by gender or other subgroups. Therefore, age and sex were adjusted for in all the models, and six different models were built by subsequently adding new risk factors and confounders to those of the previous model: 1) age and sex; 2) education, income and marital status; 3) childhood adversities; 4) social capital; 5) somatic health; 6) mental health (anxiety and alcohol use disorders, baseline depressive symptoms). For the separate analyses for MDD and dysthymia, only the results of models 1 and 6 are presented. However, there were no new-onset cases of dysthymia among persons with baseline alcohol use disorders, and therefore this predictor was left out of the model.

The R statistical software (version 3.2 for Windows) (Team, 2014; Therneau et al., 2015; van Buuren and Groothuis-Oudshoorn, 2011) was used for the MI. The imputed data sets were analysed using the Stata statistical software package (version 14.1 for Windows).

## 3. Results

The baseline characteristics of the sample are presented in Table 1.

At the follow-up in 2011, 104 persons received a 12-month diagnosis of MDD and 31 of dysthymia, out of whom 9 had both diagnoses, yielding a 12-month prevalence of 4.4% (95% CI 3.6–5.2). The 12-month prevalence was higher among women, in the age group 30–44 years, among persons with childhood adversities, persons with low trust, and among persons with an anxiety disorder or subclinical depressive symptoms (Table 1).

Table 2 presents magnitude of the risk of depressive disorders associated with different predictors expressed in odds ratios (OR) in 6 different models. Adjustment had little effect on the predictors, save for the participation dimension of social capital, which was a protective factor until barely lost significance when adjusting for other aspects of social capital.

In the final adjusted model, predictors of new-onset depressive disorders were younger age (OR 0.97, 95% CI 0.95–0.99 for each year of age), female gender (OR 1.46, 95% CI 1.01–2.12), multiple childhood adversities (OR 1.76, 95% CI 1.10–2.83), low trust dimension of social capital (OR 0.58, 95%CI 0.36–0.96 for high trust), baseline anxiety disorder (OR 2.75, 95%CI 1.36–5.56), and baseline depressive symptoms (OR 1.65, 95% CI 1.04–2.61 for moderate and OR 2.49, 95% CI 1.20–5.17 for severe depressive symptoms).

**Table 1**
Characteristics of the sample at baseline and 12-month prevalence of depression by subgroup.[a]

| | Observations (n) and prevalence (%) at baseline (2000) | | 12-month prevalence of depressive disorders at 11 years' follow-up (2011) | P for difference[*] |
|---|---|---|---|---|
| | N | % | % | |
| Complete sample | 4057 | | 4.4 | |
| *Sociodemographic characteristics* | | | | |
| Age group: 30–44 | 1789 | 45.9 | 5.4 | 0.033 |
| Age group: 45–54 | 1328 | 31.9 | 4.2 | |
| Age group: 55–65 | 940 | 22.2 | 2.9 | |
| Sex: male | 2001 | 48.4 | 3.5 | 0.018 |
| Sex: female | 2056 | 51.6 | 5.3 | |
| Education: primary | 1127 | 28.0 | 3.6 | 0.420 |
| Education: secondary | 1482 | 36.8 | 4.7 | |
| Education: tertiary | 1384 | 35.2 | 4.7 | |
| Income: 1st quintile | 356 | 8.0 | 4.3 | 0.974 |
| Income: 2nd quintile | 673 | 16.6 | 4.8 | |
| Income: 3rd quintile | 875 | 21.1 | 4.0 | |
| Income: 4th quintile | 1052 | 26.6 | 4.4 | |
| Income: 5th quintile | 1101 | 27.8 | 4.5 | |
| Marital status: married | 3163 | 79.4 | 4.4 | 0.838 |
| Marital status: unmarried | 881 | 20.6 | 4.5 | |
| *Childhood adversities* | | | | |
| No adversities | 1827 | 44.4 | 3.5 | 0.001 |
| 1–2 | 1644 | 41.0 | 4.3 | |
| 3 or more | 566 | 14.6 | 7.6 | |
| No parental mental disorder | 3785 | 94.8 | 4.3 | 0.068 |
| Parental mental disorder | 204 | 5.2 | 7.3 | |
| *Social capital* | | | | |
| Dimension 1: support low | 1183 | 27.3 | 4.1 | 0.825 |
| Dimension 1: support medium | 1307 | 33.8 | 4.4 | |
| Dimension 1: support high | 1499 | 39.0 | 4.6 | |
| Dimension 2: participation low | 1131 | 26.9 | 5.2 | 0.329 |
| Dimension 2: participation medium | 1367 | 35.4 | 3.7 | |
| Dimension 2: participation high | 1409 | 37.8 | 4.5 | |
| Dimension 3: trust low | 1066 | 28.1 | 6.0 | 0.006 |
| Dimension 3: trust medium | 1314 | 36.3 | 4.6 | |
| Dimension 3: trust high | 1336 | 35.7 | 2.9 | |
| *Somatic diseases* | | | | |
| No somatic diseases | 1504 | 37.9 | 3.8 | 0.369 |

(continued on next page)

**Table 1** *(continued)*

| | Observations (n) and prevalence (%) at baseline (2000) | | 12-month prevalence of depressive disorders at 11 years' follow-up (2011) | P for difference* |
|---|---|---|---|---|
| | N | % | % | |
| 1–2 somatic diseases | 2042 | 50.0 | 4.8 | |
| 3 or more somatic diseases | 498 | 12.1 | 4.4 | |
| *Psychiatric morbidity* | | | | |
| Subclinical depressive symptoms | | | | |
| BDI 0–9 | 3332 | 83.2 | 3.8 | 0.000 |
| BDI 10–18 | 586 | 14.1 | 6.7 | |
| BDI 19 or more | 107 | 2.8 | 11.5 | |
| No anxiety disorder | 3895 | 97.8 | 4.2 | 0.000 |
| Anxiety disorder | 162 | 2.2 | 13.7 | |
| No alcohol use disorder | 3895 | 96.3 | 4.3 | 0.375 |
| Alcohol use disorder | 162 | 3.7 | 6.0 | |

* p-value obtained by logistic regression.
[a] Multiple imputation was used to correct for non-participation in the follow-up.

The predictors were different for MDD and dysthymia. Risk factors for MDD (Table 3) were younger age (OR 0.97, 95% CI 0.95–0.99 for each additional year of age), female gender (OR 1.68, 95% CI 1.11–2.55), baseline anxiety disorder (OR 2.53, 95% CI 1.17–5.46) and depressive symptoms (OR 1.64, 95% CI 1.00–2.68 for moderate and OR 3.03, 95% CI 1.48–6.19 for severe depressive symptoms), whereas risk factors for dysthymia (Table 4) were younger age (OR 0.93, 95% CI 0.89–0.98), multiple childhood adversities (OR 3.36, 95% CI 1.22–9.27), low trust dimension of social capital (OR 0.30, 95% CI 0.09–0.97 for high trust) and having 1–2 somatic diseases (OR 3.37, 95% CI 1.19–9.52).

## 4. Discussion

We analysed risk factors for first-onset depressive disorders in a representative general population sample with an eleven-year follow-up. This is one of very few longitudinal population studies that use reliable psychiatric diagnostics and a long follow-up with information on a wide variety of predictors, including somatic comorbidities. Of all the predictors we included, younger age, multiple childhood adversities, low trust, baseline anxiety disorder and depressive symptoms were significantly associated with new-onset depressive disorders. The 12-month prevalence of depressive disorders in people with no history of depressive disorder at baseline was 4.4%, whereas it was 21% in people with MDD at baseline and 27% in people with dysthymia at baseline (Markkula et al., 2016), and 9.6% in all participants of the Health 2011 survey (Markkula et al., 2015b). Therefore, the overall risk of developing depressive disorders was lower in persons with no previous history of depressive disorders.

To our knowledge, this is the first study that compares predictors of MDD and dysthymia in a longitudinal setting. Some longitudinal studies have investigated the risk factors of depressive disorders combined, and knowledge on risk factors of dysthymia specifically has been derived from other study settings. It has previously been thought that most risk factors are common to episodic and chronic forms of depressive disorders, and an accumulation of risk factors contributes to the development of more chronic forms (Klein and Santiago, 2003). However, based on this study, the risk factors of MDD and dysthymia appear to be distinct from each other, with prior anxiety disorder and baseline depressive symptoms increasing the risk of MDD, while low trust, childhood adversities and somatic diseases increase the risk of dysthymia.

When making comparisons to previous literature on risk factors of MDD, it should be noted that our study participants were 30–65 years old at baseline, and 41–76 years at the time of follow-up. Therefore, some factors that influence risk of depression in young adulthood might have been less important in our study. Similarly, the varying follow-up times in studies influence which factors are found significant predictors, with e.g. life events being more important predictors in studies with relatively short follow-up times (De Graaf et al., 2002), whereas in our study the follow-up was eleven years, and we did not have information on recent life events. Also, the method of measuring depression is important: in the Canadian NPHS study, low education appeared as a predictor (Wang et al., 2010b), but in the CIDI-SF instrument used in the study, low education has been associated with false positives (Patten et al., 2000).

### 4.1. Gender and age

Women had a 1.5-fold risk of depressive disorders; the risk was increased for MDD but not dysthymia. In earlier literature, female gender is the most consistent predictor of incidence of depressive disorders, and is associated with 1.5–2-fold risk of MDD (Anthony and Petronis, 1991; De Graaf et al., 2002; Eaton et al., 2001, 2008; Klein et al., 2013; Wang et al., 2010a). Previous studies have found that the association with gender weakens with age (Patten et al., 2016), and specifically, that the gender difference in incidence of depression reduces significantly after 40 years of age (Pedersen et al., 2014). Therefore, it is understandable to find a rather small risk difference between genders in our middle-aged study population.

Younger age is frequently associated with higher risk of depressive disorders (Eaton et al., 2008; Stegenga et al., 2013; Wang et al., 2010a), similar to our findings. It is interesting that the association was observed also after 40 years of age, even though non-participation in older age groups is accounted for. Smaller incidence and prevalence in the older age groups may partly be caused by methodological issues: the complex questions of the CIDI depression screen may lead to underestimation of depression in the elderly (O'Connor and Parslow, 2009). However, it is also possible that the impact of both personal characteristics and environmental stressors differs according to life stages.

### 4.2. Family history of mental disorder and childhood adversities

Parental mental disorder was not a significant predictor in our study. However, we did not have information on history depressive disorders specifically, which has been a strong predictor in previous studies (Eaton et al., 2001; Klein et al., 2013; Stegenga et al., 2013; Wang et al., 2010a). Remarkably, controlling for other childhood adversities appeared to reduce the risk associated with parental mental disorder (OR 1.7–OR 1.3), but neither was significant. However, assessment of parents' mental health was based on two questions, retrospective report and the respondents' own assessment at age 30 or older, and therefore is not as reliable as progressive studies that have systematically assessed the parents' mental health.

Accumulation of three or more childhood adversities, however, was a risk factor for depressive disorders, particularly dysthymia. This is consistent with earlier literature (Bowes et al., 2015; Elovainio et al., 2015; Park et al., 2013; Pirkola et al., 2005a; Ritsher et al., 2001; Sourander et al., 2015; Stegenga et al., 2013). Specifically, childhood adversity has been associated with dysthymia (Klein and Santiago, 2003; Wu et al., 2013), although Wu et al. (2013) hypothesise that childhood events make a person vulnerable to chronic depression, and

**Table 2**
Predictors of new-onset depressive disorders in six logistic regression models (n=126).[a]

| | Model 1: adjusted for age and sex OR (95% CI) | Model 2: adjusted for age, sex, education, income and marital status OR (95% CI) | Model 3: adjusted for age, sex, education, income and marital status, childhood adversities OR (95% CI) | Model 4: adjusted for age, sex, education, income and marital status, childhood adversities and social capital OR (95% CI) | Model 5: adjusted for age, sex, education, income and marital status, childhood adversities, social capital and somatic health OR (95% CI) | Model 6: adjusted for age, sex, education, income and marital status, childhood adversities, social capital, somatic and mental health OR (95% CI) |
|---|---|---|---|---|---|---|
| ***Sociodemographic*** | | | | | | |
| **Age (per each year)** | **0.97 (0.96–0.99)**[b] | **0.97 (0.96–0.99)** | **0.97 (0.95–0.99)** | **0.97 (0.96–0.99)** | **0.97 (0.95–0.99)** | **0.97 (0.95–0.99)** |
| **Sex (female)** | **1.55 (1.09–2.21)**[c] | **1.56 (1.10–2.22)** | **1.64 (1.11–2.42)** | **1.50 (1.05–2.13)** | **1.48 (1.04–2.11)** | **1.46 (1.01–2.12)** |
| **Basic education** | 1 | 1 | 1 | 1 | 1 | 1 |
| **Intermediate education** | 1.10 (0.66–1.84) | 1.11 (0.66–1.85) | 1.21 (0.72–2.04) | 1.20 (0.72–2.00) | 1.22 (0.73–2.04) | 1.27 (0.75–2.15) |
| **High education** | 1.01 (0.61–1.67) | 1.01 (0.60–1.68) | 1.14 (0.67–1.95) | 1.18 (0.70–1.98) | 1.22 (0.72–2.06) | 1.23 (0.73–2.09) |
| **Income** | | | | | | |
| **1st quintile** | 1 | 1 | 1 | 1 | 1 | 1 |
| **2nd quintile** | 1.11 (0.50–2.48) | 1.11 (0.50–2.46) | 1.27 (0.57–2.81) | 1.23 (0.55–2.74) | 1.23 (0.55–2.74) | 1.23 (0.54–2.80) |
| **3rd quintile** | 0.92 (0.41–2.07) | 0.92 (0.41–2.09) | 0.93 (0.39–2.21) | 0.99 (0.43–2.28) | 0.98 (0.43–2.27) | 0.99 (0.42–2.34) |
| **4th quintile** | 0.9 (0.46–2.09) | 0.98 (0.44–2.20) | 1.04 (0.45–2.43) | 1.07 (0.47–2.42) | 1.06 (0.47–2.42) | 1.06 (0.46–2.47) |
| **5th quintile** | 1.01 (0.48–2.12) | 1.03 (0.47–2.26) | 1.08 (0.46–2.55) | 1.15 (0.51–2.56) | 1.14 (0.51–2.55) | 1.17 (0.51–2.67) |
| **Married or cohabited** | 1 | 1 | 1 | 1 | 1 | 1 |
| **Single** | 1.05 (0.69–1.60) | 1.02 (0.62–1.67) | 1.01 (0.58–1.77( | 0.99 (0.59–1.66) | 0.99 (0.59–1.67) | 0.94 (0.56–1.61) |
| ***Childhood adversity*** | | | | | | |
| **No adversities** | 1 | 1 | 1 | 1 | 1 | 1 |
| **1–2 adversities** | 1.27 (0.86–1.88) | 1.27 (0.86–1.88) | 1.35 (0.88–2.07) | 1.20 (0.81–1.77) | 1.19 (0.81–1.75) | 1.15 (0.78–1.70) |
| **3 or more adversities** | **2.17 (1.41–3.36)** | **2.20 (1.42–3.42)** | **2.49 (1.51–4.11)** | **1.92 (1.21–3.07)** | **1.89 (1.18–3.03)** | **1.76 (1.10–2.83)** |
| **Parental mental disorder** | 1.71 (0.92–3.16) | 1.71 (0.92–3.17) | 1.34 (0.70–2.57) | 1.33 (0.68–2.60) | 1.30 (0.66–2.54) | 1.29 (0.66–2.53) |
| ***Social capital*** | | | | | | |
| **Low social support** | 1 | 1 | 1 | 1 | 1 | 1 |
| **Intermediate social support** | 0.93 (0.59–1.47) | 0.93 (0.59–1.47) | 0.95 (0.60–1.52) | 1.04 (0.65–1.65) | 1.05 (0.65–1.67) | 1.06 (0.66–1.71) |
| **High social support** | 0.89 (0.57–1.40) | 0.90 (0.57–1.42) | 0.93 (0.59–1.47) | 1.06 (0.66–1.71) | 1.05 (0.65–1.69) | 1.07 (0.66–1.73) |
| **Low participation** | 1 | 1 | 1 | 1 | 1 | 1 |
| **Intermediate participation** | 0.62 (0.39–0.98) | **0.62 (0.39–0.97)** | **0.62 (0.39–0.99)** | 0.64 (0.40–1.02) | 0.64 (0.40–1.02) | 0.66 (0.41–1.07) |
| **High participation** | 0.70 (0.44–1.10) | 0.69 (0.43–1.09) | 0.68 (0.43–1.08) | 0.69 (0.43–1.11) | 0.69 (0.43–1.10) | 0.74 (0.45–1.19) |
| **Low trust** | 1 | 1 | 1 | 1 | 1 | 1 |
| **Intermediate trust** | 0.75 (0.49–1.13) | 0.74 (0.49–1.12) | 0.76 (0.50–1.15) | 0.77 (0.50–1.16) | 0.77 (0.51–1.18) | 0.85 (0.55–1.30) |
| **High trust** | **0.46 (0.29–0.74)** | **0.46 (0.29–0.74)** | **0.49 (0.30–0.79)** | **0.50 (0.31–0.80)** | **0.51 (0.32–0.82)** | **0.58 (0.36–0.96)** |
| ***Somatic health*** | | | | | | |
| **No somatic diseases** | 1 | 1 | 1 | 1 | 1 | 1 |
| **1–2 somatic diseases** | **1.49 (1.01–2.20)** | **1.49 (1.00–2.20)** | 0.43 (0.97–2.12) | 1.41 (0.95–2.09) | 1.41 (0.95–2.09) | 1.32 (0.88–1.97) |
| **3 or more somatic diseases** | 1.61 (0.89–2.90) | 1.62 (0.90–2.90) | 1.53 (0.84–2.76) | 1.45 (0.80–2.62) | 1.45 (0.80–2.62) | 1.26 (0.60–2.90) |
| ***Mental health*** | | | | | | |
| **Anxiety disorder** | **3.45 (1.74–6.83)** | **3.48 (1.76–6.90)** | **3.40 (1.71–6.75)** | **3.20 (1.61–6.39)** | **3.04 (1.52–6.06)** | **2.75 (1.36–5.56)** |
| **Alcohol use disorder** | 1.66 (0.77–3.59) | 1.64 (0.76–3.53) | 1.59 (0.74–3.42) | 1.47 (0.68–3.17) | 1.46 (0.67–3.15) | 1.32 (0.60–2.90) |
| **No depressive symptoms**[d] | 1 | 1 | 1 | 1 | 1 | 1 |
| **Some depressive symptoms** | **2.08 (1.37–3.16)** | **2.09 (1.36–3.21)** | **1.97 (1.28–3.03)** | **1.73 (1.11–2.72)** | **1.69 (1.07–2.66)** | **1.65 (1.04–2.61)** |
| **Many depressive symptoms** | **3.52 (1.77–7.00)** | **3.68 (1.80–7.36)** | **3.24 (1.60–6.57)** | **2.79 (1.35–5.74)** | **2.72 (1.31–5.63)** | **2.49 (1.20–5.17)** |

[a] Multiple imputation was used to correct for non-participation in the follow-up.
[b] Only adjusted for sex.
[c] Only adjusted for age.

[d] No depressive symptoms: BDI score 0–9; some depressive symptoms: BDI score 10–18; many depressive symptoms: BDI score 19 or more.

not specifically to dysthymia.

Even though reporting bias, where depressed individuals are more likely to remember and report childhood adversity exists (Colman et al., 2015), this was not the case in our study, as respondents were not depressed at baseline when reporting on their childhood conditions. The impact of multiple childhood adversities on the risk of persistent depressive disorder may be mediated through personality pathology (Klein et al., 2015; Spinhoven et al., 2015). This could explain how the risk remains increased in midlife and even later. Also, early life adversity is associated with increased methylation of the glucocorticoid receptor gene methylation, resulting in dysregulation of the hypothalamus-pituitary-adrenal axis (Turecki and Meaney, 2016).

It should be noted that we did not have information on any type of abuse, which has previously shown a strong link to depression (Norman et al., 2012). Our results highlight the fact that the accumulation of less severe childhood adversities also increases the risk of adult depression.

### 4.3. Socioeconomic position

In this study, neither education nor income predicted risk of depressive disorders. This is in line with many previous longitudinal studies (De Graaf et al., 2002; Eaton et al., 2001; Kaplan et al., 1987; Skapinakis et al., 2006; Wang et al., 2010a; Weich and Lewis, 1998), although a meta-analysis found a slightly elevated risk of the lowest socioeconomic group compared to the highest (OR 1.2) (Lorant et al., 2003). Based on our results, it appears that lower socioeconomic position, whether measured by education or income, does not have a significant impact on the risk of midlife depressive disorders in the Finnish context. The impact may, however, be mediated through consequences of social disadvantage, such as childhood adversities. In fact, some of the included adversities were directly related to socioeconomic position, such as financial difficulties and unemployment of parent.

More subjective measures, such as financial strain, have been significant predictors in various studies (Lorant et al., 2007; Skapinakis et al., 2006; Wang et al., 2010b; Weich and Lewis, 1998), although not all of them (Butterworth et al., 2009; Dijkstra-Kersten et al., 2015). Also unemployment is an established risk factor for depression (Anthony and Petronis, 1991; Stegenga et al., 2013).

### 4.4. Marital status and social capital

The prevalence of depression is higher among persons who are not in a relationship (Kessler and Bromet, 2013), and in some studies, the lack of a partner has been a risk factor for depression (Scott et al., 2010; Stegenga et al., 2013). However, we did not find an increased risk for either MDD or dysthymia among unmarried persons. This could be related to our long follow-up period, during which the baseline marital status may have changed, and the psychological consequences of separation, divorce or loss of partner attenuated. However, the causality could also be reverse. In fact, persons with mental disorders have a reduced chance of forming a relationship, and a higher risk of ending one (Breslau et al., 2011). A further mechanism contributing to higher prevalence is the prolonged course of illness among unmarried persons (Eaton et al., 2008; Markkula et al., 2016).

High level of trust was protective against depressive disorders, particularly dysthymia. The questions measuring trust inquired about feeling safe in the neighbourhood, and disappointments with persons close to you. The construct of trust as a component of social capital is closely related to personality features that may be protective against depression. The questions may also be related to actually living in a threatening, stressful environment, or actual disappointments experienced in close relationships, that liken to an interpersonal trauma.

Social participation at the intermediate level was a borderline significant protective factor (OR 0.66, p=0.09) against depressive disorders.

There is a strong association with social capital and current depressive symptoms (Nieminen et al., 2010), and it is interesting that social capital could be a protective factor also in a longitudinal setting. Social participation is a proxy of functional capacity as well, whereas trust describes the personality in addition to adverse experiences. Social support, on the other hand, seemed to have no impact on the risk of depressive disorders, which highlights the importance of the individuals' actions and attitude, rather than strength of the social network. These interesting findings merit further study.

### 4.5. Chronic diseases

Chronic somatic diseases were predictive of dysthymia, although the association was significant only in the category 1–2 diseases, with a similar but nonsignificant association for 3 or more diseases. This could be related to multiple diseases being more frequent among older persons, who on the other hand had a lower risk of dysthymia. MDD was not predicted by somatic comorbidity. Previous studies have also found a higher risk of depression among persons with somatic conditions (Patten, 2001; Wang et al., 2010a). It is noteworthy that the follow-up time was long, and the effect of recently diagnosed somatic diseases and the psychological distress related to it is likely to have already been overcome, and the risk was likely to be mediated by other paths.

The risk could be related to a common underlying factor, such as inflammation (Kiecolt-Glaser et al., 2015); the experience and consequences of a chronic illness, such as losing work ability or functional capacity; or be a direct cause of the somatic condition. Finally, it has been shown that detecting depression in persons with chronic physical conditions is particularly challenging (Menear et al., 2015), which may lead to unidentified depressive disorders becoming chronic in this population.

### 4.6. Other psychiatric disorders

The 12-month prevalence of depressive disorders was significantly higher among persons with baseline anxiety disorders; 13% of them had a depressive disorder at follow-up. Other studies have also found anxiety disorders to predict onset of depressive disorders (Eaton et al., 2008; Kessler et al., 2008; Klein et al., 2013; Stegenga et al., 2013). This is understandable, as the two groups of disorders have a shared genetic aetiology (Waszczuk et al., 2014) as well as common environmental risk factors, such as negative life experiences and personality features (Moreno-Peral et al., 2014; Moscati et al., 2015). The risk could also be related to the direct impact of the anxiety disorder and its consequences, such as social isolation. On the contrary, alcohol use disorders did not increase the risk of depressive disorders in this study, whereas other studies and a meta-analysis have found the risk to be increased (Boden and Fergusson, 2011). This could be related to controlling for multiple other factors, including baseline depressive symptoms.

Subclinical depressive symptoms were a strong predictor of later onset of MDD but not dysthymia, similar to what has been found in other studies (Ernst et al., 1992; Klein et al., 2013; Skapinakis et al., 2006). As we did not have information on the participants' health beyond the baseline and follow-up interviews, we cannot assess whether the baseline depressive symptoms represented a pre-morbid state of a depressive episode about to begin, a transient phase, or a more permanent way of expressing emotions.

**Table 3**
Predictors of new-onset major depressive disorder (MDD) in an eleven-year follow-up of a general population (n=104).[a]

| | Model 1: adjusted for age and sex OR (95% CI) | Model 6: adjusted for age, sex, education, income and marital status, childhood adversities, social capital, somatic and mental health OR (95% CI) |
|---|---|---|
| ***Sociodemographic factors*** | | |
| **Age** (per year)[b] | **0.97 (0.95–1.00)** | **0.97 (0.95–0.99)** |
| **Sex (female)**[c] | **2.00 (1.30–3.08)** | **1.68 (1.11–2.55)** |
| **Basic education** | 1 | 1 |
| **Intermediate education** | 0.99 (0.57–1.73) | 1.11 (0.63–1.95) |
| **High education** | 1.00 (0.58–1.72) | 1.16 (0.67–2.00) |
| **Income** | | |
| **1st quintile** | 1 | 1 |
| **2nd quintile** | 1.08 (0.47–2.47) | 1.18 (0.61–2.73) |
| **3rd quintile** | 0.88 (0.38–2.00) | 1.01 (0.43–2.40) |
| **4th quintile** | 0.89 (0.40–1.98) | 1.07 (0.45–2.59) |
| **5th quintile** | 0.96 (0.45–2.04) | 1.22 (0.53–2.80) |
| **Married or cohabited** | 1 | 1 |
| **Single** | 1.21 (0.78–1.86) | 1.14 (0.66–1.97) |
| ***Childhood adversity*** | | |
| **No adversities** | 1 | 1 |
| **1–2 adversities** | 1.19 (0.79–1.81) | 1.08 (0.71–1.65) |
| **3 or more adversities** | **1.91 (1.18–3.09)** | 1.52 (0.90–2.58) |
| **Parental mental disorder** | 1.83 (0.96–3.50) | 1.47 (0.71–3.03) |
| ***Social capital*** | | |
| **Low social support** | 1 | 1 |
| **Intermediate social support** | 1.01 (0.62–1.64) | 1.17 (0.71–1.93) |
| **High social support** | 0.93 (0.57–1.53) | 1.13 (0.67–1.89) |
| **Low participation** | 1 | 1 |
| **Intermediate participation** | 0.67 (0.40–1.10) | 0.71 (0.42–1.19) |
| **High participation** | 0.73 (0.43–1.22) | 0.78 (0.45–1.33) |
| **Low trust** | 1 | 1 |
| **Intermediate trust** | 0.82 (0.52–1.31) | 0.95 (0.42–1.19) |
| **High trust** | **0.52 (0.31–0.87)** | 0.78 (0.45–1.33) |
| ***Physical health*** | | |
| **No somatic diseases** | 1 | 1 |
| **1–2 somatic diseases** | 1.23 (0.82–1.86) | 2.53 (0.72–5.46) |
| **3 or more somatic diseases** | 1.44 (0.77–2.71) | 1.63 (0.73–3.64) |
| ***Mental health*** | | |
| **Anxiety disorder** | **3.18 (1.49–6.78)** | **2.53 (1.17–5.46)** |
| **Alcohol use disorder** | 2.02 (0.91–4.45) | 1.63 (0.73–3.64) |
| **No depressive symptoms**[d] | 1 | 1 |
| **Some depressive symptoms** | **1.98 (1.26–3.13)** | **1.64 (1.00–2.68)** |
| **Many depressive symptoms** | **4.01 (2.02–7.95)** | **3.03 (1.48–6.19)** |

[a] Multiple imputation was used to correct for non-participation in the follow-up.
[b] Only adjusted for sex.
[c] Only adjusted for age.
[d] No depressive symptoms: Beck Depression Inventory (BDI) score 0–8; Some depressive symptoms: BDI score 9–18; Many depressive symptoms: BDI score 19–63.

**Table 4**
Predictors of new-onset dysthymia in an eleven-year follow-up of a general population sample (n=31).[a]

| | Model 1: adjusted for age and sex OR (95% CI) | Model 6: adjusted for age, sex, education, income and marital status, childhood adversities, social capital, somatic and mental health OR (95% CI) |
|---|---|---|
| ***Sociodemographic factors*** | | |
| **Age (per year)**[b] | **0.95 (0.92–0.99)** | **0.93 (0.89–0.98)** |
| **Sex (female)**[c] | 1.07 (0.52–2.20) | 1.11 (0.52–2.35) |
| **Basic education** | 1 | 1 |
| **Intermediate education** | 1.20 (0.44–3.25) | 1.58 (0.57–4.34) |
| **High education** | 0.66 (0.21–2.03) | 1.08 (0.32–3.72) |
| **Income** | | |
| **1st quintile** | 1 | 1 |
| **2nd quintile** | 1.03 (0.19–5.52) | 1.12 (0.20–6.29) |
| **3rd quintile** | 1.26 (0.26–6.13) | 1.06 (0.18–6.32) |
| **4th quintile** | 1.38 (0.30–6.31) | 1.05 (0.18–6.04) |
| **5th quintile** | 1.03 (0.20–5.15) | 0.92 (0.15–5.54) |
| **Married or cohabited** | 1 | 1 |
| **Single** | 0.51 (0.16–1.60) | 0.37 (0.08–1.64) |
| ***Childhood adversity*** | | |
| **No adversities** | 1 | 1 |
| **1–2 adversities** | 2.29 (0.91–5.79) | 2.07 (0.80–5.32) |
| **3 or more adversities** | **3.98 (1.53–10.39)** | **3.36 (1.22–9.27)** |
| **Parental mental disorder** | 1.20 (0.28–5.19) | 0.62 (0.12–3.14) |
| ***Social capital*** | | |
| **Low social support** | 1 | 1 |
| **Intermediate social support** | 0.62 (0.24–1.59) | 0.76 (0.28–2.02) |
| **High social support** | 0.63 (0.27–1.48) | 0.77 (0.31–1.94) |
| **Low participation** | 1 | 1 |
| **Intermediate participation** | **0.40 (0.17–0.95)** | 0.45 (0.18–1.12) |
| **High participation** | **0.41 (0.18–0.96)** | 0.44 (0.18–1.09) |
| **Low trust** | 1 | 1 |
| **Intermediate trust** | 0.46 (0.20–1.03) | 0.48 (0.20–1.15) |
| **High trust** | **0.24 (0.08–0.73)** | **0.30 0.09–0.97)** |
| ***Somatic health*** | | |
| **No somatic diseases** | 1 | 1 |
| **1–2 somatic diseases** | **3.74 (1.33–10.50)** | **3.37 (1.19–9.52)** |
| **3 or more somatic diseases** | 3.15 (0.65–15.20) | 2.30 (0.44–12.05) |
| ***Mental health*** | | |
| **Anxiety disorder** | 2.86 (0.67–12.15) | 2.40 (0.49–11.72) |
| **No depressive symptoms**[d] | 1 | 1 |
| **Some depressive symptoms** | **2.38 (1.03–5.49)** | 1.51 (0.60–3.82) |
| **Many depressive symptoms** | 1.77 (0.24–12.90) | 0.81 (0.10–6.52) |

[a] Multiple imputation was used to correct for non-participation in the follow-up.
[b] Only adjusted for sex.
[c] Only adjusted for age.
[d] No depressive symptoms: Beck Depression Inventory (BDI) score 0–8; Some depressive symptoms: BDI score 9–18; Many depressive symptoms: BDI score 19–63.

## 5. Limitations

The long follow-up time in our study, eleven years, is a challenge. While it gives a distinct long-term perspective to the study, the impact of some predictors may attenuate over such a long period. We were not able to measure all relevant risk factors, such as recent stressful life events, which have been shown to increase the risk of depressive disorders (Wichers et al., 2012). The retrospective measurement of childhood adversities is another possible source of bias. The small number of new cases also limited the analyses we could carry out, and in particular in dysthymia, type II error could not be excluded. Moreover, we did not have information on the participants' mental health status during the follow-up, and probably did not capture all incident cases of depressive disorders that occurred during the follow-up. Finally, it is possible that we were unable to exclude all participants with a previous episode of depression, as information on lifetime diagnoses was based on register data and self-report of doctors' diagnosis of depression, and therefore we may have missed undiagnosed or treated prior depression.

## 6. Conclusions

Persons with subclinical depressive symptoms, anxiety disorders, low trust, and multiple childhood adversities are at a higher risk of developing a depressive disorder, and are a potential target group for preventive interventions. Several types of preventive efforts, such as psychological interventions, are effective in reducing incidence of depression (van Zoonen et al., 2014), and this is a key measure to reduce the enormous burden of depressive disorders. In addition, policies should aim at minimising the impact of different adversities, such as parental illness, unemployment, financial difficulties or bullying on children, to reduce their risk of later depression.

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## Ethical standards

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008.

## Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.jad.2016.08.051.

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