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Review

A systematic review comparing clinical features in early age at onset and late age at onset late-life depression

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ABSTRACT

Background: Although evidence suggests that there are neurobiological differences between unipolar depression in younger versus older adults, conflicting evidence exists about whether these manifest as clinically identifiable differences.
Method: We conducted a systematic review of aetiological, phenomenological and outcome studies to examine the evidence for a distinction between early onset (EOD) and late onset (LOD) depression. A literature search was completed using the computer databases MEDLINE, EMBASE, PSYCHINFO and PUBMED for papers published between January 1982 and December 2012 which compared groups with EOD and LOD. Studies were included if they were of older people and compared symptoms, aetiological factors or outcomes. We conducted a quality assessment of included articles.
Results: We identified 23 articles which met entry criteria. The only clinical feature which was different between the groups was a higher frequency of a family history of mood disorders in EOD.
Limitations: The number of studies identified was low and their quality was generally poor.
Conclusions: Although neurobiological studies have reported differences between EOD and LOD, generally these do not appear to translate into identifiable distinguishing clinical features.

Contents

1. Introduction

Depressive disorder is common throughout the lifespan but is depression the same illness in adults of all ages or are there differences in older adults? It is well recognised that depression is much more frequent overall in old age due to its association with age-related diseases in general and with specific diseases, e.g. Alzheimer's disease and Parkinson's disease, in particular. The issue of interest is whether unipolar depressive disorder occurring outside these illness contexts is different in any way. There has been dispute about whether the prevalence changes but it is now generally accepted that it does not and remains at about 2% for

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Major Depressive Disorder (MDD) (Beekman et al., 1999). But is late-life depression different in other respects, such as phenomenology or aetiology? This question is important because if it is different than alternative treatments may be warranted. The answer to this question may be approached in two ways. One is to examine features of late-life depression (usually taken as depression in people over 60) and compare these with the same features in younger adults with depression. This approach has been taken in a recent review of phenomenology, which found only a few modest differences in symptoms assessed using the Hamilton Depression Rating Scale (Hegeman et al., 2012) and in an earlier review of treatment outcomes which also found few differences in prognosis between the two groups (Mitchell and Subramaniam, 2005). The principal limitation of this approach is that real differences occurring in people who develop depression for the first time in old age may be obscured by them being examined together with adults growing old with depression and typically having multiple illness episodes, a point made by Mitchell and Subramaniam. A second and complementary approach is to divide older adults with depression into those whose illness first begun in earlier adulthood (early onset depression, EOD) and those whose first illness episode occurred in old age (late-onset depression, LOD). Research to date taking this second approach has produced conflicting results, which may be a consequence of differences in methodology, heterogeneity of samples and differing criteria used for age at onset. Therefore we conducted a systematic review (of aetiological, phenomenological and treatment studies over the past three decades) to examine the evidence base for a distinction between early onset (EOD) and late onset (LOD) depression. We chose to review clinical studies whose purpose was to examine age at onset of depressive illness and its relationship to phenomenology, aetiological risk factors and treatment outcomes; that is, we chose not to review the large literature on neurobiological investigations (including neuroimaging studies) which have assessed inflammatory markers, neurotrophic factors, genetic polymorphisms and other aspects of the biology of depression. We also chose not to include neurocognitive impairments in our review as a systematic review of the literature comparing the neuropsychological profile of LOD and EOD and healthy controls was reported in 2007 (Herrmann et al., 2007) and a search revealed only one paper with possible new data (Kohler et al., 2010a) and this did not report a direct comparison of EOD and LOD. We hypothesised that there would be no differences between EOD and LOD depression in symptoms (phenomenology excluding cognition), aetiological risk factors and treatment outcome and prognosis.

2. Methods

The literature search was completed using the computer databases MEDLINE, EMBASE, PSYCHINFO and PUBMED accessed via the web of knowledge. Searches for relevant papers published between January 1982 and December 2012 were conducted. We chose a broad approach with our search terms, using those that would be least likely to miss any articles, thus our search terms included the keywords: “Depression or depressive disorder or affective disorder or affective symptoms or major depressive disorder or mood disorder” and “age of onset or early onset or late onset or early or late or onset”. Each word was entered both as a ‘topic’ and ‘MeSH’ term. Papers were limited to those published in English and related to humans.

Titles and abstracts were screened individually by both authors searching for articles that directly compared early onset and late onset depression and included data on symptoms or aetiological factors or treatment outcomes. Reference lists from all of the relevant papers were hand-searched for any additional relevant articles which might have been missed by the original search. Papers were excluded from the analysis if they did not directly compare EOD and LOD (that is studies that compared adults of different ages rather than comparing by age at onset of depression) or if the study population contained bipolar patients or patients with dementia, or if they focused predominantly on children or young adults (wrong age cohort). As above we also excluded those studies examining genetics, neuroimaging and other biological studies because this would have greatly enlarged the review and made it unmanageable. Finally, in keeping with an earlier review (Mitchell and Subramaniam, 2005), only studies including more than 20 subjects per group were considered as providing representative data (such sample sizes would have the power to detect an important difference).

2.1. Quality assessment

In line with a previous study (Hegeman et al., 2012) we conducted a quality assessment of the papers included in this review. Both authors assessed the quality of the studies using a checklist with the following criteria (modified from (Hegeman et al., 2012)).

(1) Both age groups were selected from the same source population.
(2) Population characteristics and inclusion and exclusion criteria were described.
(3) Semi-structured diagnostic instruments were used.
(4) Differences in overall disease severity between groups were controlled for or no statistically significant differences in depression severity were reported.
(5) Chronological age was controlled for or no statistically significant difference in chronological age was reported.
(6) Physical illness burden or medical co-morbidities were controlled for or reported as not statistically significant.
(7) Assessment of age at onset of symptoms assessed subjectively using retrospective interview or objectively using medical records.

Each question was coded as 0 or 1 and criteria were combined to give a total score between 0 and 7. If no information was provided as to whether a criterion was met or not it was coded as 0. The cut-off for high or low quality was defined at a score of 5 or more based on a 60% cut-off point used in previous studies of quality assessment. Discrepancies between reviewers were resolved though discussion.

3. Results

The original search yielded 255 articles. Exclusion of duplicate articles and irrelevant references after a first screening of title and abstracts left 78 potential papers which were reviewed in full. 5 further papers were identified via hand-searching of the relevant bibliographies. Of these 83 articles 35 were excluded because they did not report on an outcome of interest; 15 focused on biological outcomes including neuroimaging, 10 focused primarily on cognition and 10 focused on genetics or endocrine markers. Of the remaining 48 articles 23 were excluded because they did not directly compare EOD and LOD. A further two articles were excluded because they reproduced data from previous studies. Finally 23 articles were included in this systematic review comparing EOD and LOD. As shown below in Tables 2–4 the extent of variety in sampling, design and measurements precluded a meta-analysis.