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Review

Prevention of depression in older age

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ABSTRACT

Depression is a common disorder in later life that is associated with increased disability and costs, and negative health outcomes over time. Antidepressant treatments in the form of medications or psychotherapy are available, but a large proportion of those treated fail to respond fully, and relapse or recurrence of symptoms is frequent among those who recover. Hence, successful prevention would avoid these negative outcomes. This paper selectively reviews currently available observational and trial data on the prevention of depression. It initially reviews risk factors associated with depression, and then discusses strategies for primary (including universal, selective and indicated), secondary and tertiary prevention. Currently available evidence suggests that selective and indicated preventive interventions are feasible and initial results look promising. Existing trial data indicate that ongoing antidepressant treatments reduce the risk of relapse and recurrence of symptoms, but benefits may not extend beyond two or three years. At this point in time, no interventions have been shown to reduce the long term complications associated with depression. Mental health professionals will need to work collaboratively to develop primary, secondary and tertiary preventive interventions that are effective at targeting relevant risk factors systematically and that can be easily adopted into clinical practice.

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1. Introduction

Understanding the pathways that lead to the onset, persistence and recurrence of depressive symptoms is a critical first step on the road to developing effective preventive measures. Over the past 50 years health professionals have described numerous factors that seem to modulate the risk of depression across the lifespan,

although translation of this knowledge into practice has been slow and haphazard. Nonetheless, progress has been made. This paper will selectively review our growing understanding of the risk factors associated with depression and the strategies that have been used to date to prevent depression in later life.

2. Risk factors for depression in later life

Over one decade ago, Cole and Dendukuri completed a systematic review of risk factors prospectively associated with depression in later life [1]. Their pooled analyses showed that depression was more frequent among women and those with a disability, recent bereavement, poor sleep and past history of depression. The association between depression and age, education, marital status, socioeconomic status, poor health, cognitive impairment, living alone and new medical illnesses did not reach statistical significance. However, the number of studies providing usable data for each the risk factors analysed was small (2-7) and the review of risk factors was limited both in scope and time. There is consistent evidence that remote factors, such as childhood abuse, increase the risk of mood disorders in young [2,3] and older adults [4]. Limited access to education, adoption of hazardous lifestyle practices (e.g., risk drinking, smoking, physical inactivity, obesity), poor social support, financial strain, lack of a confidant, chronic medical problems, and significant life events (e.g., death, divorce, moving houses) may all contribute to increase the risk of depression in later life [5,6].

Existing data suggest that this multitude of factors do not mediate the risk of depression in isolation. Instead, each seems to facilitate exposure to a novel risk factor in a chain of events that ultimately leads to the onset of depressive symptoms. For example, people who experience childhood adversity are more likely than their counterparts to adopt hazardous lifestyle practices, such as the abuse of substances [7], which in turn are associated with social disadvantage and future chronic health problems [8] that increase the risk of depression in later life [9]. In addition, empirical data from association studies suggest that certain genetic polymorphisms may decrease a person's ability to manage psychosocial or physiological stress successfully, rendering them more susceptible to the onset of depressive symptoms. For example, Caspi and colleagues [10] showed that a common polymorphism of the serotonin receptor gene increases the probability of depressive episodes as the number of stressful life events accumulates (i.e., there is a gene x environment interaction mediating the risk of depression). Likewise, variations of the C-reactive protein gene that dampen response to physiological stress seem to increase the risk of depression, possibly because they hamper recovery from insult [11,12]. Taken together, the results of these observational studies indicate that exposure to risk factors that increase the risk of depression in later life starts at the time of gamete production and continues throughout the life course. If that is true, then the benefits of early interventions, such as education, may be long lasting [13]. Fig. 1 illustrates a hypothetical model whereby individual risk factors increase the chance of downstream exposure to additional risk factors as well as to depression [5].

3. Interventions to prevent depression in later life

3.1. Primary prevention of depression

Primary preventive interventions can be classified as universal, selective or indicated – they aim to avert the onset of clinically significant depressive symptoms. Universal interventions target the entire population at risk – they are expensive and require that a large number of people receive treatment for one person to benefit. Selective preventive interventions target people at high

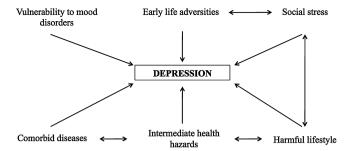


Fig. 1. The figure illustrates possible pathways along the lifespan that facilitate the expression of depressive disorders. Vulnerability to mood disorders might include genetic variations that decrease the ability of the individual to manage stress effectively (e.g., serotonin receptor and CRP genetic polymorphisms). Early life adversities might be represented by physical or sexual abuse, limited education and loss of parents during childhood. Social stress might be characterised by financial strain, social isolation and poor social support. Common harmful lifestyle practices might consist of smoking, hazardous or harmful alcohol use, use of substances, physical inactivity and poor dietary habits. Examples of intermediate health hazards could be hypertension, obesity and diabetes, and examples of common comorbid diseases associated with depression could include coronary heart disease, stroke, Parkinson's disease, dementia, cancers, sensory impairment, chronic pain and chronic respiratory diseases. In the proposed model, each factor facilitates exposure to other harmful downstream factors as well as to depression.

risk of depression (such as poststroke patients), whereas indicated preventive strategies are used to treat people with symptoms of depression who remain under the threshold for the diagnosis of a depressive episode (subsyndromal depression). Because the risk of depression is greater among those who are vulnerable or have subsyndromal depression, interventions targeting these populations are more economical than universal prevention because less people need to be treated to avoid one case of depression. But what should we do once we identify our target population?

The management of risk factors to reduce the risk of undesirable health events has been used successfully in several areas of medicine, such as cardiology. The introduction of the Framingham score, and subsequent risk tables, led to the systematic assessment and management of factors prospectively associated with myocardial infarction and strokes, such as smoking, diabetes, blood pressure, lipid profile, gender and age [14]. Ensuing interventions targeting these factors have been successful at reducing the incidence of cardiovascular events [15].

A similar approach to the assessment and management of risk factors is yet to adopted in psychiatry, but preliminary data are starting to emerge. Schoevers and colleagues reported longitudinal data arising from the Amsterdam Study of the Elderly, which recruited 4051 adults aged 65 years or over living in the community [16]. Three years later, 2244 participants were available for a new assessment that included an interview with the Geriatric Mental State AGECAT. Three hundred and nine people (13.8%) developed clinically significant depressive symptoms during the follow up period, which were more frequent among those who reported loss of spouse, sleep complaints, past history of anxiety or depression, disability or chronic medical problems, and who showed evidence of generalised anxiety or subsyndromal depression at study entry. The investigators then developed two prevention models based on these data: one for people who had subsyndromal symptoms of depression at study entry (indicated prevention) and another for those who had lost their spouse (selective prevention). Risk factors associated with conversion to depression amongst those with subsyndromal symptoms included widowhood, disability, chronic illness, living alone and female gender. Factors associated with conversion to depression among those who had recently lost a spouse included disability, chronic illness and female gender. The absolute risk of depression when all risk factors were added approached 50%. The models derived from the Amsterdam Study of the Elderly

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