

INVITED REVIEW

Origins of depression in later life

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ABSTRACT

Background. Despite the burden of depression in late life, its origins present a paradox to investigators and clinicians alike.

Method. We review biological (genetics and heredity factors, neurotransmitter dysfunction, endocrine changes, vascular disorders, and medical co-morbidities), psychological (personality attributes, neuroticism, cognitive distortions, and the lack of emotional control and self-efficacy) and social (stressful life events, bereavement, chronic stress or strain, socio-economic disadvantage and impaired social support) origins of late-life depression based upon an extensive though not exhaustive review of the extant literature. In addition, modifying psychological and social factors are discussed.

Results. Older adults appear to be at greater risk for major depression biologically, such as depression resulting from vascular changes, yet the frequency of depression is lower compared to younger adults. Older adults may be protected psychologically due to factors such as socio-emotional selectivity and wisdom, compared to younger adults, and perhaps relatively protected from social risks.

Conclusions. A biopsychosocial approach to evaluating the origins of late-life depression is heuristically valuable, a continual reminder of the many factors that contribute to the onset and persistence of clinically significant symptoms in late life.

INTRODUCTION

Depressive symptoms and disorders are frequent causes of emotional and physical suffering, decrease the quality of life and increase the risk for death among older adults (Berkman *et al.* 1986; Blazer *et al.* 2001; Blazer, 2003). Even so, the origins of late-life depression present a paradox to investigators and clinicians alike. Older adults appear to be at greater biological vulnerability to depression, yet community surveys in Western societies have repeatedly documented a lower frequency of late-life depressive symptoms and cases of

depressive disorders in controlled analyses compared to midlife (Blazer *et al.* 1991; Weissman *et al.* 1991) Therefore, a biopsychosocial model of etiology (Engel, 1977, 1980; Blazer, 2002; Lindau *et al.* 2003) is especially applicable to the elderly primarily because it reminds us that the origins of late-life depression are multiple and range across all three domains. Even so, this model can be misleading given the tight connection between the domains. For example, psychological dimensions have biological mechanisms and biological models imply input from the environment.

Psychological and social factors may be more protective against depressive symptoms and disorders in later life compared to earlier stages of the life-cycle. This paradox, however, must not blind us to the adverse consequences of

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Table 1. General and age-specific risk and protective factors for depression in later life

General risk factors (factors that predispose to depressive symptoms and disorders across the life-cycle including later life)

- Biological risks
 - Hereditary (e.g. findings from twin studies)
 - Female sex
 - Underactivity of serotonergic neurotransmission
 - Hypersecretion of cortisol (associated with hippocampal atrophy)
 - Low levels of testosterone
 - Stroke
 - Medical illness and functional impairment
 - Alcohol abuse and dependence
- Psychological risks
 - Personality disorder
 - Neuroticism
 - Learned helplessness
 - Cognitive distortions
 - External locus of control
- Social risks
 - Stressful life events and daily hassles
 - Bereavement
 - Socio-economic disadvantage
 - Impaired social support

Specific risk and protective factors (factors that are especially relevant to depressive symptoms and disorders in later life)

- Biological risks
 - Genetic polymorphisms or mutations (such as CADASIL)
 - Low levels of DHEA
 - Cortical and subcortical ischemia
 - Alzheimer's disease
- Psychological protective factors
 - Socio-emotional selectivity
 - Wisdom

severe late-life depression, including an increased risk for non-suicide mortality (Schulz *et al.* 2002), suicide (Conwell *et al.* 2002), physical disability (Berkman *et al.* 1986) and risk of adverse outcomes for specific illnesses, such as cardiovascular disease (Sullivan *et al.* 1997) and diabetes (Blazer *et al.* 2002a).

The multiple biological, psychological, and social causes of depressive symptoms and disorders in late life, however, are not competing but complementary and almost always transactional. For example, vulnerability to social stressors often derives from underlying biological mechanisms that interact with social risk factors. In this article we provide a representative but not comprehensive review, focusing upon more recent studies of the origins of late-life depressive symptoms and disorders, that is, depression experienced by older adults whether that depression had its onset in later life or earlier in the life-cycle. Where possible we

distinguish those studies that explore the origins of depressive symptoms from those that focus on diagnosed disorders such as major depression. We also distinguish those origins that are specific to older adults from those that affect adults across the life-cycle including older adults, as summarized in Table 1.

We divide the review into biological, psychological, and social origins more for convenience of organization than to suggest that these domains cannot be connected theoretically or demonstrably. We stop short, however, of providing a grand model of the web of causation (MacMahon & Pugh, 1970), for if the plethora of studies emerging in recent years has taught us anything, it taught us the danger of potentially limiting our inquiries to fit the Procrustean bed of such models.

Biological origins

The discovery of biological risk factors and their putative mechanisms has been a fertile area of basic and clinical research into the origins of late-life depression.

Genetics and heredity

Twin studies of older adults in Scandinavia provide insight into the relative contribution of heredity and environment to reported depressive symptoms. Among elderly twins in Sweden, for example, genetic influences accounted for 16% of the variance in total depression scores on the Center for Epidemiologic Studies Depression Scale (CES-D) and 19% of somatic symptoms. Genetic influences, however, contributed to a minimal amount of the variance symptoms of depressed mood and positive affect (Gatz *et al.* 1992). Therefore, not all depressive symptoms appear to be equally influenced by heredity.

Major depression is more common among women compared to men and this difference persists into late life (Krause, 1986). Many factors have been suggested to explain these differences, such as selective survival. For example, men die earlier and mortality may be affected by genetic factors (Hazzard, 1999). Since men and women are seldom compared in matched analyses, examining older unlike-sex twin pairs provides an opportunity to explore these sex differences. In one such study, women had a higher frequency of depressive symptoms and

depressive diagnoses and the sex difference increased over time (Takkinin *et al.* 2004). Even so, the likelihood of identifying a mood disorder in the relative of an older adult is lower than in midlife (Hopkinson, 1964; Brodaty *et al.* 2001).

Most hypothesized genetic markers for late-life depression have not stood the test of well-controlled studies, yet some present intriguing possibilities. Despite the considerable interest in the $\epsilon 4$ allele of the apolipoprotein E gene, no association was found in a community sample between the E4 allele and depressive symptoms (Blazer *et al.* 2002*b*). Other investigators have concentrated on genes that may be associated with cerebral vascular lesions associated with depression. In one study, patients with late-onset major depression exhibited a higher frequency of C677T mutation of the MTHFR (methylene tetrahydrofolate reductase) enzyme compared to controls. This mutation may place older persons at risk for major depression associated with cerebral vascular lesions (vascular depression) (Hickie *et al.* 2001). A rare disease, CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy), derives from the notch 3 gene. Depression is one of the initial symptoms in this condition. Investigators who analyzed three longitudinal twin studies of the elderly in Sweden (mean age 73 years), found an association between the 5-HTR_{2A} gene promoter polymorphism and depressive symptoms for the A/A genotype (Jansson *et al.* 2003). These preliminary findings suggest that genetic polymorphisms or mutations may predispose older adults to vascular depression (Desmond *et al.* 1999; Krishnan, 2002).

Neurotransmitter dysfunction

Underactivity of serotonergic neurotransmission has been the focus of much research on the pathophysiology of depression in younger adults. Other neurotransmitter systems, such as norepinephrine and dopamine dysfunction, have also been implicated as well but we concentrate on serotonin neurotransmission. Although serotonin activity, specifically, 5-HT_{2A} receptor binding, decreases dramatically in a variety of brain regions through midlife, there is less decrease from midlife to late life. In one study, the investigators found that 5-HT_{2A} receptors in normal subjects decreased markedly

from young adulthood to midlife (70 % from the levels at age 20 years through the fifth decade), and then leveled off as age advanced (Sheline *et al.* 2002). The activity of these receptors, however, may vary with age.

The relationship of serotonin depletion can be investigated indirectly by observing radioisotope-labeled or tritiated imipramine binding (TIB) sites on platelets in the elderly. There is a significant decrease in binding sites in older people with major depression compared to normal controls and subjects with Alzheimer's disease, but no decrease in binding capacity (Nemeroff *et al.* 1988).

Endocrine changes

Hypersecretion of corticotropin-releasing factor (CRF) has been associated with depression. CRF is thought to mediate sleep and appetite disturbances, reduced libido, and psychomotor changes (Arborelius *et al.* 1999). CRF, however, is also diminished with normal aging (Gottfries, 1990). Aging is associated with an increased responsiveness of dehydroepiandrosterone sulfate (DHEA-S) to CRF (Luisi *et al.* 1998). Low levels of DHEA have been associated with higher rates of depression and a greater number of depressive symptoms in community-dwelling older women (Yaffe *et al.* 1998).

Total serum testosterone levels decline with aging yet we do not know the level at which these levels become abnormal (Liverman & Blazer, 2004). Testosterone levels were lower in elderly men with dysthymic disorder than in men without depressive symptoms in one study (Seidman *et al.* 2001), yet the efficacy of testosterone treatment for major depression in men has not been established (Liverman & Blazer, 2004). Hormone replacement in women has been associated with some improvement in mood (Sherwin & Gelfand, 1985).

Anatomical changes have been associated with endocrine dysregulation as well as late-life depressive symptoms, suggesting a vicious cycle downward to chronic and moderately severe depressive symptoms. For example, depressive symptoms have not only been associated with atrophy of the hippocampus, they have been hypothesized to cause the atrophy (Sheline *et al.* 1996; Sapolsky, 2001; Steffens *et al.* 2002). Cumulative stress over the life-cycle may lead to a sustained increased secretion of

cortisol which in turn causes loss of pre-existing hippocampal neurons (Sapolsky, 1996), a loss that may be prevented in part by use of antidepressant medications (Czeh *et al.* 2001).

Depressive symptoms can lead to increased cortisol secretion (Davis *et al.* 1984). Cortisol inhibits neurogenesis leading to hippocampal volume loss that may mediate the cognitive symptoms of depression (Sapolsky, 2001). Other mechanisms that have been proposed to explain volume loss have been described above and include glutamate neurotoxicity (Sheline, 2003) decreased brain-derived growth factor (Sheline, 2003), decreased neurogenesis (Gould *et al.* 1999), and loss of plasticity. For example, early life stress may produce a permanent hypersensitivity to stress, with the production of ongoing HPA axis dysregulation and with repeated episodes of major depression plasticity may give way to permanent damage (Heim *et al.* 2000).

Vascular depression

The association of depressive symptoms and vascular risk factors has long been known (Post, 1962). For example, major depression is a frequent outcome of stroke (Robinson & Price, 1982), occurring in approximately one-third of all ischemic stroke survivors (Parikh *et al.* 1990). Hypertension has also been associated with increased risk of major depression (Rabkin *et al.* 1983), although the literature is not consistent (Lyness *et al.* 2000).

Boosted by new tools of inquiry, especially magnetic resonance imaging (MRI), investigators have proposed a vascular-based depression among the elderly (Post, 1962; Krishnan *et al.* 1988; Coffey *et al.* 1990; Kumar *et al.* 2002; Olin *et al.* 2002). In one study of 139 depressed older adults, 54% met neuroimaging criteria for subcortical ischemic vascular depression. Age was most strongly associated with the increased prevalence of these changes. Lassitude, a history of hypertension, and poorer outcome were also positively associated with the diagnosis, whereas a family history of mental illness and loss of libido were negatively associated with the diagnosis (Taylor *et al.* 2003; Krishnan *et al.* 2004). Vascular depression is linked with white-matter hyperintensities (WMH), bright regions seen in the brain parenchyma on T2-weighted MRI scans (Krishnan *et al.* 1997; Guttmann *et al.* 1998). These lesions

are thought to represent injury to white-matter tracks and may contribute to the disruption of neural circuits associated with depression (Taylor *et al.* 2003).

The vascular depression impairments resemble impairments exhibited in frontal lobe syndromes. MRI of depressed patients has revealed structural abnormalities in areas related to limbic-cortical-striatal-pallidal-thalamic-cortical pathways (George *et al.* 1994), including the frontal lobes (Krishnan *et al.* 1993), caudate (Krishnan *et al.* 1992), and putamen (Husain *et al.* 1991; Sheline, 2003). In three-dimensional MRI studies of mood disorders, many of the structures that compose this tract have been found to have volume loss or structural abnormalities (Sheline, 2003).

Recent interest has also focused on a smaller size of the orbital frontal cortex in late-life depression (Lai *et al.* 2000). The frontal white matter (not gray matter) lesions of vascular depression are associated with increased myoinositol-creatinine and choline-creatinine ratios. These changes may reflect biological changes in non-neural (glial) tissue, which in turn affects synaptic activity (Kumar *et al.* 2002). Whether investigators hypothesize endocrine dysregulation or vascular changes, a major advance in the field is the concept that older adults may be at greater risk for depressive symptoms and major depression because key pathways have been disrupted.

Cognitive function in late-life major depression supports these neuroanatomic findings, specifically the disruption of functional pathways. For example, disruption of the limbic-cortical-striatal-pallidal-thalamic-cortical pathways leads to impairment in visuospatial ability, memory, speed of information processing, and executive functioning, with executive deficits being particularly related to late age at onset of first lifetime depressive episode (Alexopoulos *et al.* 2000; Lesser *et al.* 2000; Butters *et al.* 2004). Given that late-life major depression is characterized by slowed information processing, which affects all realms of cognition, this supports the concept that frontostriatal dysfunction plays a key role.

Alzheimer's disease

Clinically significant depressive symptoms can be identified in approximately 20% of

Alzheimer's disease (AD) patients (Reifler *et al.* 1982; Patterson *et al.* 1990). Major depression with cognitive impairment (even if the cognitive problems remit) increases the risk for AD over 5 years (Alexopoulos *et al.* 1993). Depressive symptoms may be common even in older people with mild dementia of the AD type (Rubin *et al.* 2001). Given the co-morbidity of major depression and AD, the depression/dementia syndrome may have in large part a common pathophysiology (Olin *et al.* 2002). The mechanisms, however, have yet to be worked out.

Medical and psychiatric co-morbidity

Depression frequently results from and complicates the recovery of older patients who experience myocardial infarction and other heart conditions (Sullivan *et al.* 1997), diabetes (Blazer *et al.* 2002a), hip fracture (Magaziner *et al.* 1990), and stroke (Robinson & Price, 1982). In a study of community-dwelling Mexican American elderly, depressive symptoms were associated with diabetes, arthritis, urinary incontinence, bowel incontinence, kidney disease, and ulcers (Black *et al.* 1998a). In general, poor functional status secondary to physical illness and dementing disorders are perhaps the most important cause of late-life depressive symptoms (Hays *et al.* 1997; Bruce, 2001). In one study, depressive symptoms increased the risk for activities of daily living disability and mobility disability over 6 years by 67% and 73% respectively (Penninx *et al.* 1999).

Health status consistently is associated with depressive symptoms in cross-sectional studies of older adults (Kraaij *et al.* 2002). Yet it is not clear whether a change in health status leads to a change in depressive symptoms (Fiske *et al.* 2003). In one study, health status was correlated with depressive symptoms but new illnesses in the previous 3 years did not consistently predict increases in depressive symptoms (Fiske *et al.* 2003). In the same study, there was no interaction between age and health, suggesting that health is not more important in predicting depressive symptoms in late life than it is earlier in life.

Depressive symptoms and disorders in late life co-occur with psychiatric disorders as well. On balance, the co-morbidity of late-life depression with other psychiatric disorders is

less frequent than earlier in the life-cycle (the dementing disorders being an exception). The reason is that other psychiatric disorders are less frequent in late life compared to younger ages. Alcohol use and major depression are associated in community studies of older adults (Devenand, 2002). Anxiety is commonly comorbid with depressive symptoms, whether or not the symptoms meet criteria for a depressive disorder (Blazer *et al.* 1987).

In summary, biological vulnerability to depressive symptoms and disorders appears to be greater in later life compared to midlife. Many changes in the brain coupled with the increased frequency of diseases known to be associated with depression are typical of the aging process, especially as the individual advances into the era of the oldest old (Blazer, 2000). Some biological protective mechanisms may increase with age but these remain unknown at present.

Psychological origins

Several psychological factors have been proposed as causes of depressive symptoms and disorders in late life, including personality attributes, neuroticism, cognitive distortions, and emotional control. These factors, however, are not specific to the origins of depression in older adults and we, therefore, briefly address examples of recent studies.

Personality attributes

A recent study found that older patients with a personality disorder were four times more likely to experience maintenance or re-emergence of depressive symptoms compared to those without. No specific personality disorder traits were associated with clinical features of depression such as age of onset and number of previous episodes, but some of the traits were associated with psychological correlates including hopelessness and ambivalence regarding emotional expression (Morse & Lynch, 2004). When reviewing personality factors, we must recognize that a depressed mood may alter behavioral styles as well as cognition.

Personality also interacts with other factors. In a sample of patients with major depression and non-depressed controls, the effect of stressful life events was modified by cognitive/personality styles in their association with

late-onset depression, adjusting for medical illness and reduced physical functioning. Major depression was 6–11 times more likely as a function of these interactions (Mazure *et al.* 2002). In another study, older adults without high neuroticism and ongoing life difficulties were not at increased risk for major depression secondary to stressful life events. On the other hand, high neuroticism and long-term difficulties increased the risk, even without the occurrence of a stressful life event (Ormel *et al.* 2001).

Neuroticism

Neuroticism, a construct rarely applied by psychiatrists in North America, is frequently assessed in Europe and Australia and has been consistently associated with late-life depressive symptoms in cross-sectional and longitudinal studies of community samples (Henderson *et al.* 1993, 1997; Lyness *et al.* 2002). This association has been observed in residential homes as well (Eisses *et al.* 2004). In one follow-up study, older adults with low levels of neuroticism were less likely to develop a depressive disorder compared to subjects with higher neuroticism (Oldehinkel *et al.* 2001).

Cognitive distortions

Cognitive distortions (Beck, 1987) have also been proposed as a cause of late-life depressive symptoms and disorders. This theory proposes that depressed individuals may overreact to life events or misinterpret these events and exaggerate their adverse outcome. In a recent study of the experience and impact of adverse life events comparing older patients with major depression to patients with dysthymia and healthy controls, patients with major depression reported more recent life events with greater negative impact, particularly interpersonal conflicts (Devenand *et al.* 2002). The authors point out it is not clear if the reported impact reflects an increased vulnerability or a bias in reporting due to current depressed mood. In a recent study from a community sample, elderly persons with more depressive symptoms used acceptance, rumination and catastrophizing to a higher extent and positive reappraisal to a lower extent than those with fewer symptoms (Kraaij & de Wilde, 2001).

Emotional control and self-efficacy

In the Longitudinal Aging Study Amsterdam (Beekman *et al.* 1995*b*), major and minor depression and persistence and emergence of depressive symptoms over 3 years was predicted by external locus of control (Beekman *et al.* 2001). Higher levels of mastery have been shown to have a direct association with fewer depressive symptoms in older adults, and to buffer the adverse impact of disability on depression (Jang *et al.* 2002). Self-efficacy has been shown to have a direct effect and also work indirectly through its effect on social support to prevent depressive symptoms in a sample of older adults followed for 1 year (Holahan & Holahan, 1987).

Social origins

A number of social stressors have also been proposed as contributing to late-life depressive symptoms and disorders, including stressful life events, bereavement, chronic stress or strain, socio-economic status and impaired social support. These factors are not unique to older adults yet the relative contribution of these factors appears to vary across the life-cycle.

Stressful life events

One early study found a strong association between both severe life events (i.e. bereavement, life-threatening illness of someone else, major personal illness, etc.) and social difficulties (difficulties in health of someone close to subject, housing, marital and family relationships, etc.) with the onset of major depression in late life (Murphy, 1982). Those lacking a confidant(e) were particularly vulnerable to the effects of life stress, supporting the hypothesis that social support may buffer the effect of a stressful event. In contrast, a meta-analysis of 25 studies of the relationship between negative life events and depression in late life, revealed that the total number of life events and the total number of daily hassles were strongly associated with depressive symptoms, while sudden unexpected events were not related to depression score (Kraaij *et al.* 2002).

At first glance, it would appear that older adults are at greater risk for depressive symptoms secondary to stressful life events. Yet three qualifiers temper the risk among the elderly. First, ongoing difficulties may have a smaller

effect on the risk for depression in older adults compared to younger adults (Bruce, 2002). One group found that the onset of depressive symptoms were not associated with baseline (ongoing) psychosocial stressors but were associated with factors that changed through time (Kennedy *et al.* 1990). Second, most stressful events that lead to late-life depression are predictable or 'on time' events. For example, death of a spouse is a severe, at times catastrophic, event that frequently leads to depression. In early or midlife it is unexpected and the adjustment is especially difficult. In late life, by contrast, most older people recognize, just by observing their peers, that death of a spouse is frequent and have actually rehearsed the event, such as considering what they might do if a spouse dies.

Third, many events that can lead to depression are more frequent early in life compared to late life, such as divorce and difficulties with the law. In one study significant difficulty with the law (something more grave than a traffic violation) was reported during the preceding year by 9% of younger adults compared to less than 1% among older adults (Hughes *et al.* 1988).

Bereavement

One life event potentially associated with depression in late life is bereavement (Clayton, 1990). Studies have found bereavement to be associated with depressive symptoms in older adults in cross-sectional and longitudinal studies (Prigerson *et al.* 1994; De Beurs *et al.* 2001). For example, in a study of 1810 community-dwelling older adults, onset of clinically significant depressive symptoms over a 3-year follow-up was predicted by death of a partner or other relatives. While other studies have found bereavement to predict depressive symptoms (Prigerson *et al.* 1994) others have not (Prince *et al.* 1998).

Chronic stress or strain

Chronic strain is associated with depression in older adults. The prevalence of depressive symptoms in caregivers of people with dementia is 43–47% (Livingston *et al.* 1996; Waite *et al.* 2004). For this reason, providing support for caregivers is most important to prevent the

onset and progression of depression in this vulnerable group of older people. In cross-sectional analyses using data from a community study of older Mexican Americans, financial strain was associated with level of depressive symptoms (Black *et al.* 1998*b*).

Socio-economic status

Socio-economic disadvantage was associated with prevalence and persistence of depressive symptoms over 2–4 years in a sample of community-dwelling adults 50 years or older who originally met criteria for major depression (Mojtabai & Olfson, 2004). While level of education did not predict emergence of depressive symptoms over 1 year (Beekman *et al.* 1995*a*) in a study from The Netherlands, emergence of depression over a 3-year period was predicted by lower level of education (Beekman *et al.* 2001).

Impaired social support

Social support is a multifactorial construct, including perception, structure of the social network, and tangible help and assistance (Turner & Turner, 1999). The most robust relationship between impaired support and late-life depressive symptoms, however, has been found with perceived support (Bruce, 2002). In a recent community study in Hong Kong, impaired social support and depressive symptoms were associated (including network size, network composition, social contact frequency, satisfaction with social support, and instrumental-emotional support) (Chi & Chou, 2001). In longitudinal studies, poor social support predicted number of depressive symptoms at follow-up after 3–6 years (Henderson *et al.* 1997). Insufficient social network predicted the incidence of major depression in a sample of 875 non-depressed elderly people followed for 3 years (Forsell & Winblad, 1999).

Support can mediate between risk factors and the onset of depression. In a recent analysis using growth-curve modeling, perceived social support was shown to mediate the relationship between disability and depressive symptoms over time (Taylor & Lynch, 2004). In a prospective study of 1940 community-dwelling elderly, the effect of stress on incident depression was modified by environmental vulnerability

including marital status and social support (Geerlings *et al.* 2000). The importance of social support may vary by sex. In a study of middle-aged and older patients, impaired social support was associated with poorer outcome of major depression in older men but not older women (George *et al.* 1989; Dew *et al.* 1997).

In general, social support is perceived to be adequate in the elderly, even among clinical samples (Blazer, 1982). Although old social networks thin out, new ones emerge for many people. Most older people believe that they have enough contact with both family and friends as well as endorse the positive relationship that they have with their networks (Cornoni-Huntley *et al.* 1990). Yet when the social network is depleted suddenly, either through loss of a network member (such as a spouse or child) or through a change in the quality of the relationship (such as a dispute within the family), impaired social support may be a most important contributor to late-life depression.

Modifying factors

Some psychological factors may modify the biological and social risks for depression. For example, most older adults who experience a significant physical illness do not become depressed. The psychological risk factors described above may not be present and, therefore, the threshold for the onset of depressive symptoms may be heightened. In addition, older people may possess unique factors that actually protect them from the onset of clinically significant depressive symptoms. Two of these potential factors are described below.

First, the socio-emotional selectivity theory has been proposed to explain differences in the experience of events across the life-cycle (Carstensen *et al.* 2000). The theory focused on the perception by older persons of time left in life rather than past experiences. Younger adults, who have much to learn and relatively long futures, are motivated by pursuit of knowledge, even when this requires emotional well-being to be suppressed. Older adults, in contrast, perceive that they have lived longer than they will live and therefore de-emphasize negative experience and prioritize emotionally meaningful goals. For example, these investigators, in one study, found that negative

emotional experiences (the perception of stressors) declined from young adulthood until around the age of 60 years (Carstensen *et al.* 2000). In addition, periods of highly positive emotional experience were more likely to endure as meaningful among older periods compared to younger adults. In a supporting study, investigators found that age was associated with a self-reported increase in positive affect and a decrease in negative affect (Mroczek & Kolarz, 1998). In other words, compared to younger adults, older adults were more likely to selectively optimize the positive in their social experiences. This in turn could blunt the harsh reality of some of the more negative experiences among the elderly and, therefore, protect against the onset of depressive symptoms.

Second, adults are thought to acquire increased wisdom as they age. Although wisdom is a nebulous concept for most epidemiologists, psychiatrists and psychologists, investigators with the Berlin Aging group have operationalized the concept and studied it in community samples (Baltes & Staudinger, 2000). They defined wisdom as an expert knowledge system concerning the fundamental pragmatics of life, including knowledge and judgment about the meaning and conduct of life and the orchestrating of human development toward excellence while attending conjointly to personal and collective well-being. Five criteria were assessed: rich factual knowledge; rich procedural knowledge (knowing how to assess, for example, the ability to develop strategies for addressing problems); lifespan contextualization (e.g. integrating life experiences); relativism of values and life priorities (e.g. tolerance for differences in society); and recognition and management of uncertainty (accepting that the future cannot be known with certainty and that our ability to assess our sociocultural environment is inherently constrained). The acquisition of wisdom over time would appear to protect the elderly from a spiral down into depression when confronted with a complex of negative experiences.

Of course, there is major variability across older adults in terms of their psychological make-up and their acquisition of characteristics such as wisdom. Yet studies of normal psychological development, as described above, across the lifespan are critical for a better

understanding of the risk and protective factors for late-life depressive symptoms and disorders.

Summary

The biopsychosocial approach to evaluating the origins of late-life depression is heuristically valuable, a continual reminder of the many factors that contribute to the onset and persistence of clinically significant symptoms in late life. Older adults appear to be at greater risk for major depression from some biological causes (see Table 1) yet the frequency of major depression is lower, especially in community samples in Western countries. Although psychological and social risks for depression in the elderly are well documented, there is reason to believe that older adults who are cognitively intact and who do not suffer from significant functional impairment may be protected psychologically due to factors such as socio-emotional selectivity and wisdom and perhaps protected from some social risk factors compared to younger adults.

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DECLARATION OF INTEREST

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