

Mental Health and Aging

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The largest cohort in U.S. history is approaching late life, driving an unprecedented growth in the older adult population. The leading edge of the baby boom generation (born between 1946 and 1964) reached the age of 65 in 2011. By the year 2030, nearly 20% of the U.S. population will be 65 or older, double the size of the older adult population in the year 2000 (Federal Interagency Forum on Aging-Related Statistics [Forum], 2010). Along with this dramatic population increase is projected an increase in the number of older adults with mental health problems (Jeste et al., 1999), resulting from both larger numbers of older adults in the population and an elevated propensity for psychopathology in the baby boom cohort (e.g., Hasin, Goodwin, Stinson & Grant, 2005). Furthermore, the coming generation of older adults is expected to seek mental health treatment at higher rates than past generations (Qualls, Segal, Norman, Niederehe, & Gallagher-Thompson, 2002). These changes bring into sharp focus the importance of investigating the presentation, etiology and treatment of psychopathology in late life.

Psychopathology in late life can best be understood in the context of lifespan development. Developmental psychopathology has been a useful paradigm for studying abnormal behavior situated within a developmental context (Sroufe & Rutter, 1984; see also chapter XX in this volume), but the work within this field has generally focused on early development (Cicchetti & Toth, 2009). In contrast, a large body of literature within the lifespan developmental psychology tradition offers theoretical and empirical explanations for normal development across the entire lifespan and into old age (e.g., Baltes & Baltes, 1990; Carstensen, Isaacowitz, & Charles, 1999), but less often focuses on psychopathology. In this chapter, we adopt the developmental psychopathology perspective to examine abnormal behavior in late life as a function of development across the lifespan.

Several aspects of the developmental psychopathology perspective have been particularly helpful in organizing our thinking within this chapter. Consistent with this

perspective, in which psychopathology is viewed as deviation from normal development (Sroufe & Rutter, 1984; see Chapter XX of this volume), we begin with an overview of normal cognitive and socioemotional development in late life. In addition, within the developmental psychopathology perspective, emphasis is placed on understanding not only the presence of psychopathology at any point in time, but also the trajectory over time and even the absence of psychopathology in a person at risk. In this chapter, we differentiate, where possible, between individuals with early onset of disorder that persists into older adulthood and individuals who experience disorder for the first time in late life, referred to as “late onset.” It should be noted, however, that age of onset is not specified in much of the research on late life psychopathology. There has been relatively little research examining the absence of psychopathology in late life among individuals at risk, including those with early onset disorder who experienced remission. Future research in this area would make an important contribution to our understanding of lifespan developmental psychopathology. The developmental psychopathology approach also highlights the possibility that psychopathology may manifest itself differently over time, a concept that has been termed “heterotypic continuity” (Cicchetti & Toth, 2009). This issue is especially relevant to the study of late life psychopathology since diagnostic rubrics have been based primarily on symptom presentation typically seen earlier in the lifespan. As a result, some forms of psychopathology may be under-detected among older adults. We discuss this issue further in relation to several of the disorders presented below. A final issue to consider is research methodology; both developmental psychopathology and lifespan developmental psychology traditions emphasize the need for longitudinal research before drawing conclusions about age changes. We base our conclusions on longitudinal research wherever possible.

We begin this chapter with a brief overview of biological, cognitive, social and emotional development in late life. We then examine some of the most prominent forms of psychopathology with respect to epidemiology, presentation, etiology and treatment. Where the disorder appears to increase or decrease in prevalence compared to earlier points in the

lifespan, we evaluate possible explanations for these age-related changes. The disorders we cover include anxiety disorders, mood disorders and suicide, schizophrenia, substance use disorders, personality disorders, sleep disorders and dementia. For disorders not included in the current chapter, the interested reader is referred to Segal, Qualls and Smyer (2011).

Normal Development in Late Life

Normal development in late life is characterized by both stability and change in biological, cognitive, social and emotional domains. Biological aging involves changes in the central nervous system and most other organ systems, as well as a concomitant increase in the likelihood of physical illness and disability. Despite these age-related changes, however, most older adults remain independent. Only 4.5% of adults age 65 and older live in skilled nursing facilities (Zarit & Zarit, 2007, p. 13). Age-related slowing of the central nervous system and other neurological changes are associated with normative declines in cognitive abilities characterized as fluid intelligence (Zarit & Zarit, 2007, p. 23). In contrast, crystallized intelligence, which involves fund of knowledge, remains stable or even improves into late life (Zarit & Zarit, 2007, p. 23). Social changes with age include the possibility of bereavement, retirement, caregiving and other forms of role change. Nevertheless, 75% of older men and 43% of older women are married (Zarit & Zarit, 2007, p. 13).

An influential meta-theory that purports to explain adaptation to these age-related changes is the *Selective Optimization and Compensation model* (SOC; Baltes & Baltes, 1990). According to Baltes and Baltes, aging is associated with a narrowing of opportunities, referred to as *selection*. Selection may result from losses, as described above, or may be elective, as when a person chooses to focus on a particular occupation or other objective. Optimization is the process of enhancing existing abilities, such as practicing a skill. Compensation entails use of alternative methods of meeting a goal, such as seeking help or using an assistive device. Individuals age successfully, according to the SOC model, to the extent that they select goals wisely, optimize their preserved abilities and compensate for their losses. The SOC model

provides a framework for understanding some forms of psychopathology in late life, as discussed further below.

Another useful theory for understanding normal development in late life is *Socioemotional Selectivity Theory* (SST; Carstensen, Isaacowitz, & Charles, 1999). According to SST, social goals change across the lifespan. In youth (or whenever time is perceived as expansive), individuals pursue more information-oriented social goals, such as learning about social norms or evaluating oneself in relation to others. In contrast, in late life (or whenever the future is perceived to be foreshortened), the focus is on emotion-oriented social goals, such as regulating one's emotional state through contact with others. Consistent with predictions of this theory, older adults typically have smaller social networks than younger adults, but are more likely to derive emotional satisfaction from them (Carstensen et al.). Older adults also exhibit improved emotion regulation compared to their younger counterparts (Carstensen et al.; Charles, Reynolds, & Gatz, 2001). These aspects of the theory are particularly relevant for the understanding of psychopathology in late life. Against this backdrop of normal aging, we next consider major types of disorders in late life.

Anxiety

Anxiety disorders involve disturbances in affect, physiology, and cognition, and are characterized by avoidance of situations or stimuli that evoke fear or somatic distress when present (Ollendick & Byrd, 2001). Anxiety disorders include generalized anxiety disorder (GAD), simple phobia, social phobia, panic disorder, agoraphobia without panic disorder, obsessive-compulsive disorder (OCD), and post traumatic stress disorder (PTSD). (See Chapter XX of this volume.)

Anxiety disorders tend to be chronic, sometimes lasting years or decades (Barlow, 2002). Nonetheless, both the lifetime and 12-month prevalence of all anxiety disorders drops after age 65 (Gum, King-Kallimanis, & Kahn, 2009). According to Gum and colleagues, the 12-month prevalence of any anxiety disorder is highest among adults aged 18-44 (20.7%),

followed by adults aged 45-64 (18.7%), and drops to 7.0% among adults aged 65 and older.

According to Gum and colleagues (2009), the most common anxiety disorder among people aged 65 and up is specific phobia, with a lifetime prevalence of 6.8%, followed by social phobia (6.3%), generalized anxiety disorder (GAD; 3.3%), panic disorder and post-traumatic stress disorder (2.1% each), and agoraphobia without panic disorder (1.6%). Other researchers have found that GAD is the most common anxiety disorder among older adults (e.g., Beekman et al., 1998). Since anxiety disorders share the common thread of worry, and GAD is characterized by worry, some investigators consider it to be the “basic” anxiety disorder (Roemer, Orsillo, & Barlow, 2002). Because much of the research on late-life anxiety has focused on GAD, we focus on this disorder in the current chapter.

Description and Course

The Diagnostic and Statistical Manual of Mental Disorder, Fourth Edition (DSM-IV-TR; APA, 2000) describes GAD as a disorder characterized by excessive and difficult to control worry lasting for a minimum of 6 months, accompanied by at least three out of six of the following symptoms: fatigue, restlessness or feeling edgy, muscle tension, trouble concentrating or mind going blank, irritability, and disrupted sleep. The proposed set of criteria for GAD in the Diagnostic and Statistical Manual of Mental Disorder, Fifth Edition (DSM V; APA, 2010) retains the core symptom of worry, but reduces the time frame from 6 to 3 months and shifts the focus away from the 6 associated symptoms. According to the proposed criteria, one must exhibit excessive and difficult to control worry, along with either restlessness or muscle tension, and one or more behavioral manifestations of worry to meet criteria for GAD (APA, 2010). These criteria decrease the focus on somatic symptoms in GAD, and may significantly impact the diagnosis of the disorder in late-life. Because somatic symptoms of anxiety may overlap with symptoms of physical illness (Palmer et al., 1997), the proposed DSM-V diagnostic criteria will likely affect the detection of GAD in older adults. If somatic symptoms simply confound the diagnosis of GAD, then the increased emphasis on non-somatic symptoms (i.e., worry) will

simplify the diagnosis of GAD among older adults. However, the proposed DSM-V criteria could possibly result in fewer diagnoses of GAD if older adults with anxiety truly present with more physical manifestations of anxiety than their younger counterparts, as some researchers have suggested (Spar & LaRue, 1990).

Several age-related differences in presentation of GAD are worth noting. The core symptom of GAD is worry. However, worry content may change with age. Wetherell and colleagues (2003) found that some worries (e.g., worries about family) may be normative among older adults. Other worries are shown to differentiate older adults with GAD from those without GAD, including worries about finances, personal health, small matters, and social matters (Wetherell et al., 2003). Additionally, researchers have examined age-related presentations in the six associated diagnostic symptoms of GAD according to DSM-IV. Sleep disturbance and muscle tension seemed to be especially important factors in distinguishing older adults with GAD from those without, while trouble concentrating did not distinguish those with GAD from those with no diagnosis (Wetherell et al., 2003). Though muscle tension is retained as a diagnostic symptom in DSM V, sleep disturbance is not.

GAD is thought to be a chronic disorder (Roemer, Orsillo, & Barlow, 2002). A longitudinal study shows a remission rate of 38% over five years (Yonkers, Dyck, Warshaw, & Keller, 2000). However, of those with either fully or partially remitted GAD, many (27% and 39% respectively) remitted within three years. Remission rates are lower when GAD co-occurs with Major Depression than when GAD is the sole diagnosis (Schoevers et al., 2005).

Etiology

Researchers have attempted to examine the etiology of GAD and other anxiety disorders. Genetic factors, predictability or control over life events, parenting style, and attributional style may make certain individuals more vulnerable to anxiety disorders (Barlow, 2002). However, much of this research focuses on children and younger adults, and little is known about the etiology of GAD or other anxiety disorders in late-life.

Explanation for Age Differences

The reasons for the relatively lower prevalence of GAD in late life compared to earlier in the lifespan are not well understood, but there are several explanations that would be consistent with the empirical evidence. We may observe these decreases in prevalence because older adults are more adept than their younger counterparts at regulating emotions, and can reduce the impact of negative emotions (Carstensen et al., 1999). These strategies may allow older adults to successfully manage anxiety symptoms in late-life. Further, there is evidence that the probability of treatment contact for anxiety disorders cumulatively increases with age (Wang et al., 2005). Thus, many older adults may have been successfully treated by the time they reach late-life.

Certain methodological factors could also account for the lower prevalence of GAD in late life. Measures developed for use with younger adults may miss symptoms of anxiety in older adults. Further, anxiety disorders are often diagnosed with one or more comorbid Axis I disorders (Lenze et al., 2000). Physical disorders can also complicate the identification of anxiety disorders in older adults. Older adults with anxiety disorders have more comorbid physical health problems than those without anxiety disorders (Palmer, Jeste, & Sheikh, 1997). Anxiety symptoms may mimic the signs of physical illness, and conversely, physical illness may mimic the experience of anxiety (Palmer et al., 1997). Thus, the lower prevalence of GAD in late life may result in part from poor detection of it.

Assessment and Treatment

As noted above, differences in the presentation of GAD in late life compared to earlier in the lifespan should be considered when assessing for the disorder in older adults. There are several evidence-based pharmacological and psychosocial treatments available to older adults with GAD. Benzodiazepines were historically used to treat anxiety in older adults. However, antidepressants have become a first line treatment for GAD because of significant risks associated with benzodiazepines (Sheikh & Cassidy, 2000). Of the psychosocial interventions,

relaxation training and cognitive-behavior therapy (CBT) have the most support for use with older adults with GAD (Ayers, Sorrell, Thorp, & Wetherell, 2007).

Conclusions

In sum, GAD and other anxiety disorders are relatively common in late life, but are less prevalent in this age group than earlier in the lifespan. Older adults may simply experience less anxiety in late-life as a result of treatment or more successful emotion regulation strategies. However, prevalence estimates that rely on assessments developed for use with younger adults may underestimate the true number of older adults who meet criteria for these disorders. Moreover, we know little about the etiology and presentation of anxiety in late-life. To answer these questions, there is a need for more longitudinal research on the anxiety disorders.

Mood Disorders

Depression in late-life is a relatively common, yet under-diagnosed disorder, with a wide range of possible physical, emotional, and cognitive consequences (Fiske, Wetherell, & Gatz, 2009). Several types of mood disorders are categorized in the DSM-IV, including major depressive disorder, dysthymic disorder, bipolar disorder, and depressive disorder due to general medical condition. (See Chapter XX, this volume.) An estimated 1 – 3% of the population aged 65 and older currently meets criteria for depression (Cole & Dendukuri, 2004). The 12-month prevalence of major depressive disorder is significantly lower among adults aged 65 and older than in any other adult age group (Hasin, Goodwin, Stinson & Grant, 2005). Depressive symptoms at a clinically significant level of severity that do not meet criteria for any diagnosis have also been a focus of much research. It is estimated that as many as 19% of older adults may have clinically-significant depressive symptoms (Cole & Dendukuri, 2004). This is of serious concern, as minor depression in older men, and major depression in both genders of older adults, have demonstrated an increased risk of death that is not fully explained by suicide (Penninx, et al. 1999). As most research concerning depression in late life relates to either major depressive disorder or severity of depressive symptoms, these will be the focus of

this review. Bipolar disorder is uncommon in late life and will not be discussed in detail in this chapter. However, the relative absence of a disorder can also be informative from a lifespan developmental psychopathology perspective. Although the reasons for the relatively low prevalence of bipolar disorder in late life are not well understood, there is evidence of midlife remission in a substantial proportion of individuals (Cicero, Epler and Scher, 2009), and selective mortality from suicide and other causes has been well documented (Angst, Stassen, Clayton, & Angst, 2002; Jamison, 2000).

Description and Course

Major depressive disorder is characterized by pervasive dysphoria or anhedonia and associated symptoms. Depression presents differently in late life than earlier in the lifespan (Christiansen et al., 1999; Gallo, Anthony & Muthén, 1994). Older adults are less likely than younger individuals to endorse cognitive/affective symptoms, including sadness, worthlessness/guilt, and suicidal ideation (Gallo, Anthony & Muthén, 1994). In contrast, older adults are more likely than younger individuals to endorse somatic symptoms, such as insomnia, fatigue and psychomotor retardation (Christiansen et al., 1999). Cognitive deficits are more often present in depressed older adults, particularly in the case of late-onset disorder.

At least half of older adults with depression developed it for the first time in late life. Brodaty and colleagues (2001) found that 52% of depressed older adults had their first onset after age 60. Similarly, Bruce and colleagues (2002) found that 71% of depressed older adult home care patients developed depression for the first time in late life. Depression is a chronic, episodic disorder. A study examining the course of depression in community-dwelling older adults found that after three years, 48.3% had remitted, while 51.7% had relapsed (Schoevers, et al. 2003).

Etiology

The etiology of depression in late life can be viewed from a developmental diathesis-stress perspective (Gatz, Kasl-Godley, & Karel, 1996). Biological or psychological vulnerabilities

to depression, which vary across the lifespan, may interact with stressors characteristic of late life to precipitate a depressive episode. Biological vulnerability may include genetic risk as well as physiological changes related to either aging or disease, e.g., vascular disease (Dent, et al. 1999). An example of psychological vulnerability is external locus of control (Beekman, de Beurs, van Balkom, Deeg, van Dyck, & van Tilburg, 2000). Risk factor research has identified numerous stressors associated with depression or depressive symptoms in late life, including: physical limitations (Cui, Lyness, Tang, Tu, & Conwell, 2008; Dent, et al. 1999); economic strain (Samuelsson, McCamish-Svensson, Hagberg, Sundström & Dehlin, 2005); death of a partner or other relatives (Vink, et al. 2009); and being a caregiver (reviewed by Fiske, Wetherell, & Gatz, 2009). Lowered cognitive functioning has also been associated with late life depression (Cui et al.), and may be a marker of biological change or a psychological stressor. Risk factors may vary based on age of depression onset. Late onset depression is associated with lower levels of neuroticism, but poorer overall physical health than early onset (Nguyen & Zonderman, 2006; Sneed, Kasen, & Cohen, 2007).

Explanation for Age Differences

As with anxiety disorders, the reasons for the relatively low prevalence of depressive disorders in late life are poorly understood. Increased ability to regulate emotions (Carstensen et al., 1999; Charles, Reynolds, & Gatz, 2001) may play a part in reducing likelihood of depression among older adults. The current clinical diagnostic rubric might not detect depression in older adults (Weiss, Nagel, & Aronson, 1986). This may be due to the greater tendency of older adults to endorse somatic symptoms and the reduced likelihood of older adults endorsing dysphoria. Either dysphoria or anhedonia must be present to diagnose major depressive disorder (DSM-IV-TR, 2000). Since depression is associated with increased risk of death from suicide and other causes, selective mortality may also partially explain reduced rates of depression in late life.

Assessment and Treatment

Since late life depression presents differently from depression earlier in the lifespan, often without endorsement of sadness, assessment can be difficult (Fiske & O'Riley, 2008). The Geriatric Depression Scale (Yesavage et al., 1983) was developed to measure depressive symptoms in older adults without including somatic symptoms that could be confounded with physical illness, although this strategy risks under-detecting purely somatic presentations of depression, which are more common in late life. (For a discussion, see Fiske & O'Riley, 2008).

Both pharmacological and psychosocial treatments have demonstrated efficacy in treating depression in late life. (For a review of pharmacological treatments, see Beyer, 2007.) Psychosocial treatments for older adult depression that meet evidence-based criteria include behavioral therapy, cognitive-behavioral therapy, cognitive bibliotherapy, problem-solving therapy, brief psychodynamic therapy, and life review therapy (see Scogin et al., 2005 for a review). The outcomes for older adults who are treated for depression are similar to those for younger individuals (Pinquart, Duberstein, & Lyness, 2006). However, effective treatment may take more time, especially for late-onset depression. A study by Driscoll and colleagues (2005) found that late-onset recurrent depression took longer to respond to treatment than early-onset depression (an average of 12 weeks versus 8 weeks), but that those with late onset had similar results for remission (47% for late-onset single, 53% for early-onset recurrent) and relapse (19% for late-onset single, 17% for early-onset recurrent). In addition, older adults may cycle out of recurrent depressive episodes more slowly (Kessler, Foster, Webster, & House, 1992). Treatment of depression in late life may also be improved by integrating mental health treatment into primary care (Bruce, et al. 2004; Hunkeler, et al. 2006).

Conclusions

Although subsyndromal symptoms are common in late life, major depression and bipolar disorders are less common in late-life than in mid-life (Fiske, Wetherell, & Gatz, 2009). Although direct evidence is lacking, improved ability to regulate emotions may contribute to a true decline in these disorders with age. Mood disorders may also be under-diagnosed in late life due to

differences in presentation when compared with earlier in the lifespan. The etiology and risk-factors for older adult depression may differ from depression in younger adults, and these differences should be considered when assessing for depression among older adults. Effective pharmacological and psychosocial treatments are available.

Suicide

Any discussion of mood disorders would be incomplete without consideration of suicide, and this is particularly true when the focus is late life mood disorders. Suicide among older adults differs in several important ways from suicide earlier in the lifespan. In 2007, the suicide rate in the U.S. for adults age 65 and older (14.27/100,000) was over 30% higher than the suicide rate for individuals age 64 and younger (10.84/100,000), primarily due to very high suicide rates in older adult men (Xu, Kochanek, Murphy, & Tejada-Vera, 2010). Gender differences in late life may reflect the fact that older adult men are more likely to use firearms than older adult women (Xu, et al.). Additionally, suicide attempts in late life are much more likely to be fatal when compared with younger adults. It is estimated that there are 100 suicide attempts for every death by suicide among young adults (Jacobziner, 1965), whereas there are 4 suicide attempts for every death by suicide among older adults (Lawrence, et al. 2000).

Definitions

Suicide is a self-inflicted death in which there is evidence that the individual intended to take his or her life (Silverman, Berman, Sanddal, O'Carroll, & Joiner, 2007). An individual may also have suicidal ideation, which is having thoughts of ending one's life regardless of whether or not they actually intend to do so (Silverman, et al., 2007). A suicide attempt is "a self-inflicted, potentially injurious behavior with a nonfatal outcome for which there is evidence (either explicit or implicit) of intent to die" (Silverman, et al. 2007; pg. 273). In this chapter, we focus on suicide rather than suicidal ideation or attempt.

Etiology

Several theories of suicidal behavior may be informative with respect to late life suicide.

Joiner's influential *Interpersonal Theory of Suicide* (Joiner, 2005) attempts to explain why suicidal thoughts are common but so few individuals die by suicide. Joiner postulates that there are three factors that put an individual at risk of dying by suicide: thwarted belongingness, perceived burdensomeness, and the acquired capacity to enact lethal self-harm. According to Joiner, interpersonal factors (thwarted belongingness and perceived burdensomeness) contribute to the desire to die by suicide, but are insufficient to enable an individual to enact lethal self-harm. The individual must also have acquired the capacity to enact such behaviors through habituation, such as by practicing or working up to the attempt, attempting suicide in the past, or experiencing (personally or vicariously) painful or provocative experiences. As a result of habituation, fear of pain and other normal, protective barriers to suicidal behavior are reduced. This theory provides a compelling argument for why older adults are at greater risk to die by suicide. Older adults face several threats to their belongingness, such as the death of friends and loved ones as well as retirement and may also feel like a burden to friends and family if disability forces increased reliance on others. Older adults also have a lifetime of experience with pain, and painful conditions are common in older adults which may lead to habituation and, therefore, the acquired capacity, to engage in lethal self-harm behavior, increasing the risk of dying by suicide. In addition, older adults have a longer history of exposure to death and therefore may be less affected by the idea of dying than younger individuals.

Suicidal behavior in older adults may also be understood in the context of the model of Selective Optimization with Compensation presented earlier (Baltes & Baltes, 1990). Suicidal behavior may result from failures in selection, optimization, or compensation. Failure to reach one's target or goal may lead to helplessness and depression. Further, lack of compensation (including an unwillingness or inability to seek help or to redefine goals) may exacerbate the problem and lead to feelings of hopelessness and suicidal ideation. Therefore, identifying and rectifying failures in selection, optimization, and compensation may be an important step in reducing an older adult's risk of suicide.

Risk factor research suggests that having psychopathology (specifically mood and substance abuse disorders) puts older adults at significantly higher risk of suicide (Beautrais, 2002). Prior suicide attempts are also a risk factor for suicide or serious suicide attempt (Beautrais). Chronic illnesses also become more prevalent with age, and certain illnesses and physical conditions, such as cancer, vision impairment and incontinence, may put older adults at greater risk of death by suicide (reviewed by Fiske, O'Riley, & Widoe, 2008). Mild cognitive impairment is also associated with greater risk of late life suicide, but there is little evidence of increased risk associated with dementia, Parkinson's disease, or stroke. Evidence suggests that effects may be partially but not fully explained by depression (reviewed by Fiske, O'Riley & Widoe).

Assessment and Prevention

Several measures have been created to assess suicidal ideation (Heisel & Flett, 2006) or risk in older adults (Edelstein, et al. 2009; Fremouw, McCoy, Tyner, & Musick, 2009). Since late life depression is treatable, there is hope for reducing suicide risk in late life by treating depression in older adults (Bruce, et al. 2004). Since older adult suicide attempts are more likely to be fatal than suicide attempts in younger adults, there are fewer opportunities to intervene following an attempt. Preventing suicide in older adults is further complicated by the fact that older adults do not regularly seek out mental health professionals (Santos & VandenBos, 1982), although there is evidence that the majority of older adults who die by suicide visit a physician within a month of the death (Luoma, Martin, & Pearson, 2002). Therefore, identifying suicidal risk in settings that older adults frequent, such as primary care or through visiting nurses, is important to treat individuals at risk and reduce the likelihood of death by suicide. Programs that integrate treatment for depression within primary care have demonstrated success in reducing suicidal ideation (Bruce, et al. 2004; Hunkeler, et al. 2006).

Conclusions

Suicide rates are very high in late life, particularly among older adult men (Xu, et al.

2010). Several theories offer possible explanations for these age differences, but they remain to be evaluated empirically. Identifying older adults at risk of suicide is difficult, because older adults do not seek out the attention of mental health professionals. However, the primary risk factor for late life suicide is depression, and suicidal older adults do present to primary care. Therefore, a promising approach may be to develop screening tools specifically for older adults and utilize them in primary care and home care settings, where integrated depression treatment can also be offered. Improving the identification of depression and suicide risk in older adults may increase the number of older adults who receive treatment and reduce the number who die by suicide.

Schizophrenia

Schizophrenia is a life-span neurodevelopmental disorder. The prevalence is 1.3% in the population aged 18 to 54 and 0.6% among individuals aged 65 and older (see Jeste & Nasrallah, 2003). Schizophrenia with onset in late life has been the focus of recent research and will be discussed specifically below. (See Chapter XX, this volume.)

Description and Course

Diagnostic criteria (DSM-IV-TR) specify at least two of the following symptoms: delusions; hallucinations; thought disorders manifested in disorganized speech, disorganized or catatonic behavior, or negative symptoms (e.g., blunted affect, loss of motivation, or poverty of speech). Notably, no pathognomic symptom of schizophrenia has been identified, suggesting that the diagnostic category may be heterogeneous, comprised of multiple related but distinct disorders.

Several different trajectories characterize the course of schizophrenia across the lifespan. Most commonly, schizophrenia has an onset in late adolescence or early adulthood, but in a quarter of older adults with schizophrenia, onset occurred later in life (see Jeste & Nasrallah, 2003). Regardless of the age at which the disease is diagnosed, evidence suggests that the disease process begins earlier, as preclinical signs such as cognitive, motor and social

deficits are observed in children, adolescents and adults who later develop schizophrenia (e.g., Walker & Lewine, 1990). The course of schizophrenia is chronic with continuing symptoms for a minority of patients (25-35%), whereas the course is episodic for more than 50% of patients (Jobe & Harrow, 2010). A subgroup of 20-35% of patients functions well after discontinuing anti-psychotic medication (Jobe & Harrow).

Etiology

Leading theories attribute the etiology of schizophrenia to genetic or other biological vulnerability in combination with pre- and perinatal environmental stressors that interfere with normal neurological development (Bearden, Meyer, Loewy, Niendam, & Cannon, 2006). Evidence shows substantial genetic influence (Gottesman & Hanson, 2005). Environmental stressors that have been implicated include birth complications, such as those involving fetal hypoxia (Bearden, et al.) and maternal viral infection during the second trimester (e.g., Barr, Mednick, & Munk-Jørgensen, 1990).

Explanation for Age Differences

There are several possible explanations for the lower prevalence of schizophrenia in late life compared to early adulthood or middle age. Lower rates of schizophrenia in late life may result, in part, from remission of the disease. Although there are varying estimates of the rate of remission, longitudinal research demonstrates convincingly that remission is possible, even among older adults. In a sample of community-dwelling middle-aged and older schizophrenia outpatients, eight percent met stringent criteria for sustained remission (Auslander & Jeste, 2004). Furthermore, the pattern of symptoms of schizophrenia appears to be characterized by stability or a decline in severity across the lifespan (Folsom et al., 2009; Jeste et al., 2003). Older individuals with schizophrenia appear to have better coping skills than their younger counterparts, and well-being and functioning among older adults with early onset schizophrenia has been shown to be stable or even reflect an increased level at older ages (Jeste et al., 2003; Folsom et al., 2009). Similarly, in longitudinal research, cognitive performance has been shown

to remain stable over time in older adult schizophrenia outpatients (Savla et al., 2006). When compared to age-matched normal controls, however, older adults with schizophrenia show greater symptomatology, poorer functioning and lower quality of life (Jobe & Harrow, 2010). In addition, stability in symptoms and functioning is only found among non-institutionalized individuals. Institutionalized older adults with early onset schizophrenia experience more severe symptoms than both younger adults and non-institutionalized older adults as well as declines in cognitive functioning (Folsom et al., 2009; Savla et al., 2006). An alternative explanation for the lower rates of schizophrenia in late life is selective mortality, that is, an increased likelihood for individuals with schizophrenia of dying before reaching old age (Jobe & Harrow).

Late- and Very Late-Onset Schizophrenia

“Late-onset” schizophrenia refers to onset after age 40 and “very late onset” schizophrenia refers to onset after age 60 (Cohen, 1990; Howard et al., 2000). Given the theorized etiology of schizophrenia as a deviation from normal brain development, onset of the disorder in late life would seem difficult to explain. Some investigators (see Howard et al.) have proposed that late-onset schizophrenia is a distinct disorder from early-onset schizophrenia, with differing epidemiology, presentation and risk or protective factors. Evidence, however, is mixed.

Cross-sectional research suggests that symptom presentation is similar for early- vs. late-onset schizophrenia (reviewed by Howard et al., 2000), but this finding is mostly restricted to those younger than 60. For older individuals with very late onset, the expression of symptoms is less severe with more non-specific symptomatology compared to those with early onset (Häfner et al., 1998). Cognitive impairment is a symptom of schizophrenia for both early and late onset, but for those with late onset, the impairment is milder than for those with early onset (Howard et al., 2000). Some investigators have suggested that late onset schizophrenia is better understood as a form of dementia, but longitudinal evidence from two studies suggests that this is not the case (Palmer et al., 2003; Rabins et al., 2003).

Epidemiologic research suggests that risk factors may differ for early- vs. late-onset schizophrenia. Individuals with onset of schizophrenia in later life are less likely to have a family history of schizophrenia and more likely to experience sensory impairments than those with early onset (Howard, Almeida & Raymond, 1994; Howard et al., 2000). Other correlates include being female and being unmarried, but it is unclear whether individuals with late onset schizophrenia have better or worse social, educational, and occupational functioning compared to those with earlier onset schizophrenia (Howard et al., 2000). Thus, additional research is needed to better understand whether early onset and late (or even very late) onset schizophrenia are the same disorder with different manifestations or distinct disorders.

Assessment and Treatment

Several treatments have demonstrated efficacy in the management of schizophrenia in late life. Antipsychotic medications are effective in controlling positive symptoms, as in younger populations (Auslander & Jeste, 2004). Cognitive behavioral social skills training is associated with better social functioning, coping skills and even insight among older adults with schizophrenia (Granholtm et al., 2005). Supported employment has demonstrated similar success in improving outcomes in middle-aged and older adults with schizophrenia as in younger samples (Twamley & Narvaez, 2008).

Conclusions

Based on the findings from the studies reviewed above, the following conclusions can be made. Schizophrenia is rare among older adults, and even rarer is late onset schizophrenia (among older adults). Overall, older adults with late onset schizophrenia have the same rates of cognitive decline as those with early onset. With some exceptions, either stability in symptomatology and functioning or decreases in deficits or impairments are apparent for non-institutionalized individuals with schizophrenia. Evidence is inconclusive with respect to whether late onset schizophrenia represents the same or different disorder than early-onset schizophrenia, but it does not appear to predict dementia, and individuals with late onset

schizophrenia tend to have a better prognosis and quality of life than those with early onset. .

Alcohol Use Disorders

Alcohol use disorders are associated with severe mental and physical health consequences and increased risk of mortality (Thun et al., 1997). These disorders are less common in late life than earlier in the lifespan, with the prevalence of problematic alcohol use among older adults between 1% and 22% for community populations (Johnson, 2000; Oslin, 2004). (See Chapter XX, this volume.) However, several methodological, assessment and etiological factors complicate the study of alcohol use in older age. Despite the fact that excessive drinking declines as individuals enter their 70s and 80s, many older adults still drink more than suggested consumption guidelines, especially those in the cohort born after 1920, suggesting that cohort effects may be an underlying factor when assessing alcohol use across the lifespan (Moos et al., 2009).

Description and Course

To meet DSM-IV criteria for alcohol abuse, an individual must show a maladaptive pattern of substance use, resulting in significant distress or impairment. Alcohol dependence involves continued use despite alcohol-related problems. Diagnostic criteria, which were not developed or validated in older adult populations, may underestimate the prevalence of abuse among older adults (Patterson & Jeste, 1999). For example, diagnostic criteria specify age-dependent consequences (e.g. school or work) that may not be relevant in late life due to age-related role changes (Patterson & Jeste). An alternative method of studying problematic alcohol use is to measure frequency of drinking behaviors.

Studies suggest that the major risk period for alcohol initiation is over by the age of 20 (DeWit, Adlaf, Offord, & Ogburne, 2000), and the odds of lifetime alcohol use disorders drops significantly for each increasing year of age at initiation (Grant & Dawson, 1997). However, a majority of this research has relied heavily on adolescent and young adult samples without long-term longitudinal tracking. There is evidence that some older adults start problem drinking after

age 50, often referred to as late adult onset or “reactive” drinking (Sattar, Petty, & Burke, 2003). Older adults who begin drinking later in life may do so in response to aging stressors like traumatic losses, issues related to retirement, or major illness (Johnson, 2000; Sattar et al., 2003; Oslin, 2004). Regardless of age of onset, alcohol use disorders tend to follow a chronic course (Kerr, Fillmore, & Bostrom, 2002).

Etiology

There are multiple possible causes of problematic alcohol use in late life. Alcohol use disorders arise from an interaction of genetic risk and environmental factors that may vary across the lifespan (Vanyukov & Targer, 2000). Risk factors for late life drinking include pain, sleep difficulties, and depression (Brennan, Schutte, & Moos, 2005; Sattar et al., 2003; Schonfeld & Dupree, 1991). Additionally, lack of social support in late life may lead to the use of alcohol as a negative coping strategy and has been associated with higher chances of relapse (Moos, Finney, & Cronkite, 1990; Jason, Davis, Ferrari, & Bishop, 2001).

Explanations for Age Differences

There are several possible explanations for the reduced prevalence of alcohol use disorders in late life. Longitudinal research shows that excessive drinking behavior declines with age (Moos et al., 2009). Younger alcohol users have been shown to “mature out” of problem drinking well before they reach old age as a consequence of both personality development and role transitions (Littlefield, Sher, & Wood, 2009). Physiological changes may contribute to older adults’ increased sensitivity to alcohol, which may be associated with changes in alcohol use behaviors. For example, lean body mass and total body water to fat ratio decreases with age, resulting in increased serum concentrations of alcohol in the body (Oslin, 2004). Although some older adults may reduce consumption of alcohol as a consequence of physiological changes associated with aging, it should be noted that some older adults who continue lifelong drinking patterns unaware of their age-heightened risk may encounter drinking-related problems for the first time in late life (Merrick et al., 2008). An alternative explanation for reduced rates of alcohol

use disorders in late life is selective mortality. Individuals with early-onset alcohol abuse or dependence die approximately 10 years earlier than age-matched controls (Thun et al., 1997; Fried et al, 1998), and even in late-onset cases, alcohol abuse has been found to be associated with excess mortality, especially in men (Moos, Brennan, & Mertens, 1994; Moore et al., 2006). In addition, problems with detection may contribute to the low apparent rates of alcohol disorders in late life.

Assessment and Treatment

Accurate assessment of alcohol use disorders is critical for the treatment of these disorders among older adults. Unfortunately, under-detection and under-reporting occur in primary care settings, which are often considered the front-line approach to older adult mental health care. D'Amico, Paddock, Burnam and King (2005) found that problem drinkers were less likely to visit general medical providers than non-problem drinkers. Additionally, medical professionals were less likely to ask about alcohol use in persons aged 50 or older (D'Amico et al., 2005).

Several other barriers have been identified in the detection of older adult alcohol misuse (St John et al., 2010). The symptoms of alcohol use are often the same symptoms portrayed under different older adult disorders. Symptoms like falls, accidental injuries, urinary incontinence, and depression are geriatric problems that share common symptom presentations with those seen in alcohol abusers (Johnson, 2000; Sattar et al., 2003; Han, Gfroerer, Colliver, & Penne, 2009). Furthermore, the harmful effects of drinking also exacerbate problems with physical and mental health functioning. Cognitive impairment, in conjunction with or separate from alcohol disorders, may diminish the likelihood of detection, due to self-report difficulties, bias, and symptom misidentification (Blazer & Wu, 2009). In addition, because social patterns change with age (Carstensen, Isaacowitz, & Charles, 1999), many of the social consequences of problem drinking go unnoticed (Sattar et al., 2003). Further complicating this dilemma is the fact that individuals at risk for alcohol problems or binge-drinking are less likely to report overt

stress (Blazer & Wu, 2009). These factors should be considered when assessing for alcohol use problems in an older adult.

Older adults are amenable to treatment, and outcomes are especially beneficial when elder-specific treatments are used (Blow, Walton, Chermack, Mudd, & Brower, 2000; Oslin, 2004). In fact, most research suggests that late-onset drinkers may be more responsive and receptive to treatment than early-onset drinkers (Oslin, Pettinati, & Volpicelli, 2002), especially since early-onset patients may be more likely than late-onset patients to drop out of treatment (Atkinson, Tolson, & Turner 1993). Older adults attain several positive outcomes across a range of measures (e.g. alcohol, emotional, social) after receiving and completing substance treatment. Older adults may also respond well to brief interventions (Oslin, 2004). However, few intervention studies use randomized controlled designs in older adults, making it difficult to suggest which types of treatment interventions are most effective (Sattar et al., 2003; Oslin, 2004).

Conclusions

Cross-sectional and longitudinal research suggests that alcohol use disorders are less prevalent in old age than earlier in the lifespan. Research suggests that these findings may be explained by reduced drinking associated with age-related changes in physiological, personality and social factors as well as selective mortality. However, the lack of consensus on what is considered problem drinking, as well as difficulty detecting and screening for alcohol use disorders in late life, complicate the picture.

Although alcohol use disorders decrease with age, they still present a significant public health challenge. The number of older adults who will meet criteria for substance use disorder is projected to double from 2.8 million to 5.7 million in 2020 (Han et al., 2009). As the baby boom generation gets set to move into old age, researchers will have the unique ability to witness how alcohol use changes in a generation that has historically consumed more alcohol and been more open to psychological treatment than previous generations.

Personality Disorders

Personality disorders (PDs) are defined as pervasive and inflexible patterns of behavior associated with distress or impairment. A more detailed discussion of diagnostic criteria for PDs can be found in Chapter XX of this volume. Although PDs are defined by their longstanding patterns of dysfunctional behavior, some PDs may manifest differently as individuals age. PDs are found among 10% of community-dwelling older adults (Widiger & Seidnitz, 2002), which is slightly lower than the prevalence for younger adults, ranging from 10 to 13% (Weissman, 1993). The DSM-IV-TR describes ten personality disorders, but only four with evident age-related differences will be reviewed in detail. Cross-sectional research suggests that anti-social personality disorder (ASPD) and borderline personality disorder (BPD) are less prevalent in older adults than in younger adults (Segal, Hook, & Coolidge, 2001), whereas schizoid PD and obsessive-compulsive personality disorder (OCPD) appear to be more prevalent in older compared to younger adults (Engels, Duijsens, Haringsma, & van Putten, 2003; Segal, Hook, & Coolidge, 2001). Among individuals aged 65 to 98 years old, ASPD was diagnosed in 0.2 to 0.3%, schizoid PD in 0.9 to 1.9%, and OCPD in 1.6 to 2.5% of older adults (Balsis, Woods, Gleason, & Oltmanns, 2007).

Description and Course

Schizoid PD is characterized by a lack of interest in interpersonal relationships, ASPD by disregard for and violation of the rights of others, BPD by instability in relationships, affect and identity, and OCPD by preoccupation with order (APA, 2000).

Age-related differences in presentation of ASPD and BPD are evident. Older adults with ASPD are more likely to endorse lying or deception than younger adults with ASPD (Balsis, Gleason, Woods, & Oltmanns, 2007). This age difference is consistent with the interpretation that violation of the rights of others (e.g., physical fighting and aggression) requires physical strength and agility (Kroessler, 1990) and may diminish as strength declines with age. Several differences in the presentation of BPD are observed as well. Specifically, older adults with BPD

may have fewer close relationships, thus the pattern of unstable relationships may be less frequently endorsed (Agronin & Maletta, 2000). In addition, older adults are less likely to exhibit impulsive behaviors and are more likely to experience the cognitive symptoms of BPD, such as identity disturbance (Clarkin, Spielman, & Klausner, 1999).

Etiology

Personality disorders can be conceptualized as maladaptive and extreme variants of normal personality traits. As such, they are likely to arise from an interaction of genetic predisposition and early life environment exacerbated by current stressors. The *predisposition hypothesis* (Sadavoy & Leszcz, 1987, as cited in Zweig & Hillman, 1999) suggests that middle aged and older adults with PDs are more susceptible to age-related stressors (e.g., loss, physical illness, forced dependency) than their counterparts. Thus, these individuals with PDs may have a higher diathesis prior to encountering stressors. Encountered stressors may lead to poorer prognosis, increased disability, and mortality.

Explanations for Age Differences

There are a few explanations for the age differences in prevalence of specific PDs. The *maturation hypothesis* posits that certain *immature* personality types (e.g., antisocial, borderline) are more likely to improve with age as impulsivity decreases. On the other hand, *mature* personality disorder types (OCPD, schizoid, and paranoid PDs) are thought to worsen with age as a result of longstanding patterns of behavior, cohort differences (e.g., leading to “miserly” behavior), or mild cognitive impairment (e.g., hoarding in OCPD; Kernberg, 1984; Solomon, 1981, cited in Zweig & Hillman, 1999).

Longitudinal research has yielded findings that are broadly consistent with these explanations. At present, there are several well-constructed and promising longitudinal studies of personality disorders (e.g., McLean Study of Adult Development; Zanarini et al, 2007; Collaborative Longitudinal Personality Disorders Study; Skodol et al., 2005), however; the cohorts are young and yield little information about individuals older than 50 years. Despite this

limitation, there is longitudinal evidence of a decrease in impulsive behaviors over time for both ASPD and BPD. The course of ASPD varies with high mortality rates (23.9%), remission of ASPD (17.6%), and continued engagement in antisocial behavior for almost half of individuals (48.5%, Black et al., 1995). In contrast, BPD often remits with about 75% of patients remitting at 15-year follow-up (Zanarini et al., 2007). Although remission is common, some symptoms of affective instability still remain (Zanarini et al., 2003). Thus, the distress associated with personality disorders is stable across the life span, while the symptom presentation changes (e.g., Zanarini et al., 2007).

Age differences may also be reflective of biased diagnostic criteria (e.g., Balsis et al., 2007) or age differences in the presentation of symptoms. For example, the criteria for schizoid PD may falsely identify as schizoid older adults with social isolation due to disability or decreased functional status. However, an older adult with schizoid PD would have a lifelong history of few or no relationships, rather than age-related reduction in relationships. Interestingly, some of the criteria for OCPD, which is frequently identified in older adults, may be biased towards younger age groups as the criteria focus on behaviors occurring in a work setting (Agronin & Maletta, 2000). Notwithstanding the potential bias, three other criteria were more frequently endorsed by older adults with OCPD: (1) inflexible about moral, ethical, or values issues, (2) unable to discard worthless objects, and (3) assumes a miserly spending style (Balsis et al., 2007).

Additionally, selective mortality may also play a role in the apparent decrease in certain PDs that involve impulsive and potentially harmful behaviors, specifically ASPD and BPD. Individuals with ASPD and BPD are more likely to die unnatural deaths such as suicide or other violent deaths compared to the average population. In a longitudinal study of individuals diagnosed with ASPD, 24% of individuals died after a lengthy follow-up period (16 to 45 years; Black, Baumgard, & Bell, 1995). Individuals with BPD have high rates of completed suicide (about 10% overall, a rate of 50 times that of the general population) and higher rates of deaths

from natural (8%) and premature causes (18.2%; Paris & Zewig-Frank, 2001; Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004).

Assessment and Treatment

Assessment measures used to diagnose personality disorders were created using young adult samples and may be biased when used with older adults (Balsis et al., 2007). In particular, when assessing older adults, it is essential to differentiate behaviors consistent with personality disorders from behavior influenced by medical or neurological disorders (e.g., dementia), role changes (e.g., recently widowed), and other changes in life context (e.g., moving to long-term care facility; Zweig, 2008).

Among older individuals with comorbid PDs and Axis I disorders, the course of treatment may be slowed, complicated, and possibly impeded compared to individuals without Axis II disorders (Gradman, Thompson & Gallagher-Thompson, 1999). As there are few treatment studies to begin with, it is not clear whether certain types of psychotherapy are more beneficial than others for individuals with PDs. In recent years, Dialectical Behavior Therapy (DBT) has gained support in treating young adults with BPD (for a brief review, see Lieb et al., 2004), but has not been widely examined as treatment for personality psychopathology among older adults. DBT was found to be effective in conjunction with medication to treat depression in older adults with comorbid PDs (Lynch et al. 2007).

Conclusions

Personality disorders reflect heterotypic continuity across the lifespan, in that certain symptoms (e.g., impulsivity) vary across the life span, while there are some stable, underlying personality characteristics. The stability in the underlying distress of personality disorders is evident as older adults with PDs experience more distress and are more difficult to treat compared to older adults without PDs. Contrary to the assumptions embedded in the diagnostic system, longitudinal studies suggest that BPD is more likely than not to remit across the life span. Additionally, individuals with BPD and ASPD may experience selective mortality, which is

reflected by some cross-sectional research. Some researchers suggest that schizoid and obsessive compulsive PDs are more prevalent in late life. More longitudinal research is needed to elucidate the course of PDs across the lifespan and into old age.

Insomnia

A third of our life is spent sleeping, yet the important function that sleep plays in our daily life goes largely unnoticed until our sleep becomes disturbed in some way. Older adults suffering from sleep difficulties are at greater risk for falling because of fatigue, slower reaction times, and impaired daytime functioning associated with sleep impairment (Ancoli-Israel, Ayalono, & Salzman, 2008). Sleep disturbance has even been linked to elevated risk of mortality (Hardy & Studenski, 2008).

The most common sleep problem clinicians will encounter is insomnia. Insomnia is a diagnostic component of many other psychological and psychiatric disorders, and furthermore, diagnoses such as mood and anxiety disorders have been linked to persistent poor sleep quality in longitudinal studies of older adults (Morgan, Healey, & Healey, 1989; Pigeon & Perlis, 2007). While insomnia can occur at any point in the life span, rates increase by 200% around the 7th and 8th decade, and insomnia is present in 41% of women age 80 to 89 (Lichstein et al., 2006). In a large-scale study of adults age 65 and older, up to 34% reported symptoms of insomnia, and half of women reported such symptomatology (Foley et al., 1995).

Description and Course

The DSM-IV criteria for primary insomnia requires difficulty sleeping for a minimum of one month, with some type of daytime dysfunction not due to drug or medication use or other psychological, medical, or sleep disorder (APA, 2000). In contrast, the *International Classification of Sleep Disorders*, a specialized system for classifying sleep disorders, specifies diagnostic criteria for insomnia that require difficulty sleeping when provided ample opportunity to do so and some type of daytime dysfunction (American Academy of Sleep Medicine, 2005). Sub-types of insomnia are classified according to the time of the night when the problem occurs.

Sleep onset insomnia is broadly defined by difficulty falling asleep, whereas maintenance insomnia is defined by an inability to stay asleep or by waking earlier than desired (Lichstein, Stone, Nau, McCrae, & Payne, 2006). Insomnia is often comorbid with other psychological and physical health problems such as depression, anxiety, diabetes, and heart disease (Ancoli-Israel, Ayalon, & Salzman, 2008; Cooke & Ancoli-Israel, 2006; Lichstein et al., 2006).

Etiology

The predominant conceptual model of insomnia is Spielman's behavioral model (Spielman, Caruso, & Glovinsky, 1987). Predisposing factors, which may be biological, psychological or social, are thought to increase vulnerability to insomnia and interact with precipitating factors, such as acute stressors, in producing the acute phase of insomnia. Behavioral responses, such as extending sleep opportunity and using alcohol to induce sleep tend to perpetuate the sleep difficulties, leading to chronic insomnia. Predisposing, precipitating and perpetuating factors may vary in frequency across the lifespan.

Explanations for Age Differences

Changes in sleep continuity that occur with normal aging may predispose older adults to insomnia. Sleep latency, or the time it takes one to fall asleep once in bed, increases with age, becoming most pronounced after the age of 65 (for a meta-analysis, see Ohayon, Carskadon, Guilleminault, & Vitello, 2004). Total sleep time decreases by approximately 10 minutes for each decade of the life span (Ohayon et al., 2004). Additionally, time awake after sleep onset (WASO) also increases approximately 10 minutes each decade beginning at age 30 (Ohayon et al., 2004).

When examining sleep timing across the lifespan, a normative shift to earlier sleep onset is seen among older adults (Ancoli-Israel, 2005) and demonstrated by examining biochemical markers of sleep (Yoon et al., 2003). This phase shift has been associated with maintenance insomnia, such that older adults often attempt to remain awake until "normal" bed times, which are later than their biologically driven sleep onset time. In tandem, the sleep period is shortened

causing daytime fatigue. Compared to earlier life-span periods, older adults have more flexible schedules allowing for “make up” sleep via daytime naps, which serve to further complicate the advance sleep phase shift/insomnia relation (Yoon et al., 2003).

Assessment and Treatment

Assessment of insomnia often begins with complaints of trouble falling asleep, early awakening, or daytime fatigue at least three times per week, which can be documented using a sleep diary. Confirmation of sleep and wake activity levels can be done most cost efficiently by using an actigraph, which is worn on the wrist and measures motion. Effective interventions for insomnia include pharmacological treatment with hypnotics, which are highly prescribed among older adults and are helpful in the short term, melatonin and sedating antidepressants, for which evidence is mixed (Lichstein et al., 2006). Though hypnotics are most commonly prescribed for insomnia, there is longitudinal evidence that with long term use, these drugs are ineffective and can exacerbate insomnia when patients cease taking them (Hohagen et al., 1993). Behavioral techniques that have demonstrated efficacy in treating insomnia, separately or as part of a multicomponent treatment, include stimulus control, which strengthens the association of the bed with sleep, and sleep restriction, which limits time in bed (Perlis, Jungquist, Smith, & Posner, 2005). Recent research has demonstrated that the most efficacious long-term treatment of insomnia is early combined treatment with behavioral and pharmacological treatment with a phasing-out of pharmacological aids (Morin et al., 2009).

Conclusions

Older adults are at elevated risk for insomnia (Ancoli-Israel, 2005). Insomnia has been linked to daytime sleepiness which, if frequent enough, has been linked to serious consequences, including increased mortality rates (Morgan, Healey, & Healey, 1989). Because treatment of sleep problems has been related to alleviation of comorbid symptomology, effective sleep treatments, such as behavioral treatments for insomnia, provide hope not only for those suffering from sleep disorders, but also those suffering from other diagnoses.

Dementia

At present, approximately 24 million people worldwide suffer from some form of dementia (Ferri et al., 2005). Prevalence of dementia increases with age; rates for adults in their 60s are approximately 1.5%, with rates increasing to 15 – 25% for adults in their 80s. Although the underlying mechanisms are not fully understood, dementia is more common in females than in males (Shumaker et al., 2003). Life expectancy decreases with severity of dementia, especially for those older than 85 years, among whom dementia predicts approximately 30% of all deaths in men and 50% of all deaths in women (Aevarsson, Svanborg, & Skoog, 1998). Alzheimer's disease (AD) is the most common type of dementia, accounting for approximately 60 – 80% of all dementia (U.S. Department of Health and Human Services, 2008). Vascular dementia is the second most common form and has been estimated at 8 to 30% of all dementia cases (DiCarlo et al., 2002). Dementia with Lewy Bodies has recently been recognized as a common form of dementia; it is estimated to account for up to 30% of all dementia cases (Williams, Xiong, Morris, and Galvin, 2006). Because AD accounts for the majority of dementia cases, we will focus solely on AD.

Description and Course

According to the DSM-IV, the essential features of dementia are memory impairments and progressive cognitive declines in one or more areas of intellectual functioning: aphasia (e.g., language deterioration), apraxia (e.g., impaired ability to compete motor activities), agnosia (e.g., failure to identify objects), or a disturbance in executive functioning (e.g., ability to think abstractly, execute a plan). Such declines in cognitive functioning must be severe enough to interfere with occupational and social functioning. To qualify for a diagnosis of Alzheimer's disease, the deficits cannot be due to any preexisting central nervous system disorder or any other disorder that is known to cause dementia.

Age of onset of AD is typically after age 65 and cognitive deterioration is slow and progressive. Survival time ranges from approximately 3 years (if diagnosed in 80s or 90s) to 7-

10 years (if diagnosed in 60s and 70s; Cosentino, Scarmeas, Albert, & Stern, 2006). Defining features of AD are the presence of amyloid plaques, neurofibrillary tangles, and brain atrophy.

Etiology

AD involves the progressive deterioration of the cerebral cortex and the hippocampus which is expressed clinically through impairment in memory and other cognitive abilities. Cognitive reserve capacity, conceptualized as the brain's ability to sustain the effects of injury or disease, varies across individuals and is thought to influence the extent and timing of the clinical expression of AD-related neuropathology (Borenstein, Copenhaver & Mortimer, 2006). As a result, it may be particularly helpful to distinguish between risk factors for neuropathology and risk factors for the clinical expression of AD.

Among risk factors thought to influence AD neuropathology directly, genetic vulnerability is the most prominent (Gatz et al., 2006). The e4 allele of the apolipoprotein (APOE) gene is well established as a risk factor for late-onset AD (most cases of AD are late-onset; Farrer et al., 1997). APOE4 is a susceptibility gene; that is, carrying one or two APOE e4 alleles does not mean an adult will, without a doubt, develop AD. Other risk factors include head trauma and cardiovascular disease (reviewed by Borenstein et al., 2006).

In contrast, evidence suggests that premorbid intelligence and education may be risk factors associated with the clinical expression of AD (Borenstein et al., 2006). Whalley and colleagues (Whalley, Deary, Appleton, & Starr, 2004) have demonstrated that premorbid, childhood IQ acts as a protective factor against cognitive decline. More specifically, children with lower mental ability and fewer years of education tend to show greatest cognitive decline. The association between education level and AD is well established (DiCarlo et al., 2002). Gatz and colleagues (2007) reported that within adult twin pairs discordant for dementia, the twin with dementia had fewer years of education than the non-demented twin partner, and the effect was independent of genetic influences.

Some investigators suggest the "use it or lose it" hypothesis with respect to cognitive

reserve (i.e., neurological resilience), proposing that mental activity and stimulation (e.g., doing crossword puzzles, staying socially engaged) may actually increase synaptic density in the brain and thereby enhance cognitive reserve (Orrell & Sahakian, 1995). The hypothesis is that those with greater cognitive reserves experience fewer cognitive deficits during the aging process (Whalley, Deary, Appleton, & Starr, 2004), but impact on risk of dementia remains to be fully evaluated.

Explanations for Age Differences

AD is a progressive neurodegenerative disease. As such, risk increases directly with advancing age. In addition, age-related conditions such as hypertension, high cholesterol, and cardiovascular disease may play a role in AD through causing damage to the vascular system. Furthermore, an overarching theme in the literature is the influence of cognitive reserve on the onset and progression of dementia (Borenstein et al., 2006). Premorbid intelligence is an indicator of cognitive reserve. Cognitive reserve may even have an influence on cognition after a diagnosis of AD has been made (Starr & Lonie, 2008).

Assessment and Treatment

The assessment of Alzheimer's disease currently involves neuropsychological testing to evaluate memory and cognitive impairments, as well as a somatic examination and neuroimaging to rule out alternative explanations for the impairment. Autopsy studies validate that this method can be highly accurate (e.g., Gatz et al., 2006). Advances in neuroimaging technology, which have recently made it possible to view AD-related neuropathology (amyloid plaques), have made it possible to evaluate factors that affect the clinical expression of AD independent of the neuropathology (Roe et al., 2010). Although this type of neuroimaging may also be helpful in diagnosing AD, diagnostic accuracy is improved by including measures associated with the clinical expression of the disease (Roe et al.).

There are no published interventions for the primary prevention of dementia (Stephan & Brayne, 2008), and there are no available treatments to slow or reverse the progression of brain

deterioration in AD. However, the FDA has approved several drugs, which ameliorate symptoms for up to 12 months (Massoud & Gauthier, 2010). The acetyl cholinesterase inhibitors (AChE), such as donepezil (brand name Aricept), have been shown to improve cognition and functioning in mild to moderate AD, whereas the N-methyl-d-aspartate receptor antagonist (NMDA) memantine (brand name Namenda) has demonstrated similar outcomes in mild to severely impaired patients (Massoud & Gauthier). At present, over 90 clinical trials are investigating experimental therapies on slowing the progression of AD (Alzheimer's Association, 2010).

Although no preventive treatments exist, when examining protective factors, education is consistently found to buffer against age of onset and progression of dementia (DiCarlo et al., 2002). Moreover, clinicians typically focus on preventive treatments of cardiovascular disease, such as controlling modifiable risk factors (e.g., cholesterol levels, blood pressure, and diabetes; Rockwood, 2002). Prevention of such chronic conditions can be achieved through regular exercise, maintaining weight, and lowering blood pressure and cholesterol levels. Further, because amyloid plaques and neurofibrillary tangles are the hallmark of AD, drug trials aimed at influencing the development of these substances are underway.

Conclusions

Alzheimer's disease is a progressive neurodegenerative disorder that affects almost exclusively older adults. Highly accurate assessment and diagnosis is currently possible, and new methods are on the horizon. Currently available treatments reduce symptom severity and improve functioning.

Psychopathology in Late Life

There is evidence of both similarity and difference in psychopathology in late life relative to earlier ages. Contrary to popular stereotype, late life is associated with lower prevalence of many types of psychopathology, including anxiety disorders, major depressive disorder, bipolar disorder, schizophrenia, alcohol use disorders and some types of personality disorders. There is

longitudinal evidence of attenuation of symptom severity or even remission for most of these disorders. These outcomes are consistent with the improved emotion regulation that has been documented in late life. It is also possible, however, that lower rates of disorder in late life may be a methodological artifact rather than reflecting a true age-related decline. Symptom presentation of many of the disorders differs in older adults compared to younger adults, such that diagnostic rubrics established to describe disorders in younger populations may not capture these disorders when they appear in older populations. In some cases, symptoms may attenuate slightly, such that diagnostic criteria are no longer met, but remain problematic for the patient. For example, sub-threshold depressive and anxiety symptoms are common in late life. Further, selective mortality may explain some of the apparent decline in prevalence of some of these disorders (bipolar, major depressive disorder, substance use disorders, schizophrenia). More research, including longitudinal designs, will be needed to tease apart these alternative explanations.

In contrast to the disorders that appear to decline in prevalence with age, there are several disorders that increase in prevalence with age, including dementia, sleep disorders, select personality disorders and suicide. Biological changes associated with aging, which are associated with increased likelihood of executive dysfunction and other cognitive deficits, may explain not only dementia but also other disorders that are linked to cognitive functioning, such as obsessive compulsive personality disorder. Age-related changes in the circadian rhythm have been implicated in increased rates of insomnia. Of note, however, is that biological aging is not always associated with increases in disorder; e.g., changes in metabolism with age can lead to reductions in problem drinking. In addition, behavioral and environmental factors may contribute to increased prevalence of certain disorders in late life. For example, schedule flexibility in old age and sleep-related behaviors may perpetuate insomnia; habituation to painful or provocative experiences may increase risk of high lethality suicidal behavior; and social role changes may precipitate change in alcohol use.

The assessment of psychopathology in older adults can be challenging due to differences in symptom presentation and comorbid physical illnesses. Age-specific instruments are available for some but not all disorders. Many of the same treatments that have demonstrated efficacy in adult populations have also been shown to work in older adults, and several treatments have been developed specifically for older adults. Nonetheless, outcome research within this age group is scarce.

Increasingly, longitudinal research has begun to reshape our understanding of psychopathology in late life. Additional longitudinal research is needed to elucidate the trajectories of psychopathology across the lifespan. Future research focused on older adults who do not manifest psychopathology in spite of risk factors could be particularly helpful in uncovering protective factors. Finally, assessment and treatment outcome research with a focus on older adults is needed. Considering the impending expansion of the older adult population, this type of research could not be more timely.

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