Chapter 1

Introduction:
Suicide Over the Life Cycle—Risk Factors and Life-Span Development

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This book examines what is known about risk factors, assessment, and treatment of suicidal behavior at different stages of the life cycle. Chapters in this volume will approach suicide from many other vantage points than development, but in constructing a conceptual framework for the multiaxial causation of suicide, developmental stage certainly needs to be one of the organizing axes.

One of the most basic facts about suicide in the United States is that its risk increases as a function of age (see Figure 1). Completed suicide is extremely rare in children under the age of 12; becomes more common after puberty, with its incidence increasing in each of the adolescent years; and reaches a peak among youth at age 23. However, the highest rates of suicide are among elderly men (Shaffer et al. 1988). Although secular changes over the past 30 years have resulted in a tripling of the rate of suicide among young adults, 80-year-olds are still twice as likely to commit suicide as 20-year-olds. Why?

In examining the ways in which adult development may affect suicide risk, univariate answers will not be possible because, throughout the adult life span, there are many fluctuating and competing risk and protective factors that affect suicide. Usually, suicide results from the unlikely convergence of multiple predisposing and immediate risk factors, and these risk factors must come together in
the absence of multiple protective factors (Blumenthal 1988). People may move in and out of suicidal crises at different points over their lifetime as a result of disruptions in the homeostasis between these risk and protective factors. However, little knowledge exists to clarify whether suicide is the same phenomenon in childhood as it is in old age or whether it has a different meaning and set of risk factors across the life span. In part, our lack of understanding can be attributed to the dearth of prospective, longitudinal research on this public health problem. The chapters in this volume, using a cross-sectional approach, strongly suggest that suicidal behavior throughout the life cycle is on a continuum: similar risk factors appear to operate across the various stages of the life span but their contributory weights differ. Correspondingly, particular protective factors appear to impact differently at various stages of development.

Additionally, temporal trends—including age, period, and cohort effects and their interactions—are known to impact on the epidemiology of medical and psychiatric disorders. Klerman and Weissman (1989) described how these effects appear to influence suicide rates, particularly in shifting higher rates in recent years to young people.
Age effects are observed when the occurrence or frequency of an illness varies with age. Period effects occur when the rates of a syndrome or disease vary with the time period, usually months or years (e.g., the effects of unemployment on the suicide rates). Cohort effects refer to changes in the rate of illness among persons who share some temporal experience (usually the year or decade of their birth), and these effects are often sustained throughout the cohort’s lifetime. Additionally, age-period interactions can occur when the period effect changes with age-related vulnerability, observed in the increase of substance abuse among young people in recent years (Klerman and Weissman 1989; O’Malley et al. 1984). These temporal effects may help explain the changes in suicide statistics over the past several decades, particularly the increased suicide rates for youth. Higher rates of depression, bipolar disorder, suicide, and substance abuse for the baby boom generation have also paralleled important temporal, social, and economic events, including changes in family structure, shifts in gender roles, age of entrance into the work force, age of marriage, increased access to lethal weapons, and the effects of increased media exposure on violence (Klerman and Weissman 1989).

Additionally, the size of a birth cohort may “shape its destiny” (Klerman and Weissman 1989). Such a model posits that a large birth cohort may result in increased competition for scarce resources, resulting in higher unemployment, lower earnings, and decreased access to educational opportunities. These factors may help explain the higher rates of depression and suicide in the baby boom cohort (Holinger et al. 1987; Klerman and Weissman 1989). Cohorts born since World War II have been healthier and were raised during a time of economic prosperity. Nonetheless, there has still been a rise in the rates of suicide, depression, conduct disorder, substance abuse, and homicide in these groups. Yet the explanations encompassed by the effects described above are not sufficient to explain why particular individuals end their life by suicide. In all likelihood, these environmental influences interact with individual genetic and biologic vulnerability (Klerman and Weissman 1989).

In developing a complete model for suicide across the life cycle, other chapters in this volume will discuss many contributory risk factors, relatively unaffected by developmental changes, that this introductory chapter will not address. On the one hand, genes, race, and gender do not change throughout the life span. On the other hand, many effects of historical and secular changes (e.g., ever-increasing handgun availability in the United States and the protective effects of reducing carbon monoxide content of domestic coal gas in England) are not predictably affected by a developmental model.

In addition, there are two important protective factors for suicide
that are outside the scope of this introduction. First, the availability of active clinical and social intervention is relatively independent of development from age 15 to 85, although for certain age groups (children, adolescents, and the elderly) there are often barriers to obtaining treatment (e.g., lack of financial resources, access to transportation, and knowledge of appropriate resources). Second, there is hope (Beck et al. 1985). *Hope, like love,* is a most important word ignored by psychoanalysts, social scientists, and biologic psychiatrists alike. We know that “hopelessness” can identify up to 91% of future completed suicides (Beck et al. 1985; Weishaar and Beck, this volume), but we know little of hope’s determinants, operational definition, and biologic correlates, or even why hope should be so important to suicide prevention. In the future, study of the development and biology of hope and the modification of hopelessness throughout the life span merits our attention.

In organizing risk factors for suicide, it is helpful to assign them to five overlapping domains, as shown in Figure 2 (Blumenthal 1988). This chapter will examine how four of these domains—psychosocial milieu, biologic vulnerability, psychiatric disorder, and personality—

![Overlap model for suicide risk (five domains). This figure is adapted with permission from Blumenthal and Kupfer 1986.](image-url)
are affected by adult development. How the fifth domain, genetic vulnerability, changes over the adult life span and interacts with environmental influences still remains uncharted territory. However, studies have demonstrated that for both adult and adolescent suicide attempters and completers, there are important genetic and familial influences (Kety, this volume; Pfeffer 1989).

**Psychosocial Milieu**

Stressful life events, depression, physical illness, and loss, even when they occur together, rarely result in a completed suicide if the social support system is vigorously maintained (Havens 1965). Thus social support, or as Alvin Kahn (this volume) poetically reminds us, the fact that “other lives have twined themselves with mine,” is perhaps the single most important protective factor (aside from the appropriate treatment of associated psychiatric disorders) in suicide prevention. Throughout Erikson’s (1963) model of life-span development, “the human personality, in principle, develops according to steps predetermined in the growing person’s readiness to be driven toward, to be aware of, and to interact with a widening social radius” (p. 270). As adults mature, their social network usually becomes more resilient. In counterpoint, it is at the time when everyone in the environment of a potentially suicidal person seems to be moving away that the risk of completed suicide may peak. It is during the individuation and leaving home period, ages 15 to 25, and again in old age that individuals are at particular risk of losing important loves and caretakers faster than they can be replaced.

It is also in adolescence and again in old age when cultural permission for suicide is highest. The fluid identity of adolescence promotes susceptibility to “contagious” and “romantic” suicide (Gould, this volume). Erikson (1963) discussed how role confusion during this stage of development may result in an apparent loss of identity, resulting in over-identification with heroes, cliques, or crowds. This mechanism of a young person projecting his or her own “diffused ego image on another and by seeing it thus reflected and gradually clarified” may underlie imitative or cluster suicides: the phenomenon whereby one youngster’s suicide in a school or community triggers subsequent suicides in other vulnerable youth.

As individuals grow old, membership, both literally and metaphorically, in Hemlock societies becomes increasingly culturally syncronic. Indeed, among the very elderly, as the mythic death of Socrates and the actual death of Sigmund Freud illustrate, self-willed suicide can be an act of reflective choice rather than of unreasoned impulse.
Additionally, another important risk factor, medical illness, appears to have a differential impact on suicidality across the life cycle. Medical illness is associated with as many as 50% of adult completed suicides (Mackenzie and Popkin, this volume) and contributes more prominently in late life. Few studies of completed suicide in children and adolescents have been undertaken, but evidence suggests that medical illness does not play as significant a role. Juvenile diabetes and epilepsy combined with the use of phenobarbital for its treatment have been associated with some suicides in adolescents (Brent and Kolko, this volume). Shafi et al. (1988) found that 48% of the adolescent suicide victims in their study had a history of some form of physical disorder such as asthma, allergies, seizures, or congenital birth defects. However, this finding was also true of the control group and therefore was not statistically significant. In contrast, suicides in the elderly are often associated with the presence of medical illness, particularly cancer, musculoskeletal disorders, and gastrointestinal illnesses. One medical illness that appears to bridge risk between adolescence and adulthood is acquired immuno deficiency syndrome (AIDS), a disease that affects young adults more frequently than older people. A recent report found a 36 times greater incidence of suicide among AIDS victims than in the general population (Marzuk et al. 1988). People with AIDS have a high incidence of depression and cognitive impairments associated with AIDS-related dementia. Importantly, AIDS patients may suffer the humiliation of having an illness that is not well accepted by our society, and the protective buffer of cohesive social supports may be absent for many AIDS victims.

How can the differences in the importance of specific risk factors for suicide across the life cycle be explained? One hypothesis suggests that the impact of a particular risk factor such as presence of a medical illness depends on the co-occurrence of other risk factors for suicide and the extent to which protective factors buffer the risk (Blumenthal 1988). For example, the risk factor, medical illness, may have less impact in childhood and adolescence because medically ill youngsters generally have cohesive social supports, including concerned parents and health care professionals. Hope for future improvement is also greater. Additionally, the incidence of associated psychiatric disorders such as depression is lower in this age group than in adulthood. In the elderly, however, medical illness may contribute significantly to risk because of the convergence of multiple risk factors, including decreased social supports, more interpersonal losses, cognitive impairments associated with aging, the use of certain medications affecting mood and judgment, hopelessness about the future, and the loss of dignity that may accompany growing older or having a terminal illness.
BIOLOGIC VULNERABILITY

Biology governs development. As discussed earlier, completed suicide is a rare phenomenon in childhood and early adolescence, but increases dramatically in late adolescence and young adulthood, with the preponderance of suicidal deaths occurring among the elderly. These differences in rates of suicide across the life span may, in part, be attributable to biologic factors. Biology drives the child willy-nilly to the cognitive and executive skills whereby adolescent suicide can be effective. The increasing cognitive capacity of adolescents to appreciate the future and to develop and carry out a plan may also play an important role in the sharp increase of successful suicides as adolescents mature. Interestingly, young adolescents with an IQ greater than 130 have a higher rate of suicide (Shaffer 1974). Therefore, intellectual precocity may override the potentially protective effects of cognitive immaturity. Additionally, developmentally, adolescence is a time of tremendous biologic and psychological change for young people. It is also a time when the risk factors of access to drugs, handguns, and alcohol are introduced, when certain psychiatric disorders such as depression may have their onset, and when the chances of humiliation and sexual abuse peak. Risk is magnified further because protective cognitive strategies about how to control such dangers are still at a minimum and impulsive, risk-taking behaviors are more common. Then, 40 years later, biology relentlessly inflicts the loss of sight, mobility, and autonomy, which enhances hopelessness and thereby suicide. In old age, where the suicide rate is almost two times greater than the general population, cognitive capacities may decline because of impairments associated with medical illness, Alzheimer’s disease, and depression. At the end of the life span, biologic aging of the brain also increases the likelihood of dementia and organic psychosis, important suicide risk factors that impair judgment, and impulsivity, and may produce concurrent depression.

PSYCHIATRIC DISORDER

Very few people take their lives without concomitant psychiatric disorder, and the prevalence of these illnesses change dramatically across the life span (Black and Winokur, this volume). Studies of both adult and adolescent completed suicides reveal that more than 90% of suicide victims suffered from one or more psychiatric illness (Barraclough et al. 1974; Dorpat and Ripley 1960; Hagnell and Rorsman 1980; Robins 1981; Shafii et al. 1988; Shaffer et al. 1988). There are four broad psychiatric disorders that put individuals at particular risk for suicide: affective disorders, conduct disorders, schizophrenia, and
organic mental disorders (Black and Winokur, this volume). Recent studies suggest that panic disorder patients also have an increased risk of suicidal behavior (Weissman et al. 1989). The comorbidity of these syndromes with particular personality traits (including impulsivity and aggressivity) and with substance abuse appears to increase risk across the life cycle (Blumenthal, this volume). Adolescents who kill themselves tend to manifest depression and conduct disorders where the affective component is often only recognized in retrospect (Brent and Kolko, this volume). In a general population study of suicide, Rich et al. (1986) found that the psychiatric diagnosis associated with suicide changed with age. Under the age of 30, suicides were more frequently associated with antisocial personality disorder and substance abuse. In persons over the age of 30, suicides were more frequently associated with affective disorders. Between ages 20 and 30, schizophrenic patients and patients with bipolar affective disorder are perhaps the highest risk group for suicide; between ages 30 and 50, suicide peaks among the affective disorders. It is in old age, with its heightened risk of both organic brain damage and psychotic affective disorder, that the risk of suicide is at its peak (see Osgood and Thielman, this volume).

However, as Weinberger (1987) and Adams and Lyons (1982) have underscored, the four disorders can be in part a reflection of developmental stage. If one examines the dominant psychopathology that accompanies the onset of Huntington's chorea, Wilson's disease, and metachromatic leukodystrophy, there is a tendency for the result to be conduct disorder if genetic penetrance first occurs between ages 10 and 20, schizophreniform disorders if onset is between ages 20 and 30, major depressive disorder if onset is between ages 30 and 50, and dementia if after age 50. In other words, psychiatric disorder per se may be more important than subtype in the genesis of suicide.

Although it is easy to understand why the pathophysiology of dementia is associated with advanced age, our understanding of the transformation of conduct disorder to schizophreniform-like illnesses to affective disorder is speculative. However, as Weinberger (1987) suggested in his review, there is evidence for actual brain development until age 30, not only in terms of increasing complexity of synaptic and dendritic development but also in terms of increasing myelination (Yakovlev and LeCours 1967). There are also, as yet, poorly understood changes in the activity of the major neurotransmitters over the adult life span. For example, brain serotonin metabolism, thought to be implicated in suicide risk and related to increased impulsivity and aggressivity (Mann et al. 1986; Winchel et al., this volume) is believed to change in predictable fashion during adult life (Wong et al. 1984). The parkinsonian symptoms produced by halo-
peridol are different at age 18 than at age 65. Additionally, differences across the life span in biologic rhythms, including sleep and menstrual cycle function, may impact on psychological state and on the expression of psychiatric disorder. Thus the brain changes in adult life and so may its neurobiologic contributions to suicide risk.

**PERSONALITY**

The degree to which personality changes over the life span is debatable. On the one hand, there is the theoretical evidence offered by popular writers on adult development (e.g., Erikson 1963; Sheehy 1976), and there are the obvious differences in personality that make it so hard for parents and grandparents to identify with adolescents and vice versa.

In *Childhood and Society*, Erikson (1963) described the tasks that must be negotiated during the eight stages of human development and delineated the essential strengths that successful resolution of these crises imparts at each life stage:

- basic trust versus basic mistrust: drive and hope
- autonomy versus shame and doubt: self-control and willpower
- initiative versus guilt: direction and purpose
- industry versus inferiority: method and competence
- identity versus role confusion: devotion and fidelity
- intimacy versus isolation: affiliation and love
- generativity versus stagnation: production and care
- ego integrity versus despair: renunciation and wisdom

Hypothetically, according to this developmental theory, failure to negotiate the challenge of each stage successfully may render the individual more vulnerable to suicidal behavior. Conversely, successful resolution of these passages may result in the establishment of enduring personality strengths and individual resiliency.

On the other hand, as investigators like McCrae and Costa (1984) have shown, efforts to prove such popular theories of adult development or even to document the lawful stages of Eriksonian development have not been easy. Since the truth probably lies somewhere between these competing viewpoints, it makes sense to consider the interactions between adult development and personality that might affect suicide risk over the life span.

To the extent that maturation decreases impulsivity and allows increasing tolerance and mastery of anger without an individual turning it violently against the self, suicide risk will diminish. Studies of the elderly without organic brain damage suggest that, with advanced age, hostility *and* impulsivity *and* depression may all appear
muted (Neugarten 1977). With development from adolescence to mid-life comes increasing patience and the capacity to tolerate both hostile and sad feelings. Even the self-defeating, passive aggression of an adolescent can evolve into selfless but self-enhancing altruism in mid-life.

This protective effect of maturity is enhanced by the fact that one’s sense of personal control is probably greatest between the ages of 30 and 50 (Neugarten 1977). However, with more advanced age, the sense of personal controllability decreases with the onset of medical illnesses, the awareness of mortality, and the loss of significant others, perhaps canceling the diminution in risk resulting from the declining morbidity of personality disorders.

It also behooves us to pay attention to how personality development across the life span can strengthen social networks. It is the very personality variables that attenuate and mitigate depression, impulsivity, and hostility that also enhance potentially supportive new relationships. As has been suggested elsewhere (Vaillant 1977), ego strength can be defined by one’s dominant choice of defense mechanisms. In adolescence, the defenses of projection, hypochondriasis, schizoid fantasy, acting out, and turning against the self (i.e., passive aggression and masochism) are common. Although each of these defenses serves to bind conflict, these defenses repel other people. Over time, however, these mechanisms may be replaced by suppression (stoicism), altruism (empathy), sublimation, humor, and anticipation (planful rehearsal of affect). Such mechanisms become increasingly more common as adolescents mature.

For example, at 30 Beethoven was an angry, impulsive, and depressed man who, in the face of chronic illness (deafness) and the resulting loss of control, wrote that he was very close to suicide. Had young Beethoven, as an isolated, dysthmic musician killed himself, his psychological autopsy would have made perfect sense. In late mid-life, Beethoven’s chronic illness (i.e., his deafness) was still more severe. But over time both this irascible musician’s social network and his ego’s coping skills had become ever stronger. When totally deaf, he put to music Schiller’s Ode to Joy (e.g., “Be embraced all ye millions, with a kiss for all the world”), and there was little evidence that Beethoven was still suicidal.

Yet as stated in the introduction, biology, psychiatric disorder, social supports, and personality are interlocking domains. Organic brain damage, from the ravages of advanced age, drives the maturation of ego defenses in the reverse direction, increases personality disorder, shatters social supports, and increases suicidal risk.

The three facets of personality that are most closely associated with increased risk of suicide are hostility, impulsivity, and depres-
sion, with its associated retroflexed rage (Goldsmith et al., this volume). Turning against the self is an ego defense mechanism often associated with the personality of suicidal individuals and has been described as an underlying mechanism of self-destruction (Freud 1917). Additionally, shame and guilt, essential components of humiliation, are important emotions in precipitating suicidal behavior.

The simplest exogenous way to increase all of these personality risk factors—depression, hostility, impulsivity, and shame—is by alcohol and hypnotic substance abuse. The prevalence of alcoholism and hypnotic drug dependence increases steadily from age 15 until about 45 and then declines. The risk of suicide from alcoholism also goes up with advancing age. However, this correlation probably has more to do with the natural history of the disorder of alcoholism than it does with development per se. As alcoholism progresses, it destroys three factors known to protect against suicide. By means of guilty and angry outbursts, chronic alcoholism destroys social supports. Progressive alcoholism destroys brain cells and intellectual function. It also ravages health, bringing with it a high incidence of medical complications that increase risk for suicide (Flavin et al., this volume). Finally, chronic alcoholism undermines personal control. Indeed, as alcoholism progresses, social networks are so diminished and self-efficacy and self-esteem so impaired that clinicians often mistakenly blame the resulting secondary affective disorder for causing the alcohol abuse and eventual suicide.

Young suicidal adults, especially young adults from chaotic homes, have hostility, impulsivity, and depression as their most salient personality traits (Shafii et al. 1988). But why? Is the reason why a disruptive childhood is associated with an increased risk of future suicide due to the lack of stable internalized object relations? Is early object loss a predisposing risk factor to suicide, as Adam suggests in his chapter? Or is the chapter by Seymour Kety correct? Is suicide the result of the penetrance of a genetic curse? Is the individual’s disturbed childhood and early object loss simply one more reflection that his or her biologic relatives suffered themselves from suicide, alcoholism, affective disorder, and antisocial personality? We must await adequate cross-fostering studies to separate the effect of genes from environment in suicide risk before we know the answer to this developmental riddle.

We wondered at the beginning of this chapter why the rates of suicide increase with age. One explanation for higher rates of completed suicide across the life span may be related to the increased contribution of the major risk factors. The incidence of affective disorders, alcoholism, substance abuse, loss of social supports, humiliating life experiences, and hopelessness appear to increase with age.
Additionally, genetic and biologic factors may play a greater role later in life as environmental influences trigger genetic expression of vulnerability for suicidal behavior. The incidence of major risk factors for suicide, including depression and substance abuse, has been increasing since World War II. In fact, changing environmental factors (e.g., geographic mobility with its loss of attachments, social anomie, increasing urbanization, and changes in family structure) interacting with genetic liability probably have played a role in shifting in recent years the increased rates of both depression and suicide to younger age groups (Klerman and Weissman 1989). The chapters that follow address what is known about risk factors, assessment, and treatment strategies for suicidal patients throughout the life span. It is the task of future research to delineate the relative contributions of risk factors for suicide at different stages of the life cycle and to specify further gene-environment interactions in determining suicide risk. The chapters in this volume review our best knowledge about suicide across the life span.

In summary, the interaction between predisposing risk factors, immediate risk factors, protective factors, and precipitants is complex. The study of adult development from ages 15 to 85 is only one of the possible ways of understanding the complex and intricate interrelationships that can lead to that irrevocable, but fortunately rare, interaction in which a completed suicide occurs.

REFERENCES


