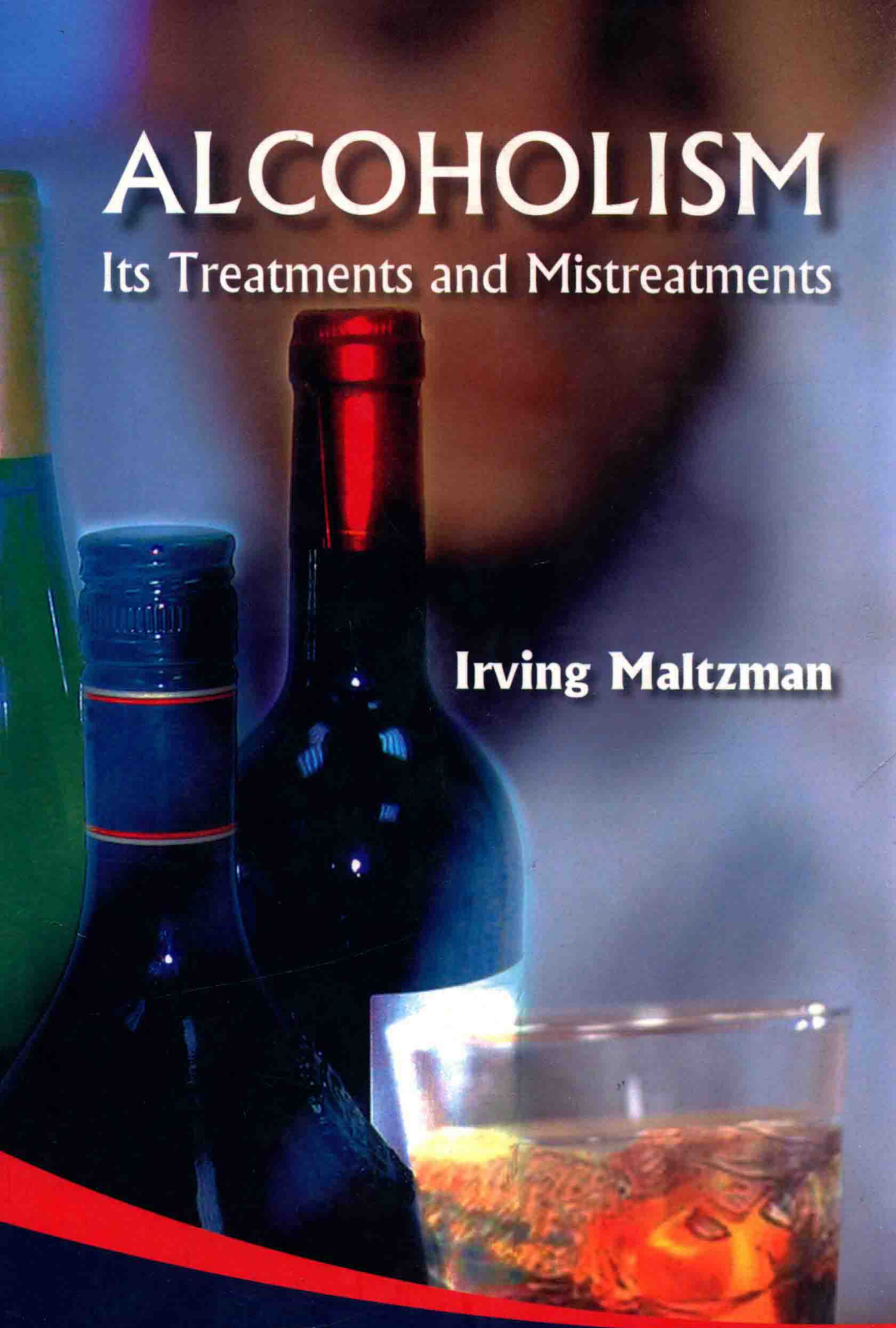
ALCOHOLISM

Its Treatments and Mistreatments



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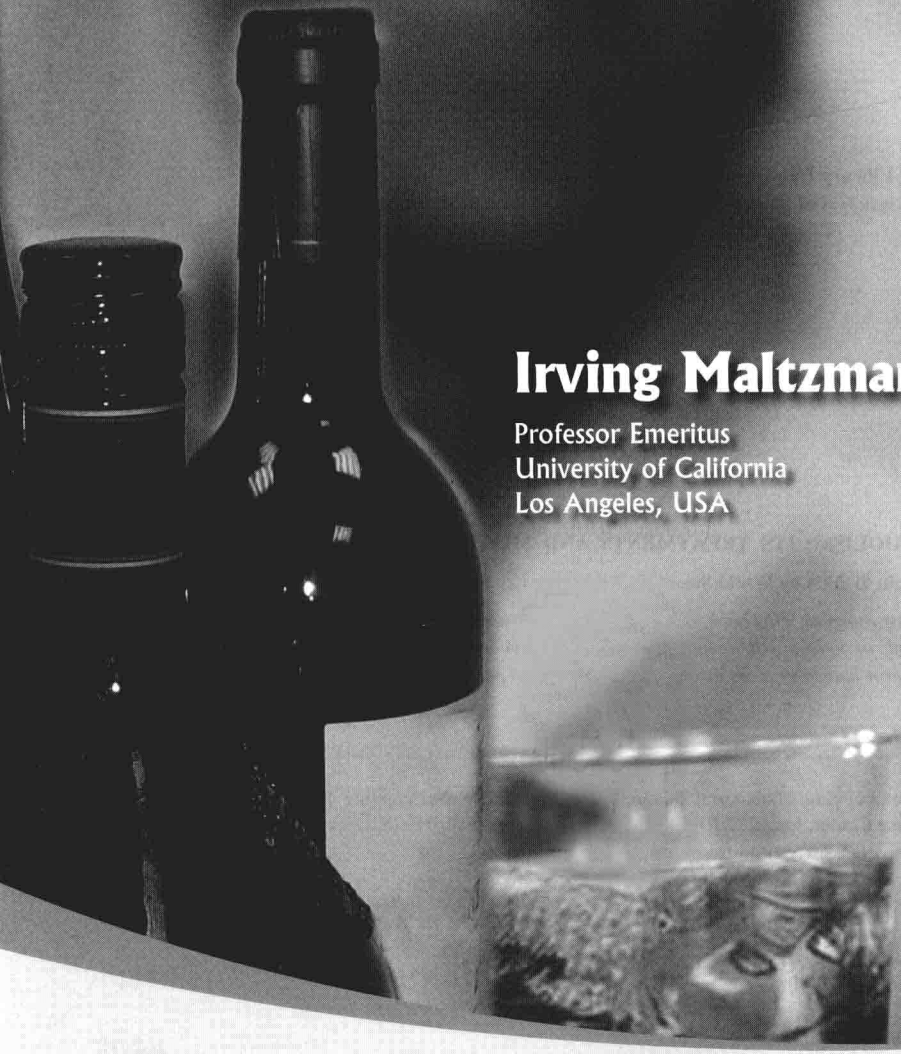

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Contents

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A Biosociobehavioral Disease Conception of Alcoholism

Miller and Hester (2003), in the introductory chapter of their *Handbook of Alcoholism Treatment Approaches: Effective Alternatives* (3rd ed.), present various "conceptual models" or interpretations regarding the characteristics and etiology of alcohol problems:

> Experts in the field ascribe alcohol problems and alcoholism to a bewildering array of causes: inherent biochemical abnormalities (Blum & Payne, 1991; Milam & Ketcham, 1981), genetic influence (Begleiter & Kissin, 1995), conflictual emotions (Denzin, 1993), irrational cognitions (Ellis & Velten, 1992), social learning processes (Orford, 1985; Peele, 1985), family pathology (Curtis, 1999; Steiner, 1971; Steinglass, Bennett, Wolin, & Reiss, 1987), sociocultural influences (Cahalan, 1987; Edwards *et al.*, 1994; Holder, 1998), self-regulation failure (Baumeister, Heatherton, & Tice, 1994), and personal choice (Fingarette, 1988). Given such disagreement about the essence and causes of alcohol problems, it is little wonder that there has been such confusion about how best to treat people who have them. [p. 2]

The above evaluation is confusing and misleading. There is evidence of multiple causes or risk factors in the environment, the host, and their dialectical interactions reported in many studies of alcoholism and its causes, characteristics, and treatments (see Maltzman, 2000). Some

"experts" named by Miller and Hester (2003) are not experts in the field of alcoholism (Baumeister *et al.*, 1994), while others are merely anointed fabulists who agree with Miller and Hester (Fingarette, 1988). There is no disagreement among neuroscientists, behavioral neuroscientists, biomedical scientists, or behaviorists concerning the "essence" of alcoholism, just as there is no disagreement about the essence of cancer, malaria, or other diseases. There is no disagreement that it is a scientifically meaningless question — it is a meaningless question, and therefore not asked, because any solution to the question would not be falsifiable. As far as "causes" are concerned, multiple dialectically interacting risk factors are generally involved in the etiology of diseases; and alcoholism is no exception.

The above introduction by Miller and Hester (2003) sets the stage for the presentation of a variety of outdated or misrepresented models, or interpretations, of alcoholism. The models — briefly presented along with their purported implications for treatment and prevention — include moral, temperance, spiritual, dispositional disease, biological, educational, characteriological, conditioning, social learning, general systems, sociocultural, and public health ones. None of these models or interpretations as described by Miller and Hester properly define "disease", nor do they reflect current analyses of a disease conception of alcoholism (Maltzman, 1991, 1994, 1998, 2000). The purpose of the present chapter is to present such an analysis of "disease", i.e. a disease conception of alcoholism and some of its implications.

Two characteristics are common to all diseases: (1) each disease is a syndrome, defined as a lawful pattern of recurring observable signs and symptoms (Igoe,1979). A disease does not cause the observable signs and symptoms, but rather the disease *is* the lawful pattern of observable signs and symptoms; and (2) the syndrome is judged by experts to be a significant deviation from an accepted standard of health. The first characteristic is an empirical matter; while the second is a value judgment and normative in nature, and therefore may vary with the culture and time of the experts' judgment.

A known etiology and pathophysiology are unnecessary for the classification of a condition as a disease (Cohen, 1961); for example, pulmonary tuberculosis and malaria were recognized as diseases for centuries prior to knowledge of their etiology and pathophysiology. The

conception of alcoholism as a disease is not a mere mental construct, an arbitrary decision to label a condition a disease for political or psychological reasons. Alcoholism is a disease entity that exists because the observable symptoms, although differing widely in appearance, are lawfully related. They are a consequence of biological adaptations and damage produced by alcohol and its effects on the nervous, neuroendocrine, immune, and digestive systems. Alcoholism is not defined by the amount of alcohol consumed: although one cannot become an alcoholic without consuming alcohol, the correlation between the amount of alcohol consumed and its negative consequences — alcohol problems — is not high (Drummond, 1990). Lack of a strong relationship may be the consequence of a variety of individual differences in the biology of the host, including changes in metabolism (Mendelson, 1964), brain damage following chronic heavy drinking (e.g. Cala, 1987; Oscar-Berman & Hutner, 1993; Parsons, 1998; Tarter, 1975), and differences in the social environment interacting with behavior and biology (Higley *et al.*, 1991). For example, an alcoholic who is independently wealthy and does not work for a living, or a college professor with tenure and teaching and research assistants, will probably manifest fewer negative social consequences and fewer alcohol problems than a cashier at the checkout stand of a supermarket who is closely supervised and whose occupation provides quantitative indices of productivity. Nevertheless, there are profound changes in general health and well-being as well as personality as a consequence of the damage produced by alcohol to the nervous, neuroendocrine, and immune systems. It is a disease of the whole person.

The assertion that alcoholism is on a continuum with normal drinking, and therefore obeys the normal laws of social learning (Marlatt, 1979; Miller, 2001; Goldman *et al.*, 1999b), is false. "Laws" of social learning are falsified by critical behavioral studies of alcoholics (Hodgson *et al.*, 1979) and by the adoption studies of Goodwin and his colleagues (Goodwin *et al.*, 1973, 1974). Hodgson *et al.* (1979) showed that a priming drink of alcohol in the morning produced a satiation effect on alcohol consumption in the afternoon and decreased the alcohol consumption of moderately dependent alcoholics; in contrast, a priming drink produced an appetizer effect in severely dependent alcoholics, increasing their later alcohol consumption. A qualitative difference in priming effects was

found, rather than a continuum as necessarily implied by a social learning continuity interpretation.

A social learning account of the signs and symptoms of alcoholism lacks verisimilitude. Another recurring myth promoted by revisionists (e.g. Barlow & Durand, 1995, 2002, 2005; Fingarette, 1988; Mendelson & Mello, 1985; Peele *et al.*, 2000) is that the conception of a lawful symptom progression is based on a study of only 98 members of Alcoholics Anonymous (AA) (Jellinek, 1946). Jellinek's (1952) later study of 2000 members of AA and the more-than-a-dozen replications of the kind of results obtained by Jellinek are ignored by revisionists (see Maltzman, 2000). Replications include both male and female participants who were not AA members (Pokorny & Kanas, 1980), a general population sample (Nelson, Little, Heath, & Kessler, 1996; Piazza & Wise, 1992), and a study of Finnish men (Park & Whitehead, 1973). Core symptoms progress in the same order, regardless of culture, gender, or social status; symptoms may progress at different rates in different cultures and as a function of age and gender, but the order of the core symptoms is essentially the same. Blackouts and denial occur before morning drinks and delirium tremens, whether a person is Finnish or North American, female or male.

Variability in characteristics among alcoholics and in the risks of becoming alcoholic are not peculiar to alcoholism. Variability in etiology is a common characteristic of diseases. Variability probably receives more attention in the case of alcoholism than other diseases because a necessary condition, alcohol consumption, is readily open to study. Necessary causal conditions are not ordinarily as readily apparent in other diseases, requiring far greater intensive research to determine necessary causes. A natural experiment described by Evans (1993) provides an unusual opportunity to examine the variability that occurs in infectious diseases. He reported an incident where the hepatitis B virus contaminated the yellow fever vaccination given in similar fashion to more than 5000 soldiers. Only 20% of the men developed jaundice, a symptom of hepatitis. The appearance of clinical symptoms varied between 60 days and 154 days in soldiers who developed hepatitis. Unknown sources of variability in resistance of the soldiers' immune system were presumably responsible for the enormous variability in the occurrence and appearance of disease symptoms.

If alcoholism meets the criteria for classification as a disease, as evidence and critical analyses show beyond reasonable doubt, then greater effort must be made to study the problem as a disease. Greater research effort is needed to elucidate the basic pathophysiology of the diseased person and the individual's interaction with the social environment. Also essential is the careful study of individual differences as a function of gender, medical history, ethnicity, age, social environment, and culture, as well as of the differences within each larger group manifested in the development of biological dysfunctions and behavioral and social symptoms. A recent international symposium summarized the progress being made in the neurobiology of alcoholism and recovery (Crews *et al.*, 2005). A research emphasis on the biosociobehavioral dysfunctions underlying the development of alcoholism and the biosociobehavioral changes accompanying its successful treatment and aftercare would be time, effort, and money far better spent than wasting it on mind-dust research such as the study of expectancy as a cause of alcoholism and its treatment outcome. A verbal report of an expectancy or its definition by responses to a series of questionnaire items is an effect, not a cause, of alcohol consumption and learning. These and related issues concerning expectancy will be discussed in greater detail in Chapter 4.

Revisionists ignore the evidence that alcohol is a powerful drug causing damage to cells, organs, and entire systems when consumed excessively. Much of the damage is unseen and unrecognized. What constitutes excessive consumption will vary both among individuals (in terms of general health, social context, gender, etc.) and for the same individual (at different times, between meals, etc.) (Eckardt *et al.*, 1998). For social drinkers generally, 2 drinks/day for men and 1 drink/day for women yield the lowest risk for morbidity and mortality compared to abstinence. This generalization is based on the results of a large prospective interview study of approximately 44 000 participants representative of persons aged 40 years or older at baseline and reassessed approximately 6 years later (Lio *et al.*, 2000). It is in accord with the recommendations of the United States Dietary Guideline Committee (US Department of Agriculture, 1995) and other health committees.

Alcoholism must be investigated as the biosociobehavioral disease that it is rather than merely reciting the mantra of "biopsychosocial"

disorder. More than a functional analysis of the discriminated operant of elbow bending is needed. Alcoholics have a higher morbidity, poorer quality of life, and shorter life expectancy than nonalcoholics. They suffer from a higher incidence of liver disease, cardiovascular disease, neuropathy, pancreatitis, cancer, infectious diseases, and structural and functional brain damage (10th Special Report to U.S. Congress on Alcohol and Health, 2000). Important negative social consequences include lost hours of work, disruption of family and social life, and increased medical expenses for the individual and society. Psychopathology is not exempt from the litany of problems; alcoholics as compared to nonalcoholics suffer from a higher incidence of depression, suicide, and anxiety. They do harm not only to themselves, but also to family, friends, and society at large, as the result of automobile crashes and other transportation/industrial accidents. Let us not forget the alcoholism-related 1989 Exxon Valdez oil spill accident, which resulted in an estimated US$2 billion worth of damage to the Alaskan environment. Its costs are still rising due to medical complications produced by the toxic effects of the petroleum to which hundreds of cleanup workers were exposed. A federal judgment more recently awarded US$6.75 billion to the plaintiffs, including US$4.5 billion in punitive damages and approximately US$2.25 billion in interest to the thousands of people who had made their living in the site damaged by the spill (*Los Angeles Times*, January 29, 2004).

Numerous important research problems arise following the adoption of a disease conception of alcoholism. They are foreign to a cognitive social learning theory conception of alcoholism as a bad habit. Damage must be assessed in the biological systems of the human agent: the nervous, neuroendocrine, digestive, and immune systems. Changes within each system produced by alcohol consumption must be studied at different levels, from the subcellular to the behavioral, social, and cultural. Changes in the interactions among systems produced by alcohol consumption must also be examined in detail and in relation to their interaction with behavior and the social environment. We assume there are multiple qualitative as well as quantitative changes within each system and in its interactions, varying from social drinking to chronic alcoholism, cirrhosis of the liver, and Korsakoff's syndrome.

A disease conception of alcoholism implies that the medical history of the mother before, during, and after pregnancy must be examined for alcohol and other drug use, viral and bacterial infections, unusual aspects of the delivery, and diet during the prenatal and postnatal periods. Social support, stress, and conflict within the family must be assessed (Huizink *et al.*, 2004; Myslobodsky, 2004), as well as the family history of alcohol and other drug use. Ethnic, racial, gender, and cultural differences must be studied, and not just between large groups such as Asians and European-Americans: examination of specific national, cultural, and ethnic differences is also necessary. Differences among Korean, Chinese, and Japanese men and women must be considered, as well as differences between Mexican-Americans and other residents in the USA originating from South and Central America. In addition, differences within as well as between religious, national, and cultural groups need to be studied; for example, Italian vs. Irish Catholics, Ashkenazi vs. Sephardic vs. Oriental Jews, etc. Effects of cultural traditions, familism, and acculturation of various immigrant groups must be studied (Hillhouse & Fiorentine, 2001; Nielsen, 2001; Straussner, 2001).

There must be greater sensitivity to cultural differences in treatment. Beneficial interactions between counselor and patient may in part be due to a therapeutic alliance that is affected by biological changes produced when a patient is treated by a member of the same ethnic or religious group. For example, Spinrad (1993) found a significant interaction between ethnic group, type of training, and compliance with a disulfiram regime. Neurohumoral states may be changed; serotonin, endorphin, oxytocin, and vasopressin levels may be increased when there is a therapeutic alliance. Basic biological factors are operating that may have important consequences on the course of treatment. Biobehavioral studies need to be conducted during the treatment process as well as before and after treatment.

Alcohol and the Brain

There is increasing brain damage from social drinking to chronic alcoholism, interacting with many variables including age and drinking

pattern. Korsakoff's syndrome involves additional damage (Brokate *et al.*, 2003; Crews, 1999; Crews *et al.*, 2005; Oscar-Berman *et al.*, 2004; Parsons, 1998). Brain damage is not the sudden end result of alcoholism. Alcohol's effects on the brain are insidious, continuous, and destructive, especially on the developing brain (Tapert *et al.*, 2002). Other serious consequences are more varied, sometimes depending upon the vulnerability of the host's immune system at the time. There are several different routes to alcohol's damage to the brain. Some are the direct toxic effects of alcohol and its metabolites on brain cells. Other sources of damage are indirect, by way of alcohol's effects on the neuroendocrine system via its effects on the hypothalamic-pituitary-adrenal (HPA) axis and its release of corticosteroids, and on the immune system's release of inflammatory cytokines.

Alcohol's deleterious effects on the nervous system and other biological systems are part of a spectrum of nonobvious negative consequences that include changes in mood, affect, and personality. For ethical reasons, experimental demonstration of these effects, including the increased brain damage produced by withdrawal, have been limited to infrahuman animal models (Paula-Barbosa *et al.*, 1993; Phillips & Cragg, 1984). Quantitative analyses by Paula-Barborsa *et al.*

> showed a significant reduction in brain cells in the hippocampus in alcohol fed rats as compared to the matched nonalcohol fed control animals. Magnitude of the effect was related to the length of alcohol treatment. Animals in the withdrawal groups showed significantly greater neuronal loss than the alcohol fed rats who did not suffer withdrawal from alcohol. Earlier research by the same group using the same experimental design examined the medial prefrontal cortex and found a significant loss of brain cells in that region as a function of duration of alcohol treatment with additional loss of cell density following withdrawal. [Maltzman, 2000, p. 63]

Clinical studies of alcoholics leave no doubt concerning the extensive brain damage caused by chronic heavy alcohol consumption (Moselhy *et al.*, 2001; Oscar-Berman & Hutner, 1993). Additional damage in alcoholics is caused by repeated withdrawal from alcohol (Glenn *et al.*, 1988; Tapert *et al.*, 2002).

According to Marlatt (1979),

> All drinking behavior, from social drinking to alcohol abuse, is assumed to be governed by similar principles of learning and reinforcement. As such, it is assumed that there is no crucial difference that distinguishes the social drinker and the problem drinker, other than the amount of alcohol consumed. [p. 324f]

Similar ill-conceived sentiments are expressed by others (Fingarette, 1988; Goldman *et al.*, 1999b; Szasz, 1972). Human experimental and clinical research with alcoholic and control populations as well as infrahuman experimental research since the middle of the 20th century have accumulated an extensive body of evidence contradicting Marlatt's and other revisionists' notion that alcoholism is nothing more than a bad habit. Most importantly, Marlatt and other revisionists ignore the dialectical interaction between alcohol, the brain, and behavior. Jellinek (1960), in his classic work on the disease conception of alcoholism, reviewed all relevant theories of alcoholism at the time, including learning and social interpretations. He noted a hypothesis formulated by Lemere (1956) concerning the etiology of loss of control, a pathognomic sign and symptom of alcoholism, in terms of brain pathology. Jellinek's discussion highlights the need for long-term prospective studies differentiating between predisposing risk factors present prior to the initiation of heavy alcohol consumption and the damage produced following its initiation (Schuckit, 1998; Tarter *et al.*, 1993).

Grohman and Fals-Stewart (2004) reported the results of a study of a new, relatively brief assessment battery that accurately discriminated neuropsychological deficits in patients receiving treatment for substance abuse. Results showed that more than one third of the patients were suffering from such deficits. It is essential that the administration of such neuropsychological assessments becomes a matter of course in treatment facilities, along with a standardized clinical diagnosis based on the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) categories. Such information is necessary for the individualization of treatment and for the development of shared databases permitting research that evaluates the status of neuropsychological function before, during, and after treatment.

The Saga of Phineas Gage

Advances in neuroscience and technology have provided evidence supporting the conceptualizations of half a century ago. Damage to the prefrontal cortex (PC) caused by alcohol may be a determinant of loss of control, denial, and the socially irresponsible behavior characteristic of many alcoholics. Pre-existing dysfunctions in the PC may predispose a person to alcoholism. Subgroups of individuals characterized by an early or adult onset of alcoholism accompanied by sociopathy may suffer the consequences of structural and/or functional damage in the ventromedial (VM) region of the PC.

A remarkable natural experiment in the mid-19th century contributed to the research and theorizing concerning the kind of decision making influenced by the VMPC (Damasio, 1994; Macmillan, 2002; Stuss *et al.*, 1992). In 1848, Phineas Gage — a diligent, hardworking head of a construction gang laying railroad tracks — suffered a terrible accident. An explosion blew a steel tamping bar through his skull. However, it did not kill him. The damage was largely limited to the VMPC. Reports suggested that there was no apparent change in his intellectual ability. His verbal facility and ability to calculate and reason abstractly seemed the same as before.

> On the other hand, he had become irreverent and capricious. His respect for the social conventions by which he once abided had vanished. His abundant profanity offended those around him. Perhaps most troubling, he had taken leave of his sense of responsibility. He could not be trusted to honor his commitments. His employers had deemed him "the most efficient and capable" man in their "employ" but now had to dismiss him. In the words of his physician, "the equilibrium or balance, so to speak, between his intellectual faculty and animal propensities" had been destroyed. [Damasio *et al.*, 1994, p. 110]

Gage apparently became a late-onset sociopath, amoral, unreliable, and untrustworthy (see Macmillan, 2002, for a detailed history and its fabulist embroidery).

Research by Damasio (1994) as well as by Bechara and colleagues (Bechara, 2004; Bechara & Damasio, 2002; Bechara *et al.*, 1998, 2001,

2002) have provided striking clinical and experimental evidence for the role of the VMPC in regulating social and antisocial behavior, including substance dependence. An experimental gambling task has been devised by Bechara *et al.* (2001) that differentiates between individuals with antisocial personality disorders, those with alcohol and other drug dependencies, and controls.

Participants in the gambling task are provided with a cash allowance to gamble with four decks of cards. Drawing any card from decks A and B results in winning US$1.00; drawing a card from decks C and D wins US$0.50. However, participants lose US$2.50 for every 10 cards drawn from decks A and B, whereas they win US$2.50 for every 10 cards drawn from decks C and D. In addition, half of every 10 cards from decks A and B lose US$1.50–US$3.50 as well as win US$1.00/card. Thus, participants lose US$12.50 and win US$10.00 for every 10 cards played from decks A and B. In contrast, 5 of every 10 cards played from decks C and D lose US$0.25–US$0.75. Participants lose US$2.50 but win US$5.00 for every 10 cards played from decks C and D.

Bechara *et al.* (2001) used the gambling task to study the performance of three groups: individuals with lesions to the VMPC; individuals with a substance dependence (SD), either alcohol, methamphetamine, or cocaine; and a group of normal control subjects. The patient groups, VMPC and SD individuals, were chosen for study because they tend to show similar kinds of behavior:

> (1) they often deny, or they are not aware, that they have a problem, (2) when faced with the choice to pursue a course of action that brings in immediate reward, at the risk of incurring future negative consequences, including the loss of reputation, job, home, and family, they choose the immediate reward and ignore the future consequences. . . . [N]europsychological and functional neuroimaging data suggest that a decision-making impairment linked to a dysfunctional VMPC cortex may be at the core of addiction to substances. [Bechara *et al.*, 2001, p. 376]

Additional measures including assessments of psychopathy, depression, and anxiety were obtained to ensure that evidence of decision-making impairment in the SD patients was not confounded with comorbidity.

A number of demographic measures were also used, such as age, gender, education, years of abuse of drug of choice, years of abstinence, cycles of relapse and return to treatment, and years of gainful employment. A prediction index was calculated to estimate the severity of decision-making impairment: "abstinence (in days) divided by the number of years of abuse, times the number of returns to treatment, multiplied by a factor of employment" (Bechara *et al.*, 2001, p. 377). Participants in the SD group met criteria for DSM-IV substance dependence; VMPC group members received neuropsychological and neuroanatomical assessments establishing a bilateral lesion of VMPC cortices. Normal control participants were solicited by newspaper advertisements. A computerized version of the gambling task was employed.

Figure 1.1 shows the results of the gambling task for the three groups: controls, SD, and VMPC lesion patients. It is apparent that

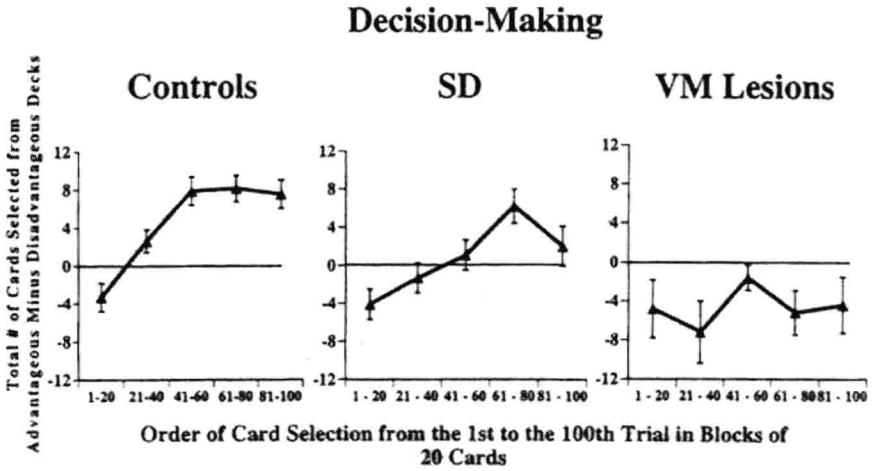

FIGURE 1.1. Relative to normal controls, substance dependents (SD) were impaired in their performance on the gambling task, but the impairment was not as severe as that seen in patients with bilateral lesions of the ventromedial prefrontal cortex (VM Lesions). Scores on the gambling task are presented as the mean + standard error of the mean (SEM) of the difference between the total number of cards chosen from the advantageous decks (C′ + D′) minus the total number chosen from the disadvantageous decks (A′ + B′). The scores are divided into five blocks of trials with 20 trials in each block, i.e. a total of 100 card selections (Bechara *et al.*, 2001, p. 383).