Robin Room

y of California at Berkeley, School
.
ited States roadside breathtesting
rsity of Michigan, Ann Arbor.
nse to alcohol-related problems,"
n Alcohol Abuse and Alcoholism,

Definitionen und Gebrauchsmus-
ıe. Lausanne, Switzerland.
rische Alkoholkonsum," Schweiz-
, Switzerland.
and tobacco in Britain," William

CHAPTER 13

A Comprehensive Theory of the Pathogenesis of Alcoholism

Ulrich Golüke

Resource Policy Center
Thayer School of Engineering
Dartmouth College
Hanover, New Hampshire

Robert Landeen

Department of Psychiatry
Dartmouth Medical School
Hanover, New Hampshire

and

Dennis Meadows

Resource Policy Center
Thayer School of Engineering
Dartmouth College
Hanover, New Hampshire

INTRODUCTION

Measured by the number of meetings and publications on the subject, the current knowledge of alcoholism is substantial and increasing. Yet one finds that the new knowledge, the numerous meetings, and all the

federal dollars spent on addressing the problem of alcohol abuse have had an insignificant impact on the prevalence of alcoholism. Recidivism rates are still 30%–70%, and estimated costs of alcohol abuse rose to about $40 billion in 1975 in the United States alone. The frustrating lack of progress can be attributed to the lack of a comprehensive theory of alcoholism. Most theories, or models, of alcoholism are like the descriptions of an elephant by blind people: they capture reality, but only partially.

To improve on the current state of knowledge, we have taken a fresh look at the life cycle of a prototypical alcoholic. Guided by a general systems perspective, we have developed a comprehensive and internally consistent theory of individual alcohol addiction. The theory includes the major physiological, psychological, social, and cultural factors that are believed to cause and sustain alcoholism. It explains the four major modes of alcohol consumption: social drinking, alcoholism, recovery, and relapse. We also use the theory to investiage several treatment modalities.

We argue that personal distress, which is an intolerable deviation of reality from some personal norm, leads an individual to search for a behavior that can reduce the level of distress. Consuming alcohol is one such behavior. From our analysis, we draw the following three conclusions:

1. Decisions about the choice of behavior are made individually; hence sociodemographic variables alone are poor predictors of drinking behavior.

2. Alcoholism alters substantially every aspect of the alcoholic's body and environment; hence, treatment must deal with the entire person as well as his or her milieu.

3. Effective treatment of alcoholism requires reducing the level of distress. This is best accomplished by eliminating the cause of distress or by improving the person's coping skills. Less attractive is substituting less destructive behaviors for alcohol consumption; worst is merely to change the agent of addiction.

A dilemma confronts anyone working toward a comprehensive theory of alcoholism. To satisfy Sargent's (1973) six requirements for such a theory (applicability, exhaustiveness, testability, universality, relevancy, and transparency), the presentation must be general enough to account for each class of drinkers. Yet therapy takes place only on a patient-by-patient basis, and therefore, no general theory is useful if it cannot be related with relative ease to each individual and unique patient. We have chosen to address this dilemma by speaking mainly in general terms. We propose causal mechanisms and use generic

terminology that applies to some extent to everyone who drinks alcohol. We strive deliberately to provide a conceptual framework for gaining insights into the behavior of all those who drink. The result is, at best, a comprehensive and consistent approach that provides useful information about every drinker, but at the cost of not providing useful data about any one drinker in particular.

This chapter, then, imposes two demands on the reader. The first is the willingness and ability to visualize the life span of an indiviual drinker as the continuous evolution and interaction of myriad social, psychological, and physiological factors. The second is the ability to move with ease from the general to the specific and back. Since our readers, in most cases, have a large fund of patient-specific knowledge and experience, we restrict ourselves to providing the general theory and some illustrations on how to bridge the gap between the general and the specific.

Primary among our illustrations is the use of a case study, drawn with a pseudonym from the files at the Dartmouth Mental Health Center. "Peter Smith" appears in several places in the chapter to illustrate how a general theory can be applied to a real case, and how a real case can be used to refine a general theory. We also use Smith to show how graphical representations of social, biological, and other variables can be used as a shorthand notation to describe the outlines of an alcoholic's life.

Peter Smith is merely one example. We rely on the wisdom and the insights of the readers to apply our theory to their personal experience with individual patients and, based on that experience, to suggest improvements in the theory.

A Case History

On admission to an inpatient alcohol-treatment program, Peter Smith, a 40-year-old divorced male, said that he "needed to get off alcohol" and "make decisions about his life." He had started drinking in his early teens. He admitted to having used many drugs briefly at one time or another, but he had used only alcohol and marijuana recently. He had never experienced detoxification and had never been through severe withdrawal. He reported only mild tremors in the morning. Usually, he drank a few beers around lunch. He had drunk heavily for at least 19 years. His parents were strict Catholics and drank rather seldom. He still went to church but did so only "perfunctiously." His brothers also had some alcohol problems, as did three relatives on his father's side of the family. Recently, he had been convicted of

driving while intoxicated, his only offense so far. He worked with a bookbinder and had held numerous jobs in his career. The longest, at the time his current job, had so far lasted 4½ years. He was divorced, the father of three children, and although he started to drink heavily when he left the army, he did not remember his former wife's complaining about his drinking until much later. Since his divorce, five years before, he had lived with a series of girlfriends. The last one had left him a week before he was admitted to the program. He was also looking for a new apartment since the rent for his current one had been raised to a level he could not afford. His consumption at the time was 20–25 beers a day and 4–5 shots of whiskey, up from a recent 15–20 beers and 1–2 shots of whiskey.

The mental status examination revealed that he was reluctant to talk and retreated to generalities. He was anxious. His speech processes and thought content indicated no sign of thought disorder. There was no evidence of hallucinations; his attention and concentration were decreased and thought to be affected by his anxiety. The physical exam showed that he was seriously overweight. Most other measures were within normal ranges, although compared with that of his age group, his general health was somewhat impaired. The Minnesota Multiple Personality Inventory and Differential Aptitude Test revealed that he was moderately anxious and depressed. His emotional controls were brittle. The diagnosis was alcoholism, chronic excessive use. After 30 days in treatment, he was discharged and he expressed confidence that he would remain sober.

This case description is typical of a good number of alcoholics: high consumption, many problems, but still functioning, at least on the surface. To show how our theory can be adapted to this specific case, and that it can simulate in broad outline Smith's life from about 10 years of age until admission to the program, we adjusted the model parameters so that they would represent his characteristics to the largest extent possible. For example, we set the body weight at 90 kg and the drinking norm of his parents at 50g/week, which is less than two beers per week. We then subjected the model to a slowly growing rate of stimulation arrival, to reflect normally growing adult responsibilities like military service, marriage, jobs, and parenthood. To reflect specific clusters of life events, we superimposed random variations on the rising stimulation rate. In Figure 1, we present key variables from the result of the tests, those alluded to in the case history. The horizontal axis represents age, from 10 to 40 years. Average alcohol consumption, in the top half of the figure, starts at zero at age 10 and begins a very gradual rise toward the end of the teens. The upward trend continues during the 20s and accelerates around age 25, the age that Mr. Smith

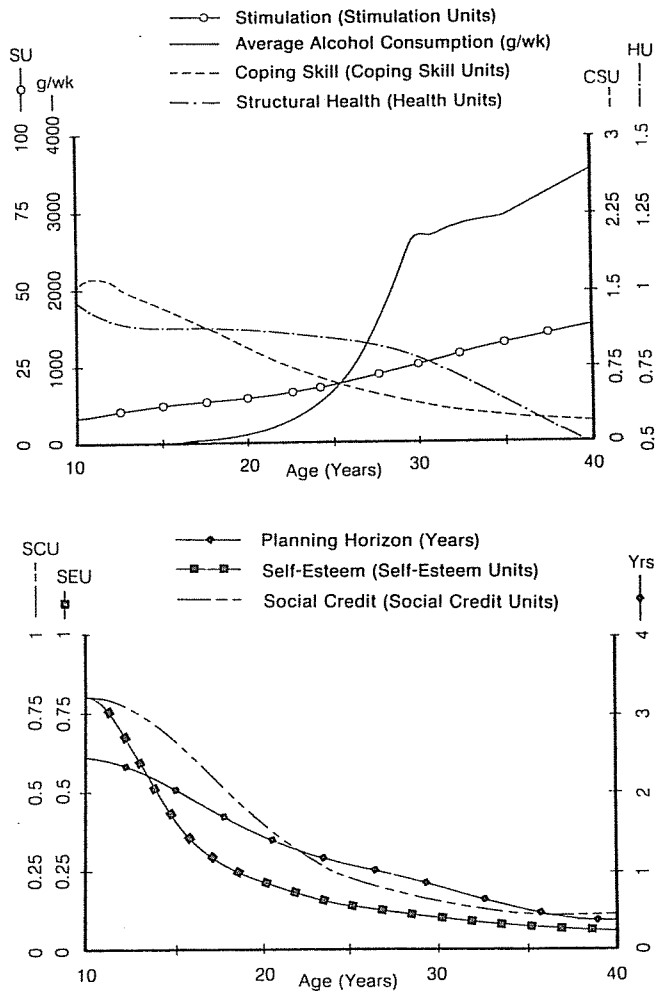

FIGURE 1. Mr. Smith becomes alcoholic.

was discharged from the army. Consumption continues to rise until his early 30s. After that, it levels off around 3000 g/week or almost two six-packs of beer a day. After the divorce, at age 35, consumption resumes a gradual climb until admission to the program. Stimulation grows steadily at some 1% a year to reflect responsibilities as mentioned. Smith's health, relative to his age group, starts to decline after age 25, a result of the markedly rising alcohol consumption at that time. Self-esteem is high originally, a sign of the maturation process of any teenager, but then declines. His social credit is highest as a teenager,

has started the decline by age 15, and continues to fall off until age 40. This development reflects his disassociation from his parents and the church, and the loss of spouse and family. Coping skills also decline continuously. This is especially worrisome since at the same time stimulation continues to rise. Consequently, the gap between actual and required capabilities grows steadily. Smith's planning horizon falls from over two years early in his life to between three and six months. Not surprisingly, a four-week treatment stay without alcohol makes him believe that he has the problem under control.

Theoretically, at this point, Mr. Smith has several options. He may resume heavy drinking immediately. He may keep consumption down for some years, only to relapse after that. Or he may stop alcohol consumption completely and show some improvement in the other variables. In graphical terms, these options are presented in Figure 2. Hypothetically, Mr. Smith might also never have become an alcoholic. He then would have presented the following history (Figure 3). His alcohol consumption would have stayed low, in ranges that are socially acceptable and do not carry the risk of addiction. His health would have stayed within the limits of that of his age cohort; his self-esteem and social credit would have risen. His coping skills would also have risen enough to let him deal adequately with normally rising stimulation.

The four possibilities, presented above—untreated alcoholism, recovery, relapse, and social drinking—have for a long time raised crucial questions in alcohol research. One would like to know what to do so that a social drinker remains a social drinker. How should one tax alcoholic beverages? What should the drinking age be? Should alcoholism be accepted as part of social drinking in the sense that one accepts highway deaths as part of the nation's transportation system?

Any of these questions, however, are irrelevant after a person has become alcoholic. The question is then one of providing the specific type and intensity of treatment from all the myriad possibilities shown in Table 1 that will prove effective for a particular patient. Here, one wants to know how much treatment, how intensive, how long, what kind, and in what sequence.

Should complete recovery remain impossible, one then wants to know how to arrest the progression of the disease. Even if all has failed, one still needs to learn as much as possible about alcoholics so that one is in a better position to help their successors.

Traditional Tools of Inquiry

The questions posed by Smith's case are not so much new as they are embarrassing in their persistence. Attempts to answer them have

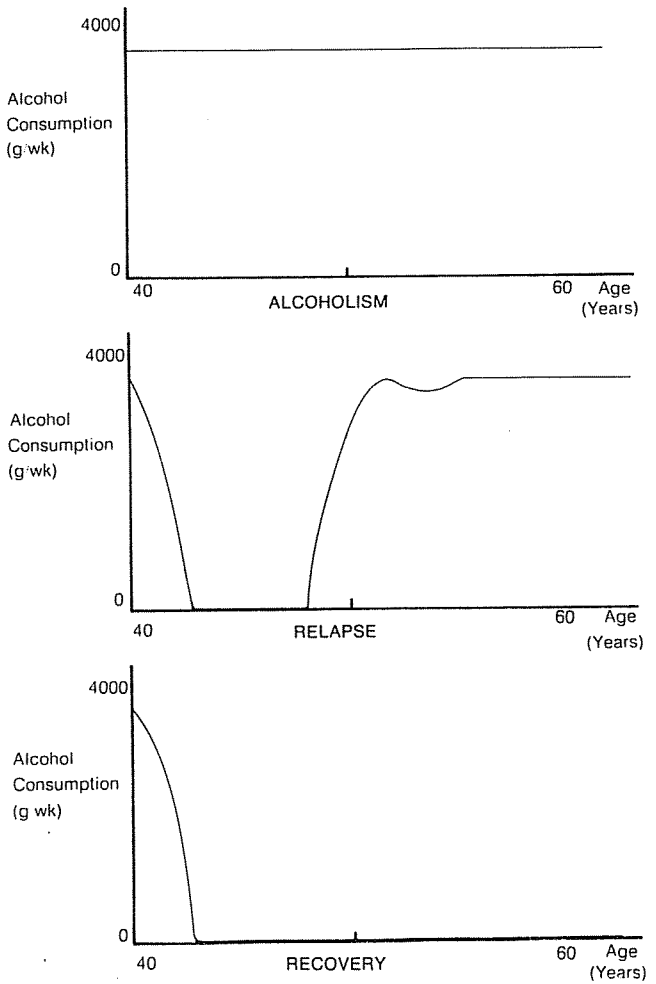

FIGURE 2. Mr. Smith's drinking options at age 40.

progressed through many forms during this century. The first approximate answers came from monocausal etiological hypotheses. In the 1940s, endocrine malfunction was thought to lead to alcoholism (Gross, 1945; Tintera and Lovell, 1949). In 1959, Williams proposed genetrophic mechanisms as the cause of alcoholism. The first psychoanalytic theories were influenced by the Freudian concept of an unconscious self-destructive tendency as the cause (Menninger, 1938, pp. 160–184). The Adlerian view of alcohol abuse as a yearning for power has been

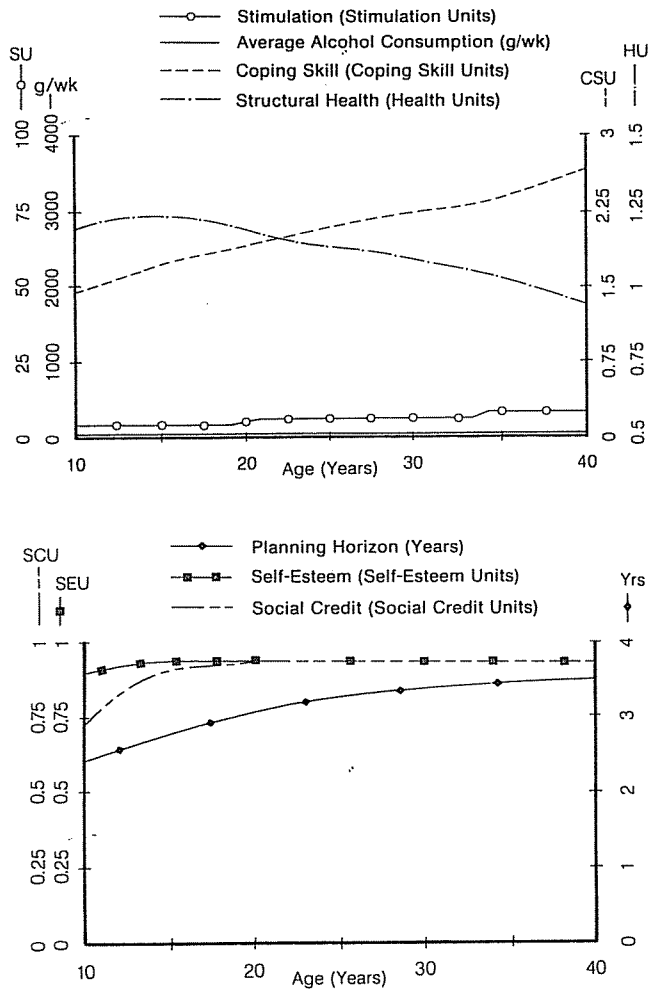

FIGURE 3. Mr. Smith as a hypothetical social drinker.

examined in detail and corroborated by McClelland and his co-workers (McClelland *et al.*, 1972).

Other theories of alcoholism are Ullmann's (1952) tension reduction theory, Bales's (1946) cultural theory, and, a little later, Jessor's theory of deviant behavior (Jessor *et al.*, 1968), Goodwin's (1976) hereditary theory, alcoholic personality theories (Blane 1968), and, more recently, theories about the availability and accessibility of alcohol (Bruun *et al.*, 1975) and the epidemic model of alcoholism and drug abuse (Bejerot,

TABLE 1. Tools of Intervention

I. Physical rehabilitation
  1. Detoxification
    a. Acute: Preventing ethanol intake by titrating with a long-acting cross-tolerant drug.
    b. Long-term: Monitoring natural decay. Use of depressants (controversial). Use of magnesium to stabilize cell membrane activity.
  2. Physical impairment secondary to alcoholism
    Vitamins for the structural and functional rehabilitation of the brain
    Proteins
    Hormones
    Patient-specific medical treatment
  3. Organic syndromes (prealcoholic)
    Lithium
    Antipsychotics
    Antidepressants
    Antianxiety drugs
    Drugs to deal with premenstrual tension
    Electro-convulsive therapy
    Psychosurgery (controversial)
  4. Psychophysiological dependence
    Disulfiram
    Amesthystic drugs
    Acupuncture
    Exercise

II. Psychosocial rehabilitation
  1. Behavior modification
    Self-control training
    Biofeedback
    Relaxation techniques
    Stress habituation
    Cognitive-behavior therapy
    Aversive therapy
    Operant conditioning
    Contracting
    Assertiveness training
    Social skill training
    Hypnotherapy
  2. Psychotherapy (existential and psychodynamic)
    Group therapy
    Family therapy
    AA
      Alanon
      Alateen
    Peer group, residential group
    Occupational therapy
    Lifestyle training
      Leisure skills
      Education

1972). One must also mention learning theories (Vogel-Sprott, 1972; Tarter and Sugarman, 1976), transitional theories (Steiner, 1971), and dissonance reduction theories (Oppenheim, 1976).

Though most theories have been modified as new data have been accumulated, and although some are sharply criticized (Cappel, 1975), most medical theories do survive in one form or another. There is a good reason for their resilience in the face of the dismal treatment records based on them: they are all necessary, but insufficient, descriptions of reality. There is always some alcoholic whose crucial enzyme is missing, always someone who drinks to experience delusions of power, always someone who will stop when the price of alcohol is high enough, and always one who has an oral fixation. Unfortunately, however, the reverse is also true: there will always be one who drinks even when he or she is biochemically sound, one who already has real power, one who will drink himself or herself to economic ruin, and always one who drinks without oral fixation. Hence "the search for a *single* cause of alcoholism may be an unrealistic goal" (emphasis added, U.S. Department of Health, Education, and Welfare 1971, p. 67).

Partial Solutions

As a result, the idea of multicausal theories of alcoholism evolved (Roebuck and Kessler, 1972). In addition, several attempts have been made to go beyond mono- and multicausality to do justice to the complexity of alcoholism. One of the simplest approaches has been the transfer of the public health model to alcoholism. The model postulates that interactions between host, environment, and agent fully determine any particular manifestation of a given disease (Figure 4). Though usually used only as a conceptualization of alcoholism (Mello, 1976, p. 175), it has also been taken more literally (Alcaron, 1969; Hughes and Crawford, 1972; Bejerot and Bejerot, 1978). Another attempt at building a comprehensive theory is presented by Jellinek's typology of alcoholics (1960, pp. 36–39). Kissin (1977b) extended the original Jellinek typology by arguing that the alpha, beta, gamma, and delta

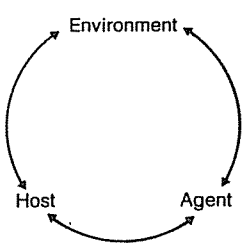

FIGURE 4. Public health model of alcoholism.

alcoholics are merely four specific points in a three-dimensional space. With this space, Kissin provided an adequate snapshot of drinking behavior at any given time, but he could not address the question of how any of the types got where they are, not is it possible to explore how to move people to more acceptable locations. Edwards (1977) has also suggested a way to synthesize the detailed and often unrelated pieces of knowledge about alcohol drinking by proposing the alcohol-dependence syndrome as the focal concept.

Meanwhile, several atheoretical approaches have been taken to help in the study of alcoholism. Probably the most prominent has been the work of Cahalan and his co-workers. (Cahalan *et al.*, 1969: Cahalan, 1970). Theirs is a massive cross-sectional study of American drinking habits. Along similar paradigmatic lines, the Rutgers center has recently launched a substantial longitudinal study (Rutgers, 1979). Though both Cahalan's work and the Rutgers study are examples of research carried out to high scientific standards, one must not forget Edwards's (1978) warning that "epidemiology seems somewhat to acquire the image of a rather desicated science, offering only the vision of a world seen through holes in IBM cards" (pp. 30–31).

A third generic line of inquiry can be described as *curve-fitting*. Some data have been observed to be reasonably well described by some mathematical function or process. For lack of any causative hypothesis, the mathematical process is then proposed as a descriptive hypothesis. Lederman (1956), for example, found that the distribution of individual consumers in a population coincided closely with the log-normal distribution curve (p. 126). He tested the proposition with cross-sectional data from France, Sweden, Italy, Finland, and the United States. DeLint and Schmidt (1968) also found a reasonable fit between data from Ontario and the mathematical model. Since then, the Lederman curve has been used *a priori* as the relevant description of alcohol consumption distributions, though Miller and Agnew (1974) strongly objected to its use. In any case, its applicability does not extend to the single drinker. In fact, the acceptance of a probability distribution is an explicit recognition that there is no way to predict alcohol-drinking behavior for a particular person.

First attempts at using the Markov chain to describe the movement of members of one drinking category to another over time have been made by Fillmore (1974), Katzper *et al.* (1976), and Roizen *et al.* (1978). As an example, the transitional probabilities from Fillmore's study (1974, p. 826) are shown in Table 2.

The growing knowledge about alcoholism is generally scientifically sound and empirically valid (e.g., Kissin and Begleiter, 1971–1977; Gross, 1973–1977; Seixas and Galanter, 1977–1979). Ironically, how-

TABLE 2. Transition Probability Matrix[a]

		Age 20					
		Abstainers		Nonproblem Drinkers		Problem Drinkers	
Age 40	Abstainers	.29	(.26)	.14	(.10)	.04	(.00)
	Nonproblem Drinkers	.61	(.67)	.77	(.80)	.68	(.67)
	Problem Drinkers	.10	(.07)	.09	(.10)	.28	(.33)

[a] $N = 109$ (97), total $N = 206$. The figures in parentheses refer to females, the others to males. From Fillmore (1974, p. 826).

ever, it has not lead to an increased consensus. Dole (1978) observed that "various specialists see addiction in different ways and as a consequence fail to communicate effectively with each other (p. 37). As a result, he continued, "clinicians have no basic understanding of the addictive process" (p. 45). The price for the profession's inability to deal with the problem of alcoholism is paid by the patient and by society:

> The treatment of alcoholism has not improved in any important way in the last 25 years, alcoholism is becoming more prevalent, not less . . . only a minority of patients who enter treatment are helped to long-term recovery; the majority is made of those who relapse soon and often and those who are lost to follow-up. (Gordis, 1976, p. 821)

In support of this finding, Baekeland (1977) found the pooled success rate of some 30 outcome studies he reviewed to be only 40%, a figure that dropped to about 35% if adjustments were made for spontaneous improvements. Along similarly disturbing lines, Edwards *et al.* (1977; but cf. Kissin *et al.*, 1977) reported no significant difference in the outcome of carefully selected patients who received extensive "treatment" or "advice alone." Not surprisingly, Clare (1977) noted very pointedly that "a small proportion of alcoholics achieve abstinence within six to twelve months of receiving treatment, whatever the nature, intensity, and duration of the treatment concerned," and that "there is little evidence that costly and intensive therapeutic interventions are more efficacious than more economic and simple approaches in bringing about remission" (p. 287).

SYSTEM DYNAMICS

Alcoholism is a systematic disease par excellence. It destroys the patient's liver as it ruins her or his finances. Cell membrane activity is

disrupted, as are all social relations. Self-esteem disappears, as does credit rating. Customarily, the classical scientific method is used to investigate each of these problems independently; the liver, the cell membrane, the self-esteem, and the social dimensions are all examined as if each one were totally removed from everything else. These methods fail, though, when the solution to the problem under investigation is not to be found within any of the separate pieces—when, in fact, the whole has properties that are more than the sum of its parts. Therefore, it is the particular point of view that originally led to today's immense wealth in specialized knowledge about alcohol abuse that is now responsible for the inability to understand the whole problem. Consequently, one needs a new point of view, one that stresses the investigation of relationships between parts, as well as the character of the parts themselves.

Such a view forms the basis of system dynamics, a specialty within the field of general systems theory (Bertalanffy, 1968). System dynamics has been developed at the Massachusetts Institute of Technology since 1955 (Forrester, 1961; Goodman, 1974; Pugh, 1976). Examples of successful system-dynamics applications have involved heroin addiction, cancer, diabetes, and fertility (Levin *et al.*, 1975; Richmond, 1977; Foster *et al.*, 1973; Meadows *et al.*, 1974).

In developing a system dynamics model, one identifies the set of forces believed to cause and sustain a certain behavior. The description is then translated into mathematical shorthand notation. The model can then be subjected to a degree of scrutiny and peer criticism that could never be achieved for a theory expressed in prose. Nevertheless, a system dynamics model is similar to the mental or intuitive model everyone employs to direct actions toward a certain goal. As in a mental model, one considers a wide range of information sources differing in precision and accuracy—from undisputed empirical data all the way to educated guesses. The flexibility of such a model allows one to update the sources at any time that more reliable information becomes known.

To illustrate the procedure of system dynamics modeling, and to familiarize the reader with the necessary technical detail, we sketch here how a model of Mr. Smith might be developed. We start with a verbal description of his life. He left home for the military; he had to deal with marital problems; the responsibility of parenthood was thrust on him; and a series of unpredictable events, such as rent increases, presented themselves. Still, by themselves, these events do not constitute problems. Others have been soldiers and have married and had three children and still did not become alcoholics. Hence, we need to speculate about the desired state that Mr. Smith would like to attain.

That may very well be a steady job, a rewarding family life, close friends, and enjoyable leisure time. Obviously, in his case, reality and desire did not match; instead, a deviation existed between them. And yet, this deviation would not have been disastrous if Mr. Smith had had the necessary coping skills to change either one: reality or desire. But he lacked them. The case history mentions that his emotional controls were brittle. Unable to change his situation, he turned to alcohol to distort his perceptions, reducing the deviation between his experienced reality and his desires. Though his parents' low drinking norm probably discouraged the use of alcohol at first, one can presume that he learned in the army about the inebriating effect of alcohol. The availability and the social arrangements for alcohol consumption then tended to set a stable pattern. He drank more and more. After some time, his body adapted to the persistent presence of ethanol. Cellular tolerance, which is one aspect of adaptation, undermined the inebriating capacity of a given amount of alcohol, and consequently, Smith consumed ever-increasing doses to achieve the same effect in reducing the perceived deviation. He also developed metabolic tolerance, another aspect of adaptation. His body became adept at metabolizing the increasing amounts made necessary by his cellular tolerance. Thus, he became trapped in a set of powerful, vicious cycles. He used alcohol to alleviate current symptoms, and it was instrumental in causing more problems later on. He will continue to try to alleviate the problems by drinking even more alcohol.

To formalize this description, we note the key concepts and the causal influences between them. If we concentrate for the sake of clarity and brevity, but at the expense of comprehensiveness, on Mr. Smith's marital relations, we can say that reality is marital distress, whereas his desired state is marital bliss (Figure 5). Normally, a person in his situation would take the deviation as a cue to engage in behavior designed to reduce the deviation; one might, for example, go to a marriage counselor. Hence, the causal influence from deviation via marriage counseling and effect to marital distress is negative; that is to say that *increasing* deviation leads to actions to *reduce* the cause of deviation.

The graphical notation in Figure 5 is called *causal loop diagramming*. In this simple convention, a plus sign at the head of an arrow indicates a link through which the causal variable and the affected variable tend to move in the same direction: as marital distress rises, deviations increase; should distress decrease, so will deviation. A minus sign at the head of an arrow indicates a link through which cause and effect

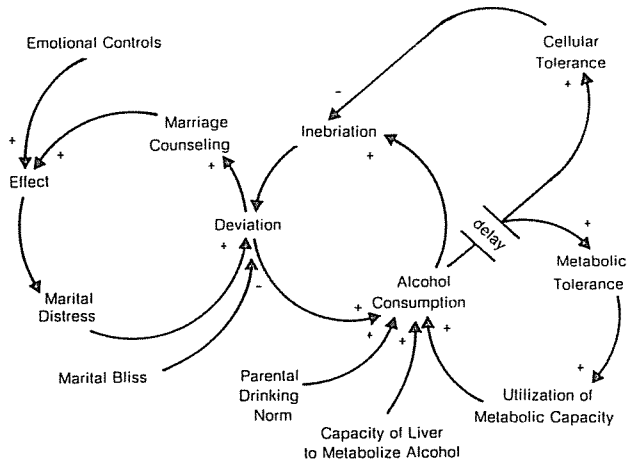

FIGURE 5. Familial problems lead Mr. Smith to start drinking. In the long run, he develops tolerance to alcohol and becomes an alcoholic.

move in opposite directions: as effect increases, distress decreases; and if the effect decreases, then distress will increase. The effectiveness of this last link, however, is subject to emotional controls. The better the controls, the more effective the counseling, and hence a positive link between emotional controls and effect. It is unfortunate that Mr. Smith does not have the skills to deal with the breakup of his marriage and the subsequent loss of three girlfriends. Instead, he engages in behavior that has already proved valuable to him in other situations: drinking. Though his parental drinking norm tended to discourage alcohol consumption (note the negative sign at the head of the arrow between the parental norm and consumption), increasing deviations still lead to more consumption. The more he drinks, the more drunk he gets, and the more drunk he is, the less he is aware of the deviation between distress and bliss. Unfortunately, persistent consumption leads, after a delay, to both cellular and metabolic tolerance; both causal links are positive. And as cellular tolerance decreases the inebriating effect of a given amount of alcohol (note the negative sign between cellular tolerance and inebriation), metabolic tolerance increases the efficiency of the metabolic system so that he can, in fact, drink more.

In the previous step, we sketched the development of a causal loop diagram of the set of forces believed to cause and sustain a behavior of concern. It is a summary statement of the model and is used in early conceptualization and in explaining the basic results of the finished

model. In between lie several technical steps that need not concern us here. Full technical documentation of the theory is available from the authors (Goluke, 1980).

A THEORY OF ALCOHOLISM

As mentioned in the introduction, to develop a general theory, we need a terminology that is divorced from the specific circumstances of any given alcoholic, yet suggestive enough to describe all alcoholics. A point in case is Mr. Smith's marital distress. It certainly played a role in the development of his drinking, but not exclusive of other things. Frequent job changes, unpredictable disturbances, and the like contributed as well. If one looks beyond the single case, loss of earning power, depression, biochemical imbalances, and congenital factors—to name but a few examples—are all part of the reality confronting a particular person. Though we could address the proliferation of specific variables by enumeration, most of the clarity and insights that can be gained from a system dynamics model would be lost. Instead, we choose generic terms to convey our ideas. For marital distress, frequent job changes, loss of earning power, congenital factors, and so on, we substitute the generic term *stimulation*. For the desired or expected state of reality we use the generic term *stimulaton norm*. In Table 3, we present a set of illustrations: the reality may be frostbitten toes. The norm, or desired state, is a temperature of around 37°C; to adjust reality to the norm, one needs coping skills in the form of warm water, blankets, and some rudimentary medical knowledge. A lottery win may require sound investment talent if the norm of steady income is to be maintained.

TABLE 3. Generic Concepts and Specific Examples

Stimulation	Stimulation norm	Coping skill
a. Loss of income	Steady income	Good references, savings
b. Lottery win	Steady income	Knowledge of investing
c. Death of spouse	Marital life	Mourning
d. Hunger	Food	Knowledge of nutrition, means of obtaining food
e. Frostbite	37°C body temperature	Blankets, warm water
f. Depression	Happiness	Close friends, therapy
g. Hemophilia	Coagulation	Protective clothes and surroundings
h. Social ostracism	Social acceptance	Migration to a new milieu

These examples are only suggestive of the type of bridges that must be built by the reader to move from the general to the specific and back.

Overview

We identify four specific stages in the course of alcoholism:

Stage I: Daily stimulation continuously moves the person away from the stimulation norm along physiological, psychological, and social dimensions. The resulting deviation causes the person to invoke his or her set of adjustment mechanisms to keep himself or herself within tolerable proximity of the stimulation norm. The level of his or her coping skills determines how well he or she succeeds in this task. The level of coping skills and the ability to develop new skills is partly genetically determined and partly learned. During this stage, successful coping is the active mechanism that controls alcohol intake. When problems remain small, drinking is not necessary. Reasons to drink at all are usually social, as, for example, at a party or in a bar with friends after work.

Stage II: However, if the given level of coping skills does not suffice to return the person close enough to his or her norm, then he or she will engage in behavior designed to augment intrinsic coping skills, for example, calling on outside help, consuming substances that reduce the perception of deviations between norm and reality, thus enabling him or her to make better use of what coping skills are available. Alcohol has that distortive effect, and we call it its *homeostatic capacity*. At this second stage in the development of alcoholism, the drug's homeostatic capacity controls its intake: one drinks only until the world looks rosy again. It is imperative, however, that the person use the time during which the perception is distorted to remove the real source of the deviation. Otherwise a second drink is necessary when the effect of the first one has worn off.

Stage III: Should drinking become the preferred way of "dealing" with deviations, the person adapts physiologically to the presence of alcohol and need progressively more ethanol to get the same effect. At this point, the person has entered what Kissin (1977b, p. 5) called the "addictive cycles." At this stage, no functional control over intake exists. This is the stage where one observes the dramatic increase in consumption, from a few daily drinks to a case or two of beer or more. While subject to these vicious circles, the person surmounts several obstacles to success in his or her career as an alcoholic: he or she must overcome his or her own developing acknowledgment of a drinking problem and, at some point, the restraining influence of the social environment.

Stage IV: Should the person make it past all constraints, of stages I–III, an alcoholic eventually has to taper off consumption increases because of the purely physiological limits of his or her metabolic system.

Though presented in four distinct stages, the progression is in reality gradual, taking years, if not decades, to go through.

These four stages can be explained in more detail with the help of a causal loop diagram. First consider Stage I in Figure 6. The norm defines the acceptable level of stimulation. Recall that this embraces physiological, psychological, and social norms. If stimulation differs from the norm, a deviation exists. And if coping skills are adequate, the deviation itself gives rise to adjustment processes designed to alter the level of stimulation so as to minimize future deviations. In the terminology of system dynamics, we have described a negative loop: changes in one variable, stimulation, give rise to effects that eventually work to counteract the original change. Negative loops are goal-directed. They allow oscillations around norms, but not sustained growth or decay. They tend to maintain the behavior of the system within bounds.

In Stage II, if the deviation surpasses tolerable limits and if coping skills do not suffice, alcohol is consumed to reduce temporarily the perception of the deviation, thus making it appear as if the deviation were amenable to intrinsic coping skills. This is the homeostatic capacity of ethanol: "People under conditions of strain and stress may experience relaxation after a drink and thus may cope better with a . . . situation" (Myrsten, 1977, p. 326). Again, we have described a negative loop. This loop, however, does not deal with stimulation, norm, and coping skills,

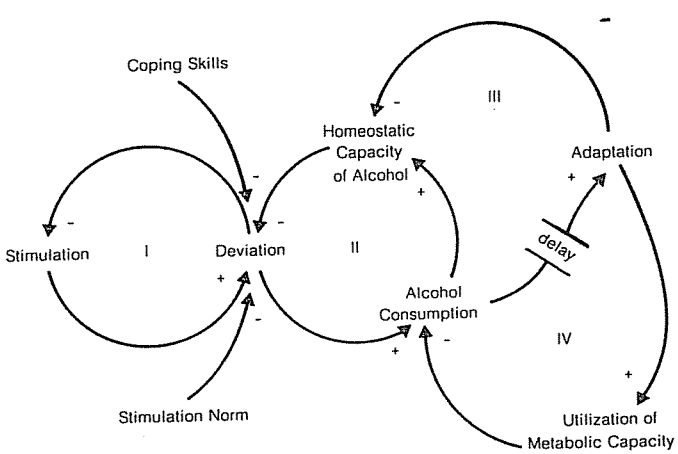

FIGURE 6. Deviations between stimulation and stimulation norm lead to drinking. If excessive, this leads in the long run to adaptation and to alcoholic drinking.

the combination of which originally determines the deviation. Paraphrasing Kissin (1977b, p. 32), alcohol helps the person only to ignore the problem, but not to reduce it.

In Stage III, "alcoholismic drinking is the result of a complete failure to discriminate and cope with data coming from social as well as 'internal' . . . environments" (Naitoh, 1972, p. 422). After a delay, consistent alcohol consumption produces adaptation to the presence of ethanol. One of the effects of this process is a reduced homeostatic capacity for a given amount of alcohol. Following the arrows in Figure 6, one sees that as stimulation deviates increasingly from the norm, alcohol consumption increases, leading to adaptation. This reduces the homeostatic capacity, hence increasing even further the original deviation, all else being equal. This is a positive loop. An original increase in any variable sets off a chain of events around the loop that eventually increases the original variable even more. Explosive growth or precipitous decay is the characteristic behavior of a system dominated by positive loops.

In Stage IV, the person eventually reaches his or her physiologically determined metabolic limit, if nothing is done to stop the growth in alcohol consumption. Adaptation, through a more efficient utilization of the metabolic system, lifts the restraint to a higher level, but sooner or later, the alcoholic runs into this last-but-one constraint. Beyond the metabolic limit is only death.

Lest our model be judged to imply that only stress leads to alcoholism, let us say that several other conditions lead to the same result. In Figure 7 we show alcohol consumption as it developed from the following assumptions:

1. For the curve labeled "social drinking," we subjected the model to a slowly rising input in stimulation, together with random variation. The capacity of the adaptive system proved sufficient, and no alcoholism resulted.
2. Doubling the rate of growth of stimulation leads to alcoholism from overstimulation.
3. The same alcoholic drinking behavior can be achieved in the model when the original stimulation input is coupled with increased coping skills. Here, the model simulates a person who drinks out of boredom.
4. The original stimulation input together with very low self-esteem can also lead to alcoholism.
5. Lastly, alcoholism can be caused by social interactions.

In all these cases, consumption becomes excessive enough to reach the metabolic limit.

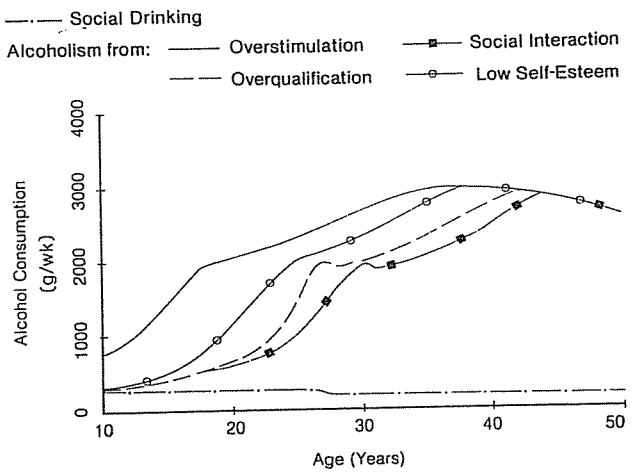

FIGURE 7. Alcoholism from four causes.

The description of the four stages also answers the question of what it is that makes a social drinker stay a social drinker. Though we cannot change Mr. Smith's life history, we can examine the hypothetical case of what change would have been necessary for him to lead a life of controlled social drinking. There are six strategies to prevent excessive alcohol consumption. First, a slower rise in the level of stimulation would have left him in a position where his coping skills sufficed to deal adequately with his problems, without his using alcohol. Second, if the problems had been less disruptive, his coping skills again would have sufficed. Third, a higher level of coping skills would have kept deviations within tolerable limits. Fourth, had alcohol been less available and less appealing to him, he would have avoided excessive consumption. Fifth, a much stricter enforcement of his parents' low drinking norm may have sufficed, and lastly, a more rapid acknowledgment of his developing alcoholism would have been enough to control the impending disease. Figure 8 shows that each of these changes by itself has the desired effect of keeping Mr. Smith's hypothetical consumption below 500 g/week, or about two beers a day. The labels in the figure refer to the change in the model that produced the particular outcome.

Scope of the Study

Before we delve into the details of our theory of factors governing the life cycle of an individual alcoholic, it is important to characterize our principal contribution and indicate the precise role of critical

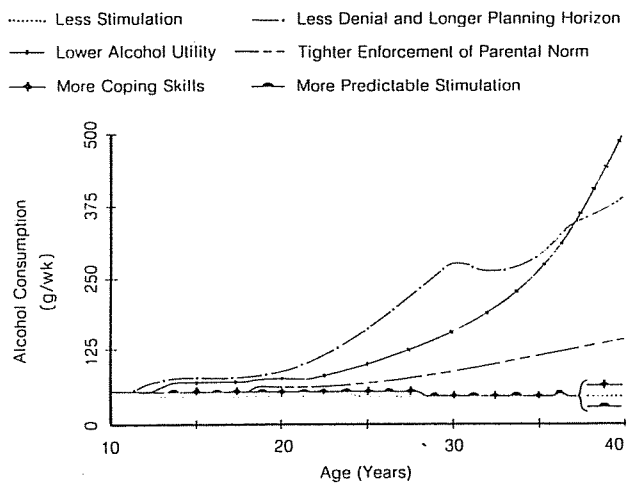

FIGURE 8. Six different strategies result in social drinking.

analysis, because every reader is likely to disagree with some aspect of the theory.

We have *not* conducted new empirical studies. Our theory is based on existing empirical research, social surveys, and the deductions of researchers and therapists interested in the phenomenon of alcohol addiction. Instead, we have provided three kinds of synthesis:

1. We selectively chose a body of facts and theories from among the many presented in the literature.

2. We integrated these causal facts and hypotheses in a detailed, precise, and comprehensive theory of alcohol addiction, and we present several computer simulation runs of the model to indicate the implications of the current version.

3. We convey this theory in a fashion that will let others substitute one or more of their own facts and theories for those incorporated in our theory, and we work to provide the readers of this paper with some intuitive impressions of how changes in the model are likely to change the overall conclusions.

The result is a tool that need not be discarded even if several pieces of it have to be changed. Our central contribution is not the specific set of hypotheses we selected, but the way in which they are interrelated. The interrelation remains unaltered even if many of the specific pieces are changed. In this way, we have put a special obligation on the reader. Where someone disagrees with a particular piece of the theory, it would be helpful if they would suggest a specific change, insert it in the model,

and then analyze the result to determine whether any of the model's basic results have been altered.

Figure 9 illustrates the boundaries of the system we examine. Variables that are entirely determined by other variables of the system and in turn influence others are called *endogenous*. Examples are health, self-esteem, stress, optimal stress, planning horizon, and adaptation. Variables that influence other variables but are not themselves changed by any change in the model are *exogenous*. Examples are treatment regimes, aging, body weight, genetic makeup, and early social environment. The third category is *excluded variables*. These neither influence nor are influenced by the system. Trivially, they are the residuals of the universe and the other two categories. Specifically, we excluded morals, religion, incidence of alcoholism, and treatment-provider attitudes. We do not maintain that the excluded variables are totally unrelated to alcoholism; clearly this is not the case. Nor do we maintain that exogenous variables are in reality completely unaffected by changes in the model. Rather, we justify our particular boundary as a point of departure for our discussion. We wish to stress that both Figure 9 and this paragraph are illustrative and not exhaustive; they present only tiny samples of the variables in each category actually included in the model.

Reference Modes of Drinking Behavior

Minimally, our theory must explain the four major modes of drinking behavior: social drinking, alcoholism, recovery, and relapse.

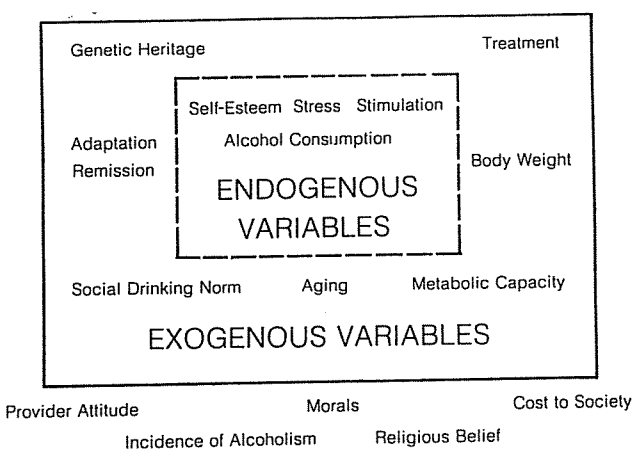

FIGURE 9. Sample variables included in the model.

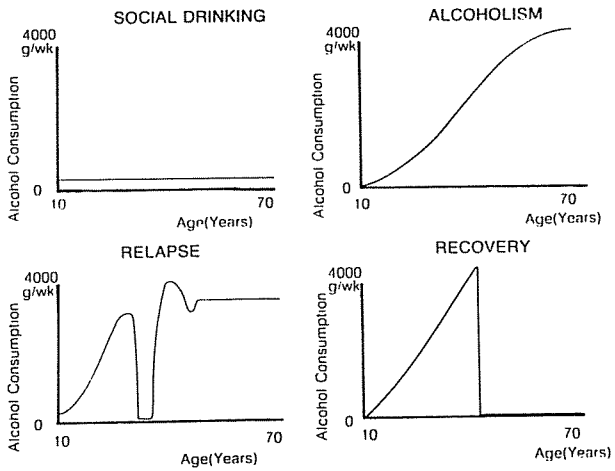

FIGURE 10. Reference modes of drinking behavior.

These four modes, originally presented in our discussion of Smith's options, can be expressed in terms of average alcohol consumption. They are presented in Figure 10. For all four modes, the horizontal axis represents age, from 10 to 60 years, and the vertical axis represents average alcohol consumption, from zero to 4000 g/week (about 20 beers daily).

Dynamic Hypothesis

The hypothesis is that a deviation of average stimulation from its norm produces attempts to eliminate the cause of deviation. These efforts may or may not succeed, depending on the quality of the coping skills available at that time. Should the deviation persist, the person may resort to the consumption of alcohol. Alcohol is chosen because of its homeostatic capabilities; it brings perceived problems back within acceptable levels and thus reduces the deviation. Consistently high alcohol consumption leads through several addictive, or positive, feedback loops to a shift in the control of consumption from psychosocial to physiological mechanisms (see Figure 6).

Major Feedback Loops

We present the major feedback loops in detail. For each, we discuss assumptions, structure, and behavior.

Adaptive Responses

As Bacon (1943) has been insisting for almost 40 years, to study the pathogenesis of a system presupposes a study of the system under

normal and healthy conditions. Vaughan (1979) made an analogous point in his discussion of brain research: "Research on psychopathology divorced from the investigation of normal brain mechanisms is severely handicapped in the search for neurobiologically significant indicators of deviant mental functioning" (p. 438).

The most recent Rand study (Polich *et al.*, 1980) shows that there is a subset of problem drinkers who do revert to social drinking. Short of defining those reverting as never having been alcoholic in the first place, one must conclude that social drinking and alcoholism are two possible behavior modes of the same underlying system. The descriptive work of Cahalan *et al.* (1969), Fillmore (1974), and Roizen *et al.* (1978) also indicates that people do move from one category to another, thus strengthening the argument that social drinking and alcoholism are, respectively, the normal and the pathological manifestations of the same system, though representing significantly different points on a continuum.

In delinating the essence of the system under normal conditions, two seminal studies by Pribram (1971) and Ashby (1960) provide important insights. Pribram concluded that a healthy person creates neural images, somewhat like neurochemical holograms, of events, processes, and expectations in the brain. The person then compares actual occurrences against these images. Any deviation calls forth a reaction to lessen the deviation. This reaction can be directed toward the actual occurrence, to bring it into line with the neural image, or it can be directed toward the image, to make it conform with reality. An example of this principle is the orienting reflex (Beck, 1976, pp. 149–150). People, and animals, focus the majority of their attention on a novel stimulus when it is first presented. If the stimulus is repeated, the response becomes progressively weaker, until it disappears. This habituation could be interpreted as a loss in sensitivity on the part of the nervous system, but Sokolov (1963) showed that a decrease in the intensity of a tone to which a subject had become accustomed called forth a full-fledged new orienting reflex. A new stimulus for which no neural image exists arrives in the brain. Hence, a deviation between neural image and occurrence exists. As a result, one observes the orienting reflex. The subject identifies the stimulation and makes sure that it presents no threat. It is creating a neural model, thus lessening the deviation. When the stimulus is repeated, the subject still notices the occurrences, but, because of its ever more accurate image, does not cause an orienting reflex to occur. Once the intensity is changed, reality and model no longer coincide. A new deviation exists, calling forth a new orienting reflex. This change occurs as long as the subject expects

to hear only one tone with one intensity. Once the subject is accustomed to random variations in the tones presented, changing intensities no longer cause the orienting reflex to occur.

Ashby (1960) also came, from quite a different direction, to the conclusion that the capacity to respond adaptively is essential for life. Having set himself the task of designing a hypothetical machine that could mimic lifelike behavior, he concluded that both the necessary and the sufficient condition for life is adaptive capabilities. Norms need to be established against which reality is compared. Deviations must then give rise to behavior capable of reducing the deviations.

Norms exist on all levels of analysis, and within the parameters set by biological constraints, people have considerable choice of the norms they accept as relevent to their life. Attempts to delineate a universal and exhaustive set of norms are frought with difficulties. Only at a very general level (Maslow, 1954; Bossel, 1977) has there been some success, but at the price of losing a good deal of the set's relevance when it is applied to a particular individual. Human variability thwarts any effort to be more specific. In general, however, one can say that behavior is determined by three concomitant factors: genetic potential, cultural heritage, and phenotypical idiosyncrasies. Culture sets the parameters for individual behavior, but in the long run, it is an expression of the aggregate behavior of its members. Similarly, genetic potential sets rigid limits on individual behavior, but in the very long run, it also is merely an expression of the aggregate success of individuals dealing with their environment. In our model, which spans only one person's lifetime, we take genetic makeup and cultural influences as given, though they can be reset as exogenous influences in the model to describe specific people.

The homeostatic capacity that maintains the organism and its subsystems within narrowly defined ranges despite wide fluctuations in the environment (Lashley, 1929; Bethe *et al.*, 1931; Cannon, 1932) is not merely concerned with physiological norms, such as steady body temperature and adequate food intake. It also regulates other areas of human endeavor. Festinger (1957) has made this capacity his central concern in the theory of cognitive dissonance, and Allport (1955), Brunswick (1956), and especially Menninger and his colleagues (1963) have extended this feature into psychological and psychiatric areas. Cofer and Appley (1964, Chapter 7) reviewed the application of the homeostatic principle to the study of motivation. In our usage, *homeostatic capacity* is the selection and implementation of all adaptive strategies, both physiological and behavioral, to achieve certain goals. The goal is to maintain deviations from the norm within viable boundaries. The

norms can be static, variable, or unpredictable. An individual has numerous norms, and they can even be in direct contradiction to each other.

Three generic mechanisms constitute the adaptive capacity that we argued was essential for life. They are stimulation adjustment, stimulation norm adjustment, and coping-skill development. *Stimulation* refers to the average volume of inputs, both material and informational, that a person has to deal with at each point in time. The *stimulation norm* is that current level of aggregate stimulation to which the person expects or desires to be exposed. *Coping skills* are the strategies and actions necessary to minimize the deviation between norm and stimulation. To illustrate these concepts, recall Table 3, or consider a person's food intake. The stimulation norm reflects physiological need (calories, protein, fat, carbohydrate, minerals, and vitamins) modified by taste and availability. Actual food intake is the stimulation. Coping skills in this example range from an awareness of the need for food, the earning potential to buy it, and the skills to prepare it, to the muscle coordination involved in lifting a fork and sipping coffee.

Stimulation Adjustment. Due to the inherent difficulty in combining differing types of stimulation, we assume their effect to be roughly additive and concern ourselves only with the volume of stimulation, not the content. An intense, but brief, bout of loneliness or physical pain may not by itself lead to increased alcohol use, whereas both combined might. This amounts to an assumption of optimal content composition, and hence, we underestimate the difficulties a person will have in reality. We must stress, however, the distinction between our concept of stimulation and that of the psychologists' stimulus. Our unit of analysis is an averaged stream of stimuli, called *stimulation*, and not the discrete stimulus.

Much of the stimulation one is exposed to is given: noise, light, smells, taste, thoughts, job pressure, and so on. But one is not a passive object in a stream of stimulation: "It is only by the exploitation of the spatial and temporal orderliness in their environment that organisms survive in a world governed by the Second Law of Thermodynamics" (Johnston, 1979, p. 1). Johnston went on to say that the brain "is organized to seek out and to store the temporal relationships between events." The seeking goes so far as to impose subjective orderliness on events that are truly random (Dochin, 1979, p. 55). "People, to some extent, can affect the particular demands to which they will be exposed and the pace of exposure" (Mechanic, 1973, p. 168).

Consequently, we hypothesize that one type of adaptive response to a deviation of stimulation from its norm is the attempt to influence

stimulation. To facilitate the discussion, consider Figure 11. Stimulation arrives as a stream of inputs to the person. A backlog, or level, of stimulation builds up and must be dealt with by a portion of the available coping skills. Routine matters are dealt with through the basal reaction rate. Assume, for the moment, that stimulation norm and coping skills are constant. Should some exceptional event occur, the stimulation arrival rate increases. The event may be anything from the news of the death of a grandchild to substantial winnings at a casino or the destruction of one's home by a storm. Since the basal reaction rate depends only on the level of coping skills, which we assumed to be fixed, the level of stimulation builds up. Consequently, a deviation from the norm starts to build up. That deviation we call *stress*. Stress mobilizes the portion of coping skills that is not used for basal reactions, in order to provide emergency adjustments. As a consequence, the overall stimulation reaction rate, which is made up of basal reaction and emergency reaction, increases. This increase reduces the backlog of stimulation and continues to do so until the deviation from the norm has disappeared. Only then does stress subside and the coping-skill reserve become dormant again. Functionally similar is the attempt to reduce the rate of new stimulation, for example, as one who removes himself or herself from the company of others after some tragic event. In both cases, the level of stimulation is reduced so that it is more in line with the norm.

Our concept of stress is central to our theory, and to preempt misunderstandings, we look at it in some detail. Functionally, any

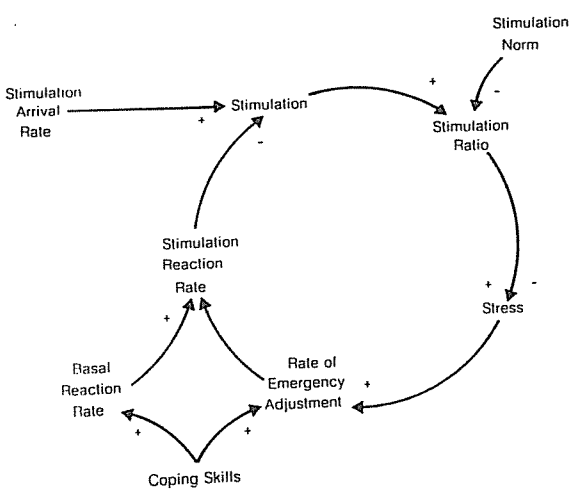

FIGURE 11. Mechanisms of stimulation adjustment.

deviation between stimulation and its norm generates stress in the model. Since the deviation can be caused by changes in stimulation, and since changes are only volume changes, the existence of stress is not necessarily negative, as colloquial English implies. The examples we gave—storm, death, and lottery win—include both positive and negative events. The death of a grandchild, for example, is a tragic event and forces readjustments in the life of all family members. In contrast, a win in the lottery is a cause for celebration. Nevertheless, adjustments to the new wealth have to be made: the bigger the win, the more disruptive the changes. Additionally, a deviation can also be caused by a reduction in the stimulation arrival rate, as in sensory deprivation experiments (or solitary confinement), which also result in stress. In short, our use of the term *stress* is close to Selye's (1956) concept, extended to allow psychological and social causes of the deviation as well (Levi, 1972; Lazarus, 1966). To translate deviations into stress, we use the tabular relationship shown in Figure 12 (cf. Selye, 1974, p. 32). The horizontal axis is the natural logarithm of the ratio of stimulation to stimulation norm. We chose the logarithmic relationship so that the stress produced when stimulation is twice its norm is the same as the stress produced when the ratio equals one-half. And since "log(l/u) = − log(u)," this condition is elegantly fulfilled by taking the logarithm of the ratio. Since both understimulation and overstimulation lead to stress, we employed the *U*-shaped curve. We also assume that beyond a certain level, further deviation does not produce

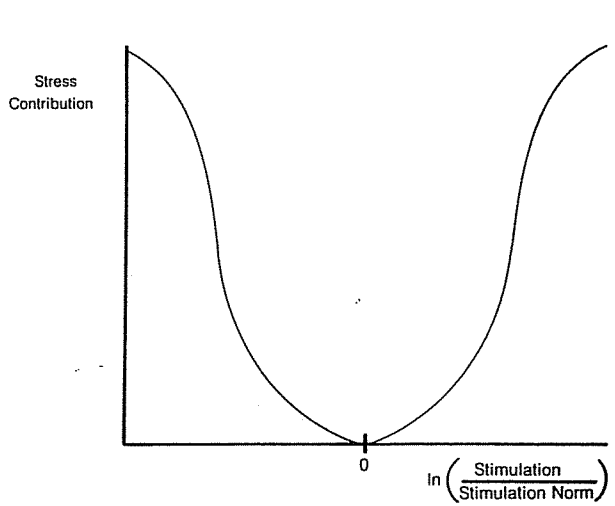

FIGURE 12. Stimulation and stress.

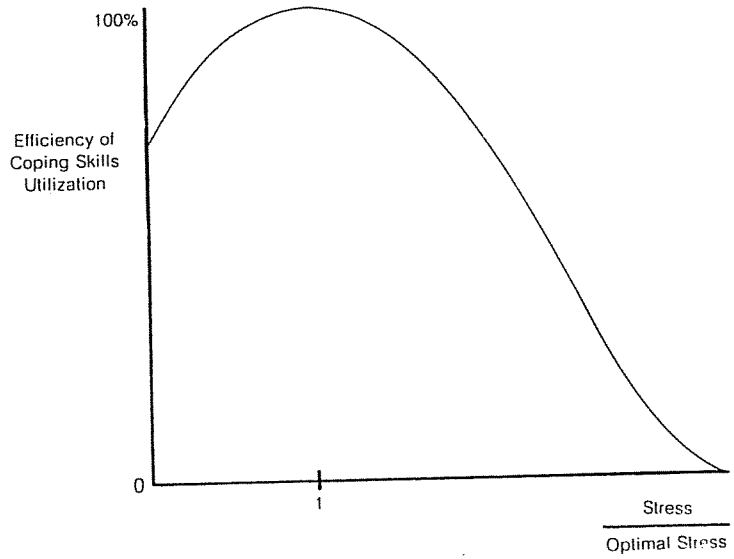

FIGURE 13. Stress and utilization of coping skills.

more stress, and hence, the curve flattens out when stimulation exceeds about twice its norm.

A second concept that merits further elaboration is the use of available coping skills. Recall that stress mobilizes the emergency reserves of the coping skills. It is common wisdom that a little stress is useful; it keeps you on your toes. But when stress starts to mount, it causes suffering. Empirical evidence for this phenomenon has been provided by Vince (1949, p. 35), who demonstrated that with increasing information input, the frequency of response increased monotonically. The frequency of *correct* responses, however, reached a maximum only to decline back to zero again as the amount of information continued to rise.

To incorporate this idea into our theory we developed the tabular relationship between stress and utilization of emergency coping skill that is shown in Figure 13. The horizontal axis is the ratio between stress and optimal stress, the latter being determined by coping skills and self-esteem. When stress is at the optimal level (that is, when the ratio between them is one), emergency coping skills are, when needed, used most efficiently, at 100%. Both below that level (that is, to the left of the optimal level) and above it, underutilization of emergency coping skills occurs.

Stimulation Norm Adjustment. The assumption of a constant stimulation norm in the previous section is, of course, invalid. In reality, norms are as subject to change as stimulation. As a prime example, we cite Festinger (1957), who developed an intricate theory of cognitive-dissonance reduction by way of norm adjustment. Miller (1978, pp. 448–464) reviewed Festinger's contribution and extended the idea of norm adjustments to other levels. Accordingly, we assume that a second adaptive response is stimulation norm adjustment (Figure 14). As before, a deviation between stimulation and norm gives rise to stress. Stress, in turn, mobilizes part of the available coping skills to alter the norm so as to reduce the original deviation. Again, the utilization of emergency coping skills responds to stress as shown in the tabular relationship of Figure 13.

Which adjustment mechanism predominantes depends on the fraction of coping skills allocated to each task: basal reaction, stimulations, and stimulation norm adjustment. If the allocation is 80:10:10, then most is tied up in basal reaction, and the emergency reserve, 20% of the total, is split evenly between stimulation and norm adjustment. As a result, this person is as likely to attempt to change norms as she or he is to attempt to change stimulation in response to stress. If the allocation is 95:2.5:2.5, then the person is wise to diligently avoid all potential sources of deviation, for only 5% of coping skills are reserved for emergency measures. If the emergency reserves are split 100:0 between stimulation and norm adjustment, then the person will never change his or her norms; he or she is a rigid dogmatic. On the other hand a 0:100 split indicates that the person takes whatever comes his or her way as the gospel truth. It follows that any particular person is somewhere between these polar extremes.

Coping Skills. So far, we have assumed coping skills to be constant. Again, this is not a realistic assumption; it is relaxed in this section.

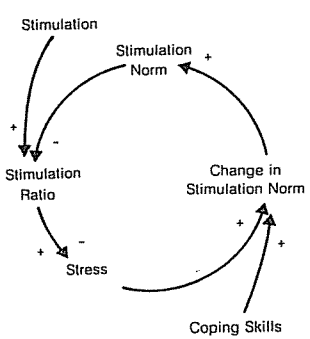

FIGURE 14. Stimulation norm adjustment.

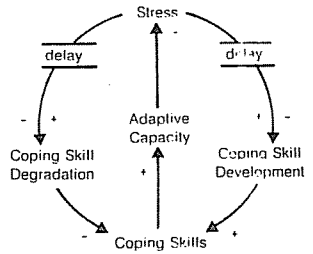

FIGURE 15. Stress and coping skills.

Coping skills can be acquired and lost. Growing into an adult, one learns to perceive and interpret stimulation so as to enhance coping skills. Once acquired, skills must be practiced to be maintained. A simple example is the gradual loss of the foreign language learned in high school. An extreme example is that of the Padaung tribe in Burma, where women wear metal neckbands as a status symbol (Keshishian, 1979). The bands serve as a firm support for the head, so much so that the neck muscles that normally keep the head upright atrophy, a fact of which the Padaung are well aware, since the punishment for adultery is the removal of the bands. The woman's own muscles can no longer support the head, and she suffocates.

The list of examples can be extended at will. Suffice it to say that some stress is necessary to develop and maintain coping skills; yet too much stress will accelerates the degeneration process. Consider Figure 15: at first, stress increases the rate of coping-skill development and

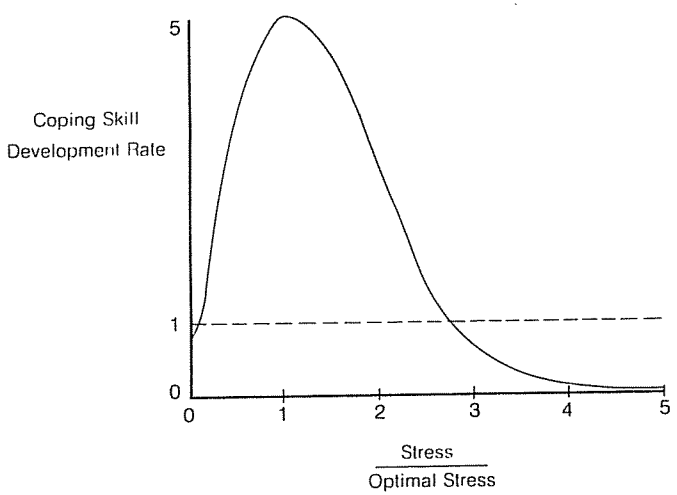

FIGURE 16. Stress and coping-skill development.

decreases its degeneration rate. Both effects combine to increase the level of coping skills, to boost adaptive capacities, and thus to reduce stress. The process takes place over a very long time, as compared with emergency adjustments, and hence the insertion of explicit delays into the causal links. The relationship between stress and coping-skill development is shown in Figure 16. As in Figure 13, the horizontal axis is the ratio between stress and optimal stress. As stress rises from zero, the development rate also rises to a maximum value when stress equals optimal stress, then drops off to zero as stress continues to increase. The relationship between stress and degradation rate shown in Figure 17 is a mirror image of Figure 16. Up to the optimal stress level, coping-skill degradation falls off; after that, it increases significantly. In Figure 18 we superimpose Figures 16 and 17. One can now deduce the development of the level of the coping skills themselves. At zero stress (that is, when stimulation is equal to its norm), the degradation rate exceeds development, and hence, new development fails to make up for the continuous degradation of skills. Consequently, the level of coping skills falls. As stress moves toward the optimal level, the difference between development and degradation rates increases. As a result, more skills develop then degenerate, and coping skills increase. As stress moves beyond the optimal level toward Point A, the difference between development and degradation remains positive, but less and less so. The level of coping skills grows, but more and more slowly. Once one moves beyond Point A, degeneration surpasses development at an accelerating rate: coping skills decline faster and faster. Figure

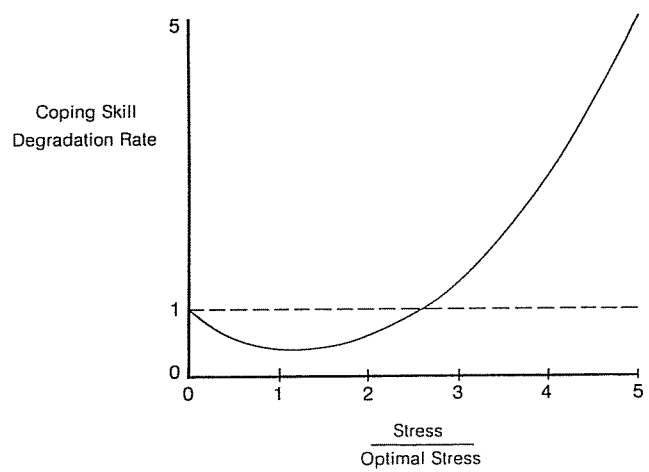

FIGURE 17. Stress and coping-skill degradation.

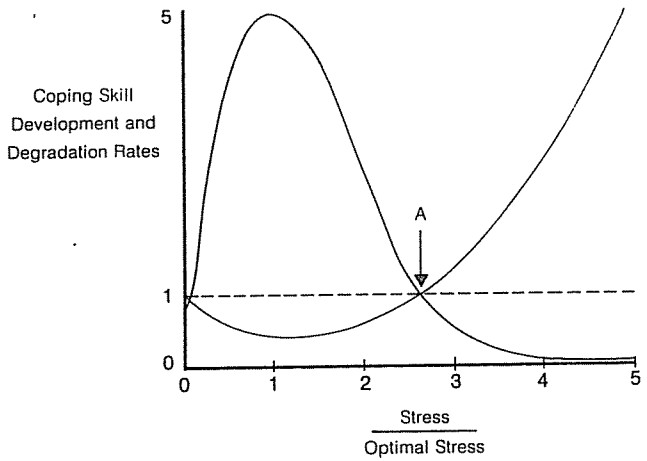

FIGURE 18. Development and degradation of coping skills.

19 plots the net rate of change (that is, development minus degradation rate) and the level of coping skills. It very clearly illustrates Selye's (1974) concepts of eustress, diminishing returns, and distress. "Mastery of stress is not a simple repertoire: it is an active process over time in relation to demands that are themselves changing" (Mechanic, 1973, p. 168).

One needs stress to build up coping skills, and coping skills are required to eliminate future stress. Hence, "complete relaxation of

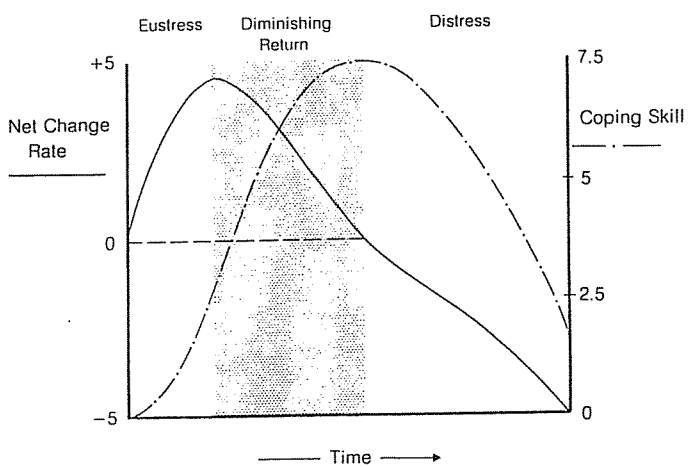

FIGURE 19. Net coping-skill change rate and the resulting level.

tension as in sensory deprivation experiments is not an ideal state . . . the psychosocial organism needs an amount of tension and activity for healthy existence" (Bertalanffy, 1968, pp. 209–210).

Self-Esteem

By *self-esteem*, we mean the estimation of one's own worth. It evolves in response to the way a person copes with the demands of life. Successful coping leads to high self-esteem; a string of failures tends to lower one's opinion of oneself. What, though, is success other than keeping stress below the optimal level? Seen from this perspective, failure is stress above and beyond the optimal level. Hence, a stress ratio between zero and 1 increases self-esteem; a ratio of 1 and above decreases it (Figure 20).

Some people are more influenced by external events than others. The concept of field dependence has been developed to characterize the degree of susceptibility to external control. Accordingly, we hypothesize two types of self-esteem: one to represent someone entirely guided by external cues (externally determined self-esteem) and the other for internally guided people (internally determined self-esteem). Any particular person is somewhere between those extremes. The exact location is determined by his or her field dependence. High field dependence puts the person closer to externally cued people, and low field dependence (that is, in our terminology) puts him or her closer to internally cued people. Self-esteem is the contribution of internally

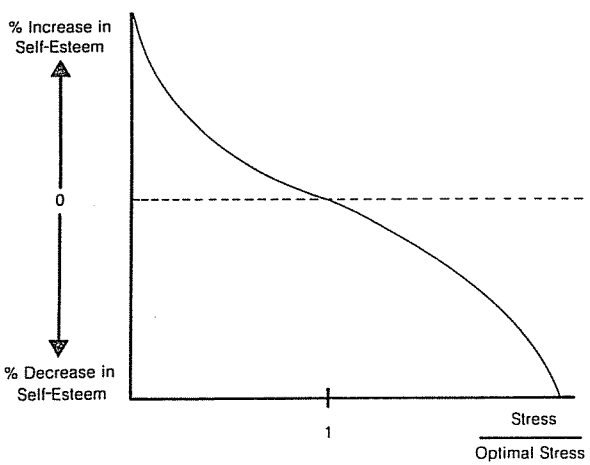

FIGURE 20. Fractional change in self-esteem.

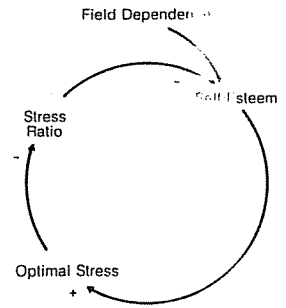

FIGURE 21. Self-esteem and optimal stress.

and externally determined self-esteem, each weighted by the person's field dependence.

In the model, self-esteem has two effects. First, it influences the level of optimal stress. This influence reflects the fact that high self-esteem leads people to be more daring; they tend to anticipate challenges in the knowledge, based on past success, that they will probably cope with them. Since stress is one of the determinants of stress ratio, a feedback loop has been closed (Figure 21). Assume the stress ratio to be below 1, which occurs when stress is below its optimal level. This raises self-esteem, which, in turn, increases optimal stress. Because optimal stress is the denominator of stress ratio, a rise in optimal stress lowers the ratio further: a positive loop. If this were the only feedback loop governing self-esteem, then it would keep rising or falling forever, depending on the original change.

There is, however, a second effect caused by self-esteem. It not only makes people more prepared to face challenges, it also makes them seek out more stimulation. That is, they increase the variation in the stimulation arrival rate, given that they have the necessary coping skills to do so. Wider fluctuations in stimulation, however, raise stress. Consequently, the stress ratio rises, thus checking the growth in self-esteem: a negative loop (Figure 22).

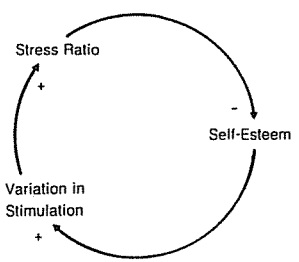

FIGURE 22. Self-esteem and stimulation variation.

These two loops also work in the opposite direction. When self-esteem starts to fall, it lowers optimal stress, causing self-esteem to fall even further. People try to stop the decline by reducing the variation in their incoming stimulation. In extreme cases, they may become totally apathetic and may neglect basic hygiene and even essential living functions, such as eating.

What are the implications of these two loops for the capacity to respond adaptively? Assuming adequate coping skills, the loops tend to stabilize the organism in the face of continuously changing demands. But when coping skills deteriorate, they exacerbate an already precarious situation by letting self-esteem reach dangerously low levels, complicating any attempts at improving the situation.

Homeostatic Capacity of Alcohol

The capacity to respond adaptively is the essence of life. Without it, people could not survive, and our species would not even be here. Since alcohol has been consumed since time immemorial (at least, since it was warm enough to grow plants and ferment them; Klausner *et al.*, 1980), it has been suggested that the reason for the popularity of alcohol can be found in the way it interacts with people's capacity to adapt. So far, we have provided details only of our assumptions about the functioning of the adaptive system. We noted that some stress is necessary for life, hence good, but that too much is dangerous. What, though, are the options for someone who is under a lot of stress? First, she or he can sit back and do nothing. Since all stimulation has a random element in it, one can always hope that the cause of the deviation will go away by itself. Suppose, however, that it persists. The person is in distress, which is the region where the coping skills allocated for emergency adjustments are not used very well (see Figure 13) and the level of coping skills degenerates faster than it is restored (see Figure 18). Waiting, then, is a bad strategy, since things can only get worse. Figures 13 and 18, however, suggest a way out. If the person could distort the perception of the deviation just enough to move into the region where stress was about optimal, then she or he could use her or his coping skills more effectively. In addition, she or he would actually build up the level of skills at the fastest rate possible. There are many ways to achieve the distortion: team work, meditation, concentration, drug intervention, and alcohol consumption are just a few examples. What else is concentration if not a deliberate focusing on a subset of stimulation at the expense of all else? It narrows the spectrum of incoming stimulation. Each of the strategies has its pros and cons. Meditation is a skill that has to be specially learned, as does correct drug intervention, where type of drug and dosage have to be carefully considered. Consuming alcohol, on the other hand, is rather easy. Liquor is readily available, and its use requires only skills that everyone already possesses. The major drawback of alcohol is the crudeness of the intervention. It is very difficult to drink just enough to move the stress ratio to the level of optimal stress; often people drink too much, thus moving the stress ratio to zero and losing any beneficial impact of moving to optimal stress. Additionally, the effect of ethanol wears off rather quickly, leaving only the hangover, which, however mild, adds to the level of stimulation. As we shall see later, there are other reasons to drink, but the ability of alcohol to reduce the perception of stress is one of the most powerful ones. "The widespread integration of drinking in cultural rituals and ceremonials is further evidence of an adaptive function of drinking" (Barry, 1973, p. 260).

Pharmacologically, ethanol is generally classified as a depressant. There is evidence, however, that it also has stimulating effects, especially at low doses. The stimulus is partly the result of its disinhibitory action and partly due to direct effects (Wilder, 1967, p. 244; Barry, 1973, p. 252). Thus, the effect of ethanol would seem to be state-dependent. An elated person who drinks will feel depressed, whereas a bored person will feel agitated after a drink. This finding fits very well with our own hypothesis, namely, that moderate ethanol consumption enhances the adaptive capacity of organisms, and hence, it possesses a homeostatic capacity. Wilder (1967) strongly supported this view by maintaining that alcohol effects are such that they will lift you up when you are down and they calm you when you are upset (p. 244). Alcohol is regarded as a great restorer of normality.

As we mentioned earlier, other behaviors can achieve the same distortion of the perception of stress. We believe that distress does not deterministically result in alcohol consumption. Rather, distress initiates a search, usually unconsciously, for a behavior to achieve the sought-after distortion. There are attributes of each behavior that influence the selection of one over the other. These attributes include price, availability, accessibility, social setting, acceptability, knowledge of effects, previous use, speed of action, and difficulty of use. All of these may be aggregated into the concept of a behavior's utility. One would expect that the behavior with the highest utility would be chosen most frequently. One must remember, though, that this utility is specific to the person, so that at any given time, one should expect a wide variety of behaviors in any particular population.

Here, by the way, is the leverage point of most public health or

policy measures. Higher alcohol prices affect the utility of alcohol consumption across the board. But price has a different relative weight in the utility consideration of a social drinker from that of an alcoholic, whose considerations are dominated by previous use. Thus, one may expect that alcohol purchase is more price-elastic for social drinkers than it is for alcoholics (Medicine in the Public Interest, 1979, p. 48). In our theory, which concerns itself with a single drinker, we do not consider in great-detail the search process that leads to the selection of a particular behavior. Instead, we aggregate the utility considerations into the tabular relationship shown in Figure 23. A particular person's utility considerations may yield Curve A. For him, a stress ratio above 1 increases consumption, and a ratio below 1 decreases it. Someone with a lower alcohol utility may respond as shown in Curve B. For this person, a given value for the stress ratio yields a smaller increase and a bigger decrease in consumption than Curve A would. On the other hand, a heavy drinker who has developed an alcohol dependency operates on Curve C: a given stress ratio translates into larger increases and smaller decreases than would either Curve A or Curve B.

Assume now, that the person is in distress. Curve A from Figure 23 translates this distress into an increase in average alcohol consumption. As consumption increases, the capacity to distort stress (what we have termed the *homeostatic capacity*) increases as well. Thus, perceived stress is reduced until the person is no longer in distress. This is a negative feedback loop that tries to augment intrinsic adaptive capacity

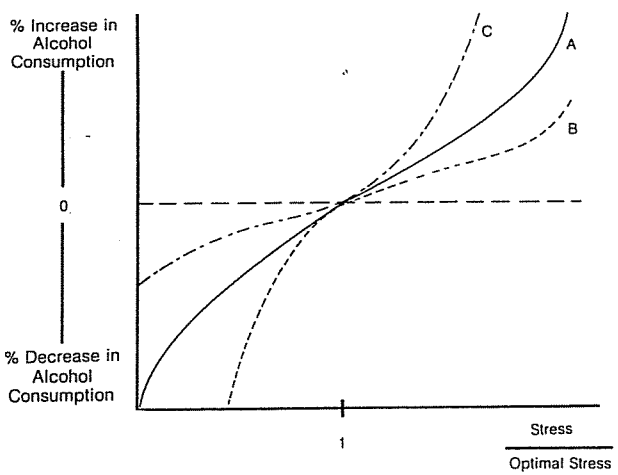

FIGURE 23. Utility considerations.

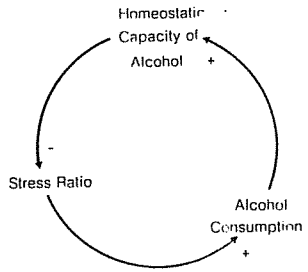

FIGURE 24. Homeostatic capacity of alcohol.

through alcohol consumption. From another perspective, the loop maintains alcohol consumption at a level sufficient to distort stress until it appears beneficial (Figure 24). One must not overlook the fact, however, that alcohol has strong side effects. They range from physiological effects that include everything from mild hangovers to delerium tremens, over financial difficulties, to health and social problems. Unfortunately, the delays involved in the positive and negative effects of alcohol are very different. The homeostatic effect takes place within minutes after intake, whereas delerium tremens does not occur until after years of steady drinking. It is often difficult to make the connection between today's benefits and next year's difficulties, especially since heavy alcohol consumption impairs the ability to consider the long run. Only a person with a long planning horizon is able to make that connection consistently. Yet whether or not the person is aware of it, each suppression of stress is payed for by a contribution to the eventual collapse of the system.

Physiological Adaptation

The presence of ethanol represents a deviation from the body's physiological (ethanol-free) norm. This process is in accordance with Miller's (1978) concept of hierarchies of systems, each having its own norm, or range of stability, as he calls it (p. 106), and each having regular and emergency adjustment mechanisms at its disposal. Consuming alcohol to improve overall performance is done at the cost of disturbing the physiological equilibrium of the system. Once disturbed on that level, those components use their adjustment capabilities to reduce the deviation at their level. "[The] addictive state is characterized by a newly established homeostatic equilibrium, so that the organism functions 'normally' under these conditions. Disturbance of the homeostasis by withdrawal of the addictive drug causes pathophysiological changes which manifest themselves as withdrawal signs" (Herz and Schulz, 1978, p. 376).

Functional Adaptation. One aspect of adaptation is a reduction in the capacity of a given amount of alcohol to suppress stress. Or, expressed from a different viewpoint, as the organism adapts to the presence of ethanol, larger and larger amounts are needed to achieve the same effect (Okamoto *et al.*, 1977; Fig. 2; Goldberg, 1943). To see the behavioral implications of this development, consider Figure 25. The loop formed by the variables of stress ratio, alcohol consumption, homeostatic capacity, and their causal links replicates Figure 24, a negative loop controlling alcohol consumption. In Figure 25, we have added physiological adaptation, which develops gradually, hence the delay. Once developed, the more adapted the organism, the less effective the homeostatic capacity. Consequently, adaptation gradually but consistently weakens a major control mechanism that limits consumption. The outer loop is positive. Recall that positive loops are the ones responsible for the dramatic increase in consumption. They are the addictive cycles.

Metabolic Adaptation. The body begins to metabolize ethanol as soon as it enters the system. Oxidation takes place predominantly in the liver; there are no storage mechanisms for ethanol in the body, and the surface and pulmonary excretion rate is below 10% of the total (Lieber, 1977). Oxidation is proportional to the amount of ethanol in the body when it is below the metabolic capacity, and oxidation is equal to the metabolic capacity when it is above the latter. The exact pathways of metabolism are still a matter of some debate, but consensus exists that alcohol dehydrogenase (ADH), catalase, hydrogen peroxide (H_2O_2), and the hepatic microsomal ethanol-oxidizing system (MEOS) are involved (Rix, 1977, p. 1). The relative importance of alternative systems shifts during of the progression of alcoholism. This shift allows for variation in the effectiveness of the metabolizing process and may account for the fact that alcoholics are able to metabolize alcohol faster than nonalcoholics (Mezey and Tobon, 1971).

What, however, does a faster metabolizing rate mean? Metabolic

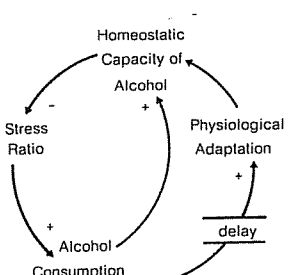

FIGURE 25. Adaptation.

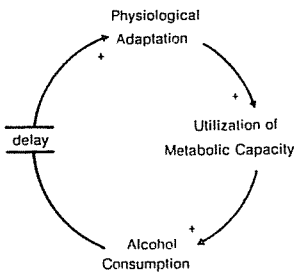

FIGURE 26. Metabolic capacity.

capacity is a purely physiological limit that remains after all other psychological and social constraints have failed. Though the metabolic capacity is determined by the structural integrity of the organism (Lieber and DeCarli, 1977), the varying utilization of the metabolic system makes the effective metabolic capacity a *variable* constraint on consumption. As adaptation increases the effectiveness of the metabolic capacity, it acts as a disinhibitory agent on consumption. It is part of the second positive loop that leads to excessive alcohol consumption (Figure 26).

Protracted Recovery Syndrome. A third result of physiological adaptation is the pathophysiology that is caused by the withdrawal of the addictive drug and that is terminated immediately by its readministration (Herz and Schulz, 1978, p. 376). The first and most visible expression of that process is the withdrawal syndrome. In humans, this syndrome lies "on a continuum of increasing severity, from acute hangover to delerium tremens" (Kalant, 1977, p. 57). It manifests itself as neuronal hyperexcitability, such as tremors, insomnia, irritability, hyperreflexia, seizures, and, most dangerously, delerium tremens. "The patient is in constant hyperactivity, agitated, disoriented and hallucinating vividly. There is an intense autonomic storm. . . . Hyperthermia, profuse flushing and sweating, and severe tachycardia result in cardian overload with the risk of cardio vascular collapse" (Kalant, 1977, p. 59). Kalant estimated the mortality rate of delerium tremens at around 2%.

Although even a delerium tremens episode is over within days, there is evidence that the early reactions to the withdrawal of ethanol are merely the tip of an iceberg that extends for months if not years into the patient's recovery phase. In an alcoholic, innumerable biochemical processes have adjusted to perform "normally" in the presence of ethanol, and it may take years for the readjustment to be fully achieved after drinking stops. Martin and Jasinski (1969) reported such a protracted abstinence symptomatology for heroin addicts, and Kissin (1979) extended the concept to alcoholics. Here, we use the term

protracted recovery syndrome to describe this development. Dynamically speaking, the organism has, under the influence of ethanol, established a new range of stability. To stay within the new range, it is necessary that ethanol be present. On cessation of drinking, a gradual and painful process has to take place as the organism returns to the prealcoholic state. Drinking during this process of recovery returns the organism to its accustomed, alcohol-bathed state. Hence, alcohol "normalizes" many body functions during this stage (Kissin and Hankhoff, 1959; Kissin, 1974, p. 8).

Functionally, the protracted recovery syndrome is another positive loop, another addictive pathway. Figure 27 illustrates this effect: alcohol consumed in response to an excessive stress ratio leads, after a delay, to adaptation. This, in turn, gives rise to the protracted recovery syndrome, which adds considerably to the stimulation that originally gave rise to distress. This addition to the stimulation is especially significant shortly after cessation, when it is very intense. But the syndrome's effects can also be important several months into recovery, when they are less severe, but completely unexpected. It also becomes evident from Figure 27 that the more the organism is adapted to alcohol, the more severe and the longer the recovery will be. This loop alone explains the observation that the probability of recovery declines as excessive drinking continues.

Figure 27 also suggests a way to avoid the pain of recovery. As Herz and Schulz (1978, p. 376) noted, readministration of the drug immediately terminates the pathophysiology associated with withdrawal. The patient is in a no-win situation: she or he drinks to stave off the pain of recovery, but each drink enhances adaptation even further, thus making any future recovery even more painful, hence less likely. It is worth pointing out that the causal relationships described in this present section are by themselves often powerful enough to sustain a high and increasing level of consumption. Many, if not all, of the problems that caused the original distress and its associated drinking

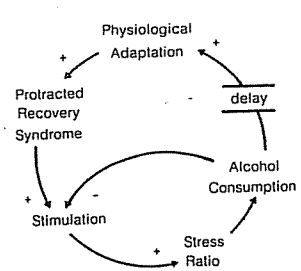

FIGURE 27. Protracted recovery syndrome.

may have long since gone, and yet fear of the pain of recovery is sufficient to sustain heavy drinking.

Diseases of Adaptation. The physiological adaptation process, like any other, has costs associated with it. Slowly but surely, adaptation is paid for with a deterioration in the structural integrity of the organism. These are, as Selye (1974) and Lumsden (1975) called them, the "diseases of adaptation." Since they are very long-term phenomena, usually developing over decades, we have aggregated the affected organs and functions into one concept called *structural health*. Most visible is the damage to the liver (Salaspuro, 1971, pp. 171–172). Just as serious is the degeneration of neural capabilities, ranging on a continuum from mild blackouts to the irreversible Wernicke–Korsakoff syndrome (Victor *et al.*, 1971; Ryback, 1971; Ryan and Butters, 1980).

We have incorporated structural health into our theory of alcoholism by acknowledging the normal aging process. We develop in the model an "age-specific structural health." This is modified by the extent of adaptation to alcohol and the severity of the stress that the person experiences. Hence, adaptation, itself a result of heavy consumption, causes the level of structural health to be lower than would be indicated for a healthy person of the same age.

Structural health determines metabolic capacity and is responsible for the slight decrease in consumption usually found in long-term alcoholics (Strel'chuck, 1976), when metabolic capacity is the binding constraint on consumption. Structural health also influences field dependence. Here, it increases the genetically predetermined level of field independence. Lastly, structural health plays a major role in the acknowledgment of a drinking problem (see below).

Adaptation Remission. The argument about controlled drinking for recovered alcoholics is an argument about the possibility of remission of adaptational changes originally induced by heavy alcohol consumption. Those maintaining that alcoholics will always be different essentially argue that adaptation to ethanol is not fully reversible. Other evidence suggests that this may be an extreme position. Begleiter and Porjesz (1977) recorded the visual evoked potentials (VEPs) of 18 rats at the visual cortex, the recticular formation, and the thalamus. After obtaining baseline VEPs, they intubated two-thirds of the rats with alcohol over a two-week period. Four and a half hours after the last intubation, they measured the VEPs every half hour for up to eight hours. Hyperexcitability was present in the rates using alcohol, but not in the controls (p. 213). Two weeks later, they administered a challenge dose to half the original users and to half the original controls. Marked hyperexcitability existed in the rats originally intubated with alcohol,

but not in the original controls (p. 216). After five weeks of abstinence, a challenge dose still showed some hyperexcitability in the original users, but not in the original controls (p. 217). This experiment suggests two things. First, once an organism is adapted to ethanol, the physiological effects of alcohol exist for quite some time. Second, even though they persist, the effects eventually show signs of remission. Had the rats not shown these signs of remission, one would have expected the challenge doses two and five weeks after intubation to elicit the same response as they did originally. When studying temporal patterns of voluntary alcohol intake in mice, Goldstein (1977), found a similar process at work: "These one-day 'breaks' [in consumption] apparently allow the previously acquired level of physical dependence to decay" (p. 539).

While the evidence is far from conclusive even for rodents, the general implications from the laboratory experiment seem plausible when extended to human subjects. Accordingly, we incorporate this aspect of alcoholism into our theory in a flexible way. The remission of adaptation is controlled by the "adaptation remission time." A value of 10 years, for example, indicates that within about 30 alcohol-free years, virtually all of the adaptational changes would have returned to the prealcoholic conditions. When more exact research findings begin to provide better numerical estimates for the remission times associated with different bodily and psychological functions, it will be possible to incorporate them into the theory by setting the adaption remission time at the new values. Should it turn out that there is no remission, this fact can be incorporated by setting the value extremely high, at, say, 1000 years.

Arguing strictly from the theory, we would expect the chances of an alcoholic patient's achieving successfully controlled drinking habits to be negatively correlated with the extent of adaptation existing at cessation. This extent depends on previous consumption. Hence, a long-term alcoholic has low chances of ever being a successful social drinker again. In his or her case, the slow remission process will cause a new drink to reactivate immediately all the addictive cycles for a long time to come, probably beyond his or her normal life expectancy.

Psychological Adaptation

Adaptation to the presence of ethanol is not restricted to purely physiological dimensions. Though the distinction may be merely a reflection of our limited knowledge, people do draw lines between physiological and psychological traits. In our theory, we balance the

two modes of adaptation, but psychological determinants do play significant roles. Whereas most of them are subsumed in the structure of adaptive capacities (see above), one direct influence on consumption merits special attention.

We mentioned earlier that distress merely sets the stage for maladaptive behavior and does not deterministically result in alcohol consumption (cf. Vogel-Sprott, 1972, pp. 492–493). What we hypothesized was a direct causation between distress and a search for behaviors to alleviate the stress. At the end of the search, some people select alcohol consumption based on their utility considerations. Consistent and excessive use, however, eventually influences these utility considerations in such a way as to shorten the search process and to reduce considerably the number of possible outcomes. This may best be called *habituation*. The alcoholic knows the drug intimately, probably has some within reach, and consequently chooses a drink in a given stressful situation where a novice may consider and eventually select some alternative.

This process of psychological adaptation modifies the tabular relationship between stress ratio and percentage change in consumption depicted in Figure 23. A beginner's utility may be represented by Curve A. Consistent alcohol consumption shifts the curve upward toward C. As a consequence, a given distress causes a progressively more substantial increase in consumption as the person follows the career path of an alcoholic. Tokar *et al.* (1973) have shown that alcoholics are more likely than normal controls to go to the bar, drink, smoke, and take pills whenever they feel helpless, depressed, angry, and anxious. Additionally, we assume that psychologial adaptation can also contribute to the prolonged recovery syndrome. The development and remission of psychological adaptation are incorporated into the model analogously to physiological adaptation.

Drinking-Problem Acknowledgment

Many heavy drinkers are able to maintain a steady, albeit very high, average consumption level for years. What is more, some even recover spontaneously (Lemere, 1953; Kendall and Statton, 1966; Pomerleau *et al.*, 1976).

Very little is known about the nature of the addictive process. The general belief for years has been that is is a rather deterministic progression from bad to worse (Jellinek, 1960). Recent concern about treatment evaluation (Smith, 1978), however, has led to a modification of this view. Baekeland (1977, p. 396), for example, made an attempt

to quantify the extent of spontaneous improvement. Mello (1972, pp. 259–263) questioned the deterministic progression hypothesis when she stated that "[no] empirical support has been provided for the notion of 'craving' by direct observation of alcoholic subjects in a situation where they can choose to drink alcohol in any volume and at any time by working at a simple task" (p. 259). She cited no less than 15 different studies with findings that support the conclusion that alcoholics exercise some degree of control over their average alcohol consumption.

It should, however, be no surprise that even alcoholics retain some control. Though people usually begin to use alcohol by choice, sooner or later their behavior becomes dominated by a small number of vicious circles. To stop drinking calls forth the pain of withdrawal and the destabilization of recovery; to continue drinking means temporary relief, but it implies increased problems in the future. In such a situation, there is no way out. One should expect that a person faced with this dilemma would see the connection between distress and alcohol intake, but to translate this insight into a program of abstinence is simply beyond the capabilities of the person. His or her coping skills were low to begin with and have suffered further from consistent alcohol abuse. The result is continuous vacillation between the desire to stop drinking and the realization that it is impossible. Nevertheless, these doubts result in stretches of abstinence that serve to constrain average alcohol consumption. Polich *et al.* (1980, pp. 179–182) stressed the fact that drinking patterns do indeed show great variation between short-term abstinence and heavy drinking. Often, the resources of the person are inadequate to bring about a significant reduction, but they are sufficient to keep average intake below what it would have been if governed only by physiological capacity.

The acknowledgment of a drinking problem is dependent on the information available to the person about the true situation, as well as the willingness to believe what he or she sees and feels. To translate any acknowledgment that might develop into action, the person also needs an ability to consider the future consequences of current actions. He or she needs a long planning horizon. As we mentioned earlier, drinking heavily is certainly not pleasant, but it is less painful over the short run than cessation would be. Any improvements come months, if not years, beyond the first withdrawal pains. To be able to wait that long, a person must consider the next several years, and not just days, when making decisions. Only then is a short-term cost (pain) worth a long-term benefit (recovery). Unfortunately, the planning horizon itself is detrimentally affected by drinking, since heavy alcohol use accelerates

the deterioration of many body processes, including neuronal functions (Seixas and Eggelston, 1973). Additionally, because of the planning horizon's dependence on coping skills, which have to be low to result in alcohol drinking, a high correlation exists between high consumption and a short planning horizon. Thus, at the very time when high consumption could be constrained somewhat by a long planning horizon, the decrease in coping skills undermines the effectiveness of this control considerably.

Social Credit

Social factors play important roles in any drinking pattern that is eventually established. Although we do not share the belief that drinking behavior is entirely caused by social interactions, we do acknowledge their importance.

Social interactions take place on several levels: family, friends, job, and community. To focus on the generic features of all these levels, we aggregate them into one unit. This social unit responds to an individual in a way that interacts with his or her coping skills. Problems that seem insurmountable when faced alone become easier when approached with the help of someone else, and trying to solve a problem with active obstruction from another person diminishes the efficiency of a given level of coping skills.

Calling on outside help is by no means automatic. Friends are a bigger help than hired hands, all else being equal. Thus, the effectiveness of help depends on the other's assessment of the worth of or personal concern for the one seeking support. The situation is a little similar to getting a bank loan. The better the credit rating, the more eager the bank is to help out. And the more social credit, the more willingly help is rendered. Current credit, though, is a measure of past performance. Our indicator of performance being the stress ratio (the relation of stress to optimal stress), credit is gained or lost depending on stress ratio. Several possible tabular relationships are presented in Figure 28. The exact shape depends on the social entity with whom the person interacts. If we use the family as an example, Curve A characterizes a spouse who values peace and quiet above else. The closer the person is to achieving no stress at all, the more readily the spouse grants social credit. Curve B may represent a more realistic spouse. He or she knows enough to expect some problems, to get suspicious when things are too calm, and to become rather hostile when they heat up too much. Curve C represents a masochist; the bigger the trouble, the higher the esteem

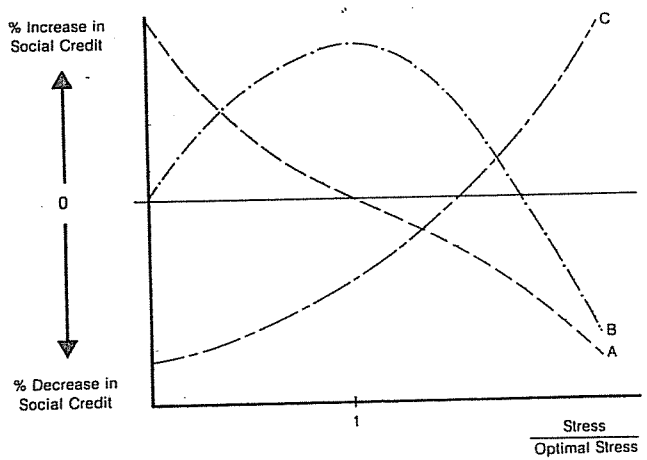

FIGURE 28. Fractional change in social credit.

for the person causing it. Which curve one chooses cannot be determined generically. It must be selected to fit each individual when the actual type and quality of his or her social environment is known.

Besides serving as an amplification of individual coping skills, high social credit accelerates the rate at which the drinker develops insights into the drinking problem by giving him or her valuable feedback about his or her condition, and it also affects alcohol consumption directly. Each drinker drinks within an environment that contains a social drinking norm. The relevance of this norm to a person depends on the social credit extended by the group holding the norm. High credit produces a better adherence to the norm than low credit. In the extreme case, no credit means that the social drinking norm is irrelevant to a person's drinking behavior.

The impact of the norm also depends on how strictly the norm is enforced. Assuming both social credit and enforcement to be constant, then the amount of socially encouraged drinking is important. Average alcohol consumption is, for example, higher in France than in the Muslim world, and the prevalence of alcoholism in France exceeds that in the Muslim world. This does not imply that the French are genetically prone to drinking alcohol; it suggests instead that the social drinking norms—hence, the restraints on individual drinkers—are different. Consider Figure 29. The bottom line is the Muslim norm for alcohol consumption, 0 g/week; the upper diamond-shaped line is the French norm, perhaps 100 g/week. A person consuming 30 g/week (the dashed line) would, if she or he were a Muslim, experience pressures to reduce

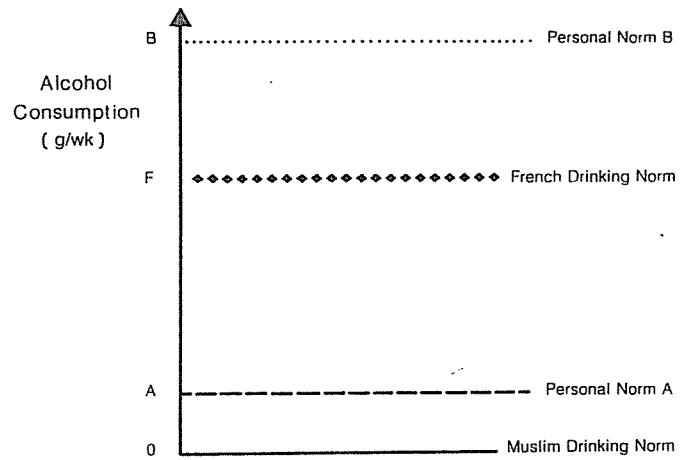

FIGURE 29. The effect of different social drinking norms.

consumption proportional to the distance between the two norms, that is, the distance OA. If that person were French instead, she or he would be subject to pressures to increase consumption, proportional to AF. And if the person drank 150 g/week, (the dotted line), pressures to reduce drinking would be a distance of OF (that is, OB − FB) greater if she or he were Muslim that if she or he were French. As a result the Frenchperson needs less of an individual pathopsychology to become a drinker than a Muslim: "The more widespread abuse becomes, the more exposed to drugs and drug-taking behavior the non-abuser will be, and the less predisposing personality disorders and social difficulties are required for an individual to be drawn in" (Bejerot and Bejerot, 1978, p. 106).

A causal loop diagram can best illustrate the role social influences play in our theory (Figure 30). Effective coping skills are a combination of individual skills and social credit. As effective skills increase, the stress ratio decreases, and, in turn, depending on the tabular relationship of Figure 28, social credit increases. This loop tries to maintain the stress ratio at the level where the spouse "rewards" most, that is, grants the most amount of social credit. Here, in fact, lies a fundamental cause of possible friction. Since the individual aims to maintain his or her stress at a level optimal for him or her, there is compatibility between him or her and whatever social unit he or she interacts with only if the reward structure is such that the social entity pursues the same goal. A spouse, for example, insisting on perpetual quiet, hence granting social credit only when the stress ratio is zero, creates contin-

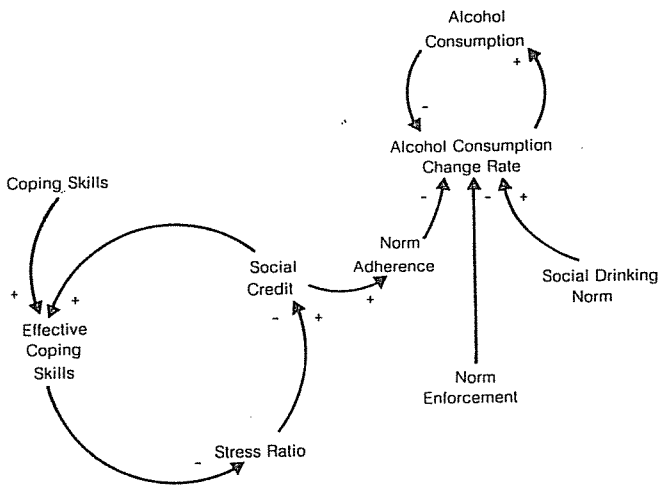

FIGURE 30. Social influences on drinking alcohol.

uous conflict. A family unit, depending on its values, may come to regard high alcohol consumption by the alcoholic as important and may resist very strongly his or her efforts to become sober.

Intimately liked to social credit is the drinking norm of that social unit. As social credit rises, adherence to the norm increases. This process, together with a strict enforcement policy, tends to keep the individual's average consumption in line with the social norm. Thus, the individual's reasons to drink more or less than the norm are overridden. Either lax enforcement or declining social credit, however, lessens the weight assigned to social consideration in the eventual determination of how much a person consumes. In this latter case, individual concerns dominate.

Model Summary

The theory we presented in this section represents only the first tentative steps toward understanding diseases as complex and intractable as alcoholism. We have striven to satisfy Sargent's (1973) six requirements for a theory of drinking: applicability, exhaustiveness, testability, universality, relevancy, and transparency. By adjusting social drinking norms and the normal metabolic capacity to account for genetic differences, our theory can be made applicable to all cultures and all societies. By reproducing all major drinking behaviors, our theory is exhaustive. By specifying each assumption and each relationship with (often necessarily assumed) numerical precision, the theory is testable. By incorporating, albeit generically, most genetic, physiological, psychological, and cultural causal factors, the theory is universal. By illuminating various treatment regimes, as we shall do in the next section, our theory is relevant. And by providing a manageable framework for a complex issue, it is transparent.

As no one will buy a city map that reproduces reality on a scale of one-to-one, a useful theory of alcoholism can not cover all aspects of reality. If it did, it would become as prohibitively complicated as reality itself. We have selected those aspects of reality we consider essential to develop a generic theory of drinking. Others will disagree with our choice and develop different theories. Far from being harmful, this is a necessary procedure at the present level of understanding of the disease.

INVESTIGATING WAYS TO DEAL WITH ALCOHOLISM

If our model is to be useful to therapists and policymakers, it must help them to prevent unacceptable drinking behavior and to intervene favorably in the remission process. We use the model to examine several generic responses to alcoholism.

Ways of dealing with alcoholism are efficient only if they improve the entire picture of the patient's health. Components of that picture are low alcohol consumption, no recovery syndrome, ample coping-skill reserves, high coping skills, near-optimal stress, high self-esteem, high social credit, and good structural health. Polich *et al.* (1980) rejected abstinence alone as a valid indicator of improved health: "Even a stable remission is not a guarantee of general rehabilitation, . . . even the alcoholic groups whose drinking problems have ceased or abated are considerably lower than the general norm in levels of social adjustment and psychological health" (p. 182). To give an indication of what the intervention policies should strive toward, we first present the "social-drinking-only" run.

To get from a causal loop diagram to a simulation run as in Figures 1, 3, 7, and 8, and as in the figures of this section, one must draw up a detailed flow diagram of the model. In contrast to a causal loop diagram, which provides only an overview of a part of the model, a flow diagram has a one-to-one correspondence with the complete model. One then transforms the flow diagram to equations. In this way, the computer is instructed to compute the value of each variable at every solution interval, which we set at about 2½ weeks of simulated time and

thereby trace the development of all variables over time. For the sake of clarity, we present only 8 of the almost 300 variables in the model.

In the current social-drinking run, we subjected a simulated person to a gradually rising input of stimulation and superimposed a random variation on the rising input. This progression simulates the effect of ever-increasing demands on a person as she or he matures from childhood into adulthood, parenthood, and old age. Figure 31 shows how a particular person may react to this stream of stimulation. All curves are deviations, in percentages, from the normal level. *Normal*

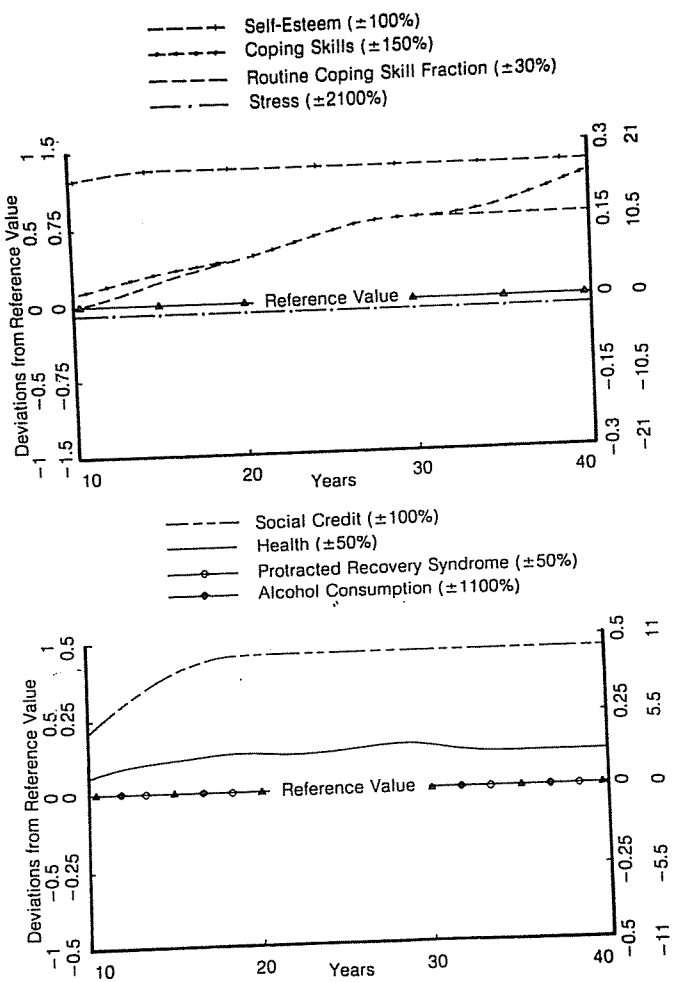

FIGURE 31. Social drinking: reference run.

has here no clinical or judgmental meaning. It only serves as a reference for comparison among runs. The horizontal axis is age, 10–60 years. The top half of Figure 31 summarizes the person's stress reaction. Stress is always close to the optimal level, hence coping skills and self-esteem rise above normal. The fraction of coping skills used routinely rises, but reserves remain adequate throughout life. The lower half of Figure 31 summarizes alcohol-related aspects. Consumption stays normal, that is, at the social drinking norm; health is better than age alone would suggest; social credit rises; and adaptation to ethanol is so low that the protracted recovery syndrome remains insignificant.

Next, we increased the stimulation impinging on the person. Figure 32 shows the result. In the top half we note that the stress ratio is initially slightly higher than the optimal level. As a consequence, coping skills are higher at age 13 than in Figure 31. Self-esteem is lower, but still considerably above its reference value. By drawing heavily and increasingly on his or her coping-skill reserves, the person is able to reduce the stress level to optimal conditions briefly during the early teens. This success is only temporary, and stress begins to climb steadily after that. As a result, coping skills and self-esteem drop off. The latter falls below its reference value at around age 18, the former at 20 years. Shifting the attention to the lower half of Figure 32, one sees that social credit is initially higher than in Figure 31 but soon begins a decline that never stops. Health is slightly below average, and alcohol consumption is still low by age 20. It is important to note that the inadequacy of the adaptive response mechanisms is fully established by age 20: coping skills, self-esteem, social credit, and health are by then all lower than they were in Figure 31. Still, alcohol consumption is only about twice the social norm, a value that is rarely, if ever, labeled alcoholic. And yet, the seeds for the eventual disease are firmly implanted. As one looks beyond age 20, the adaptive mechanisms continue their decline, until they reach dangerously low levels at age 40; alcohol consumption explodes during the latter half of the 20s and settles at a dangerously high level of about 10 times the social norm. The protracted recovery syndrome starts to play an ever-increasingly crucial role from age 30 on. At that stage, alcohol is mainly consumed to avoid withdrawal.

The policies that follow are all designed to prevent, or to help the person recover from, the situation depicted in Figure 32.

Prevention

All policies available to maintain drinking levels at social norms have three generic effects: they help achieve low stress, high coping skills, and low alcohol utility.

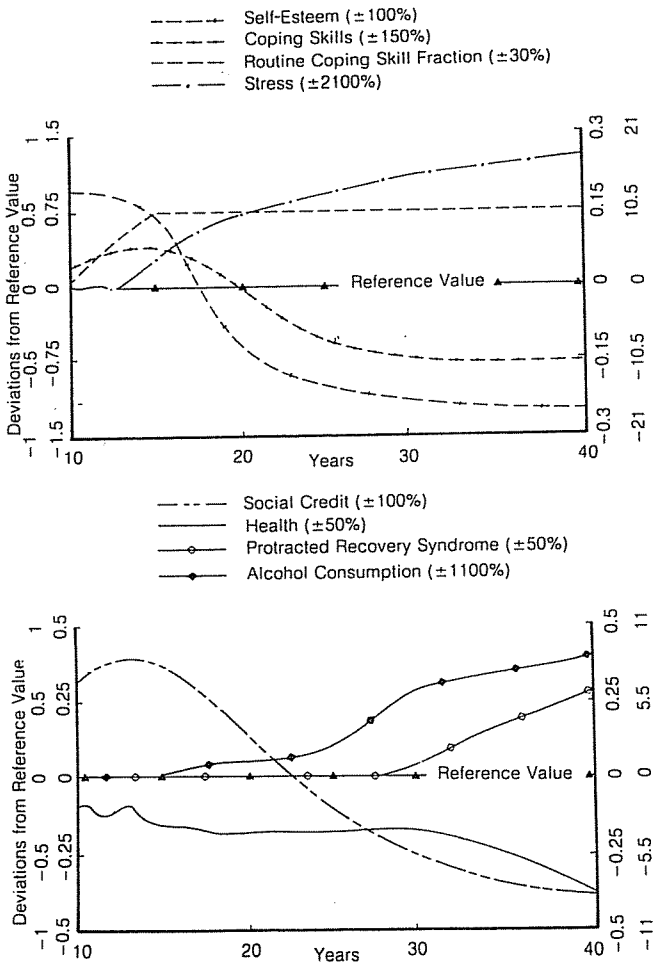

FIGURE 32. Alcoholism: reference run.

Low Stress

Although alcoholism can be caused by many different factors in reality as well as in our model (cf. Figure 7), personal distress always contributes to the probability of becoming an alcoholic. Therefore, the first line of defense is to create and maintain a state of low stress, with distress arising only occasionally and temporarily. This is more easily said than done. Even though people actively solicit and reject environmental and internal stimulation, control is limited. It is more a question of modifying a given stream of stimulation then being in complete

control of what goes on. Still, lower stress can mean the difference between social drinking and alcoholism. Merely reducing the percentage of increase and the variation in the stream of incoming stimulation from Figure 32 led, in Figure 31, to social drinking instead of alcoholism. At that level, coping skills are adequate to deal with the stimulation arrival rate. Consequently, stimulation remains in a healthy balance with its norm and rarely leads to a level of stress that is considered distress. In turn, the utilization of coping skills for emergency adjustments plus the development of coping skills themselves takes place in an orderly fashion, ensuring future resilience to outside stimulation. The person never needs to resort to alcohol in an attempt to suppress distress, or any other maladaptive behavior for that matter, and remains a social drinker throughout life.

High Coping Skills

A second strategy is to take the stream of stimulation as given and to concentrate on ways to enhance the volume and efficiency of the coping skills at one's disposal. In such a situation, when problems arise, the person is better equipped to deal with them. Higher coping skills can be gained in two ways: (1) increasing the volume and (2) improving the acquisition rate of new skills. To put it simplistically, the first is analogous to giving someone a meal, and the second is like teaching someone how to cook. A higher volume of skills and a faster assimilation rate enable the person always to stay ahead of the ever-increasing stimulation arrival. Again, normal adaptive skills suffice, without the aid of alcohol to suppress deviations. Consumption, for a social drinker, stays low enough to that he or she is not drawn into the addictive cycles and into disaster. However, should the rate of stimulation arrival drop, or even just level off, the person would be overqualified because of his or her efficient coping-skill assimilation habits, and there would be a danger that he or she would use alcohol to suppress the resulting boredom.

Low Alcohol Utility

The third preventive strategy is to lower the utility of alcohol consumption. This strategy leaves stimulation and coping skills as they are but strongly discourages the selection of alcohol as the behavior of choice under distress. Lowering the utility of alcohol can be done by raising its price, restricting its accessibility, reducing its appeal as a desirable drink, and so on. Lowering alcohol utility, however, inevitably raises the utility of other behaviors, for example, drug taking. Hence,

it may be relatively easy to affect the number of people choosing alcohol, but merely by shifting consumer preference between various modes of maladaptive behavior. What is needed to solve the problem completely is to shift preferences to adaptive behaviors. Yet, this shift is extremely difficult. A primary component of all utility considerations is the speed of action and the obstacles associated with use. As a general rule, maladaptive behaviors are fast-acting and easy to employ. Drinking has an effect within minutes, and the motor movement involved in drinking alcohol is identical to drinking any other liquid. In contrast, meditation, also an adaptive behavior, takes months, if not years, to learn. Also, it cannot be learned just when needed; the skill has to be learned long before. It is an unfortunate fact that people tend to choose quick solutions that may turn out to be harmful in the long run, rather than waiting and investing in the development of lasting solutions. This trait seems widespread and is probably responsible for the extensive use of alcohol throughout most cultures. Often, the potential danger is minimized by very strict constraints on the occasion and the manner in which alcohol can be consumed. The constraints disappear, alcohol stays, and there is then a prevalence of use and abuse.

Our theory does not deal with responses to distress other than alcohol consumption. Lowering the utility of alcohol, therefore, seems like an unequivocally good strategy. In reality, however, one must be aware of the dangers of thereby encouraging other equally undesirable behaviors.

Figure 33 summarizes all three prevention strategies and repeats the alcoholism results for comparison.

Treatment

For some 10 million people in the United States alone, prevention of alcoholism comes too late, because they are already heavy drinkers (U.S. Department of Health, Education, and Welfare, 1974). Even for those who have not yet begun to drink, no set of preventive measures will be entirely successful; as a result, there will be a perpetual need for effective treatment.

Alcoholism is a disease of the entire organism. It affects, in varying degrees, the person's entire physiological, psychological, and social well-being. It is not a temporary aberration from the norm of an otherwise healthy person. It is a profound shift to a new and stable equilibrium centered on alcohol. Alcoholics as a group are rather stable and predictable, so much so that for a long time scholars tried to identify the alcoholic personality, not realizing that the limited and stable behavioral repertoire of alcoholics is mostly alcohol-induced: their-self

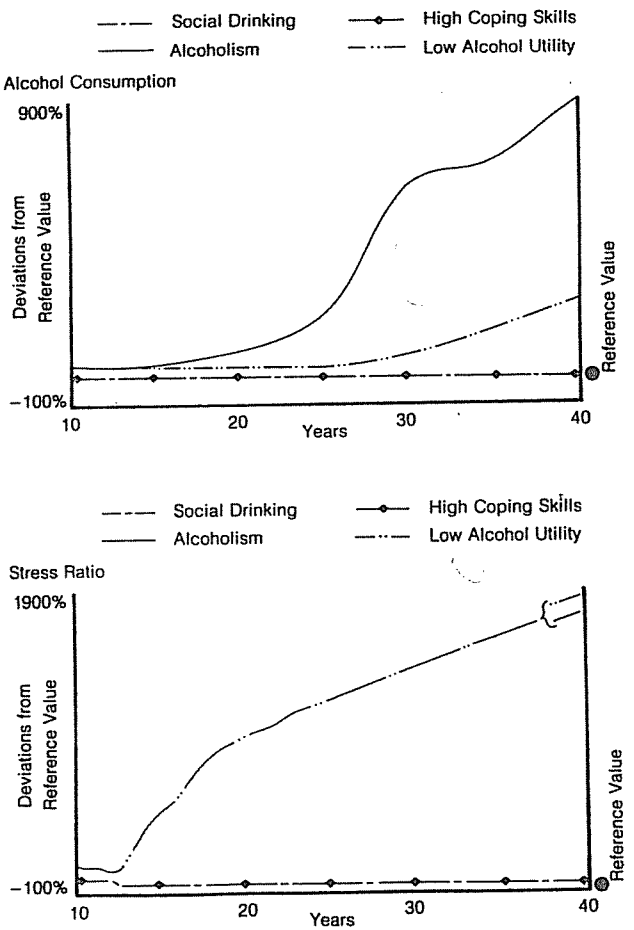

FIGURE 33. Generic prevention results.

esteem and social credit is low, their health is worse than indicated for their age, they have short planning horizons and low coping skills, and they drink excessively. Theirs is certainly not a happy existence, but changes are slow. They take place over the course of years and not days. To have an impact on such a sluggish system requires treatment on several levels for a prolonged period of time.

Generally speaking, treatment must cover six areas:

1. There must be "spiritual" therapy. Alcohol, which is the center of the alcoholic's universe, must be replaced by a consistent alcohol-free value system.

2. The new system must be made concrete and measurable. Standards derived from the new system must be established and internalized by the recovering alcoholic to provide concrete guidelines for daily behavior and to help her or him monitor her or his progress away from the alcoholic drinking.

3. Treatment must embrace skill training to enable the patient to meet the standards derived from the new value system. Otherwise, unbearable frustration may arise.

4. Treatment must alter the psychosocial reward system of the patient to favor the new value system and its internalization and implementation.

5. Treatment must deal with preaddictive and drug-induced organic syndromes.

6. Treatment must impress on the patient the importance of time delays and the relationships between the disease and his or her future well-being.

The list and its implications are substantial. But not all treatment must, or even should, be given by professionals. Nor does the intensity remain the same throughout the entire recovery phase. If we group all treatment into either physical or psychosocial rehabilitation, then a graph of the amount of treatment against time may look like Figure 34. As treatment is started, management of the acute withdrawal syndrome is of paramount importance. To try to teach a new value system to an alcoholic going through withdrawal would be a little like trying to convince someone who is starving of the intrinsic worth of the balance of power in a democracy. As physical conditions stabilize, though, progressively less effort out of the total is spent on treating the physical aspects of the disease, and more emphasis is placed on

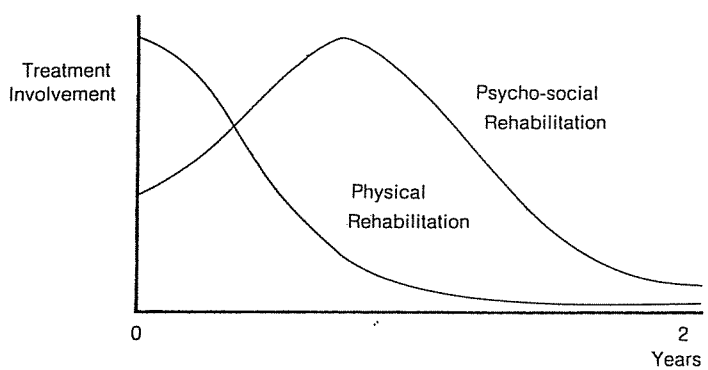

FIGURE 34. Treatment itensity.

extricating the alcoholic from his or her alcohol-centered lifestyle. Figure 34 is, therefore, only suggestive.

The goal of treatment is to improve the overall well-being of the patient. There can therefore be no specific guidelines as to what should be done and what should be avoided. Each treatment regime must be tailored to a particular patient (Glaser *et al.*, 1978; Chapter 17). Miller and Caddy's (1977) comment on the controversy of controlled drinking may serve as a reminder that it is not theoretical positions that are at stake, but the patient's health: "Few who work with problem drinkers would deny that there are some for whom abstinence is necessary. Few would now deny that controlled drinking is a reasonable goal for others. Our task for the future is one not of polemic, but of prediction" (p. 1003).

Treatment consists of weakening the power of the addictive cycles and strengthening the constraints on consumption. Tools to achieve these ends have been listed in Table 1. The applicability of these tools is discussed in detail elsewhere (Kissin, 1977a; Rix, 1977; Smith, 1978; Pattison, 1979). Here we examine only the efficacy of various generic treatment interventions on the patient presented in Figure 32.

The figures still have the same vertical scales, percentage of deviation from normal. The horizontal scale, age, runs from 30 years to 60 years, the latter being an arbitrary cutoff point for the simulation run, except for Figure 35, which runs up to 34 years. Figures 35–38 present the response of four selected variables (average alcohol consumption, stress ratio, social credit, and self-esteem) to the following interventions: aversive therapy; aversive therapy and coping-skills training; aversive therapy, coping-skills training, and family and self-actualization therapy; and all of the above plus functional alcohol replacement.

Aversive Therapy

If abstinence alone is the treatment goal, then techniques and therapies designed to reduce alcohol consumption are adequate. Figure 35 shows that by aversive therapy alone (for example, disulfiram), consumption falls below the social norm. Since we plot average consumption, an instantaneous cessation of alcohol intake will be averaged over some time. The slow decline in consumption is a consequence of the averaging process of our simulation technique, and not an indication of a treatment policy that seeks to reduce consumption slowly. If one were to monitor the first four years after treatment contact, which is long compared with usual practices, and which we have done in Figure 35, then one might call this policy successful. Consumption is down,

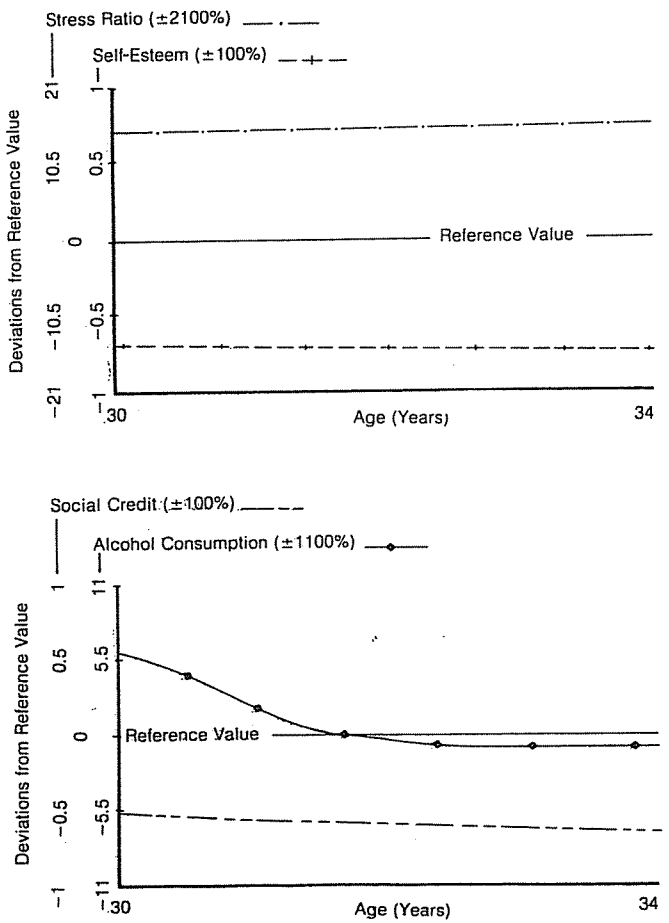

FIGURE 35. Aversive therapy; 4-year follow-up.

and although social credit, self-esteem, and stress levels show no improvement, they do stabilize. Our finding is in agreement with Polich *et al.* (1980, p. 182), who found in their four-year follow-up study that patients in remission fared better than nonremissive patients in general rehabilitation, yet did not show any significant improvement relative to their nonalcoholic cohort.

Unfortunately, even this relatively unimpressive result is lost if one looks at the next 30 years instead. Since we are not concerned with evaluating treatment facilities or treatment regimes and want instead to explore the possible development of an alcoholic patient given certain

treatment interventions, we must focus on the patient's expected lifetime, even if a 30-year follow-up study may seem unreasonably long. Since alcoholism takes 10 or sometimes 20 years to develop, and since data show relapse after a considerable number of years of abstinence, we must observe at least equally as long the patient's attempt to overcome the problem. As Figure 36 shows, seen from the patient's life-cycle perspective, the aversive therapy we assumed to last three years is not effective. The impression of stability gained after four years is illusory because of the short time interval of concern. Self-esteem and social credit continue their decline, stress continues to grow, and alcohol consumption rebounds after treatment is stopped. It finally increases

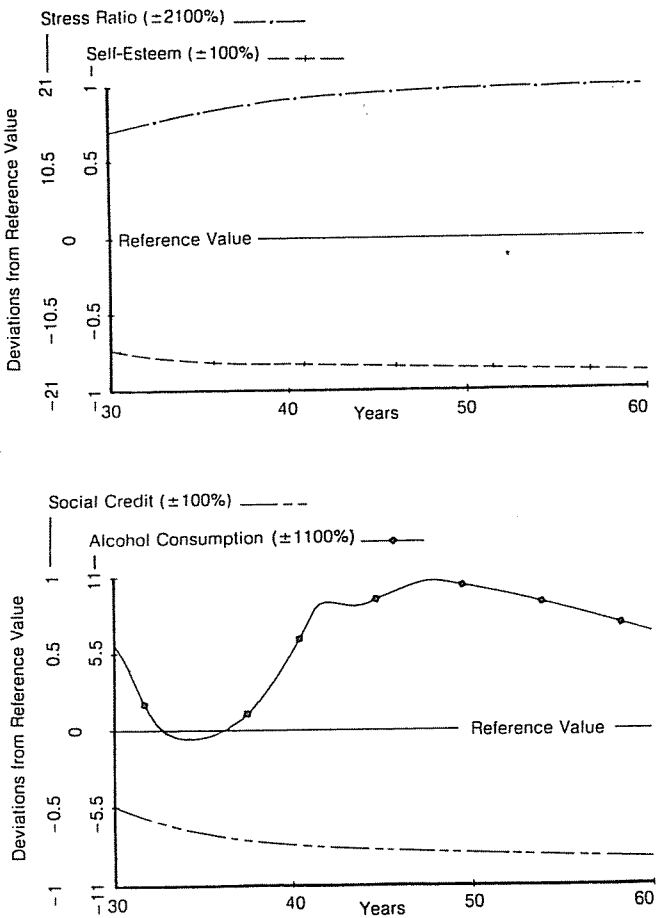

FIGURE 36. Aversive therapy; 30-year follow-up.

above the original level. Consumption falls off slightly after age 45 because of the increasing constraint imposed by the physiological limit of the metabolic system.

Aversive Therapy and Coping-Skills Training

We then simulated the effect of intensive coping-skills training during the time the patient abstained from alcohol. This approach would in reality be comparable to using disulfiram in order to buy time for the other therapy to take hold. To test this hypothesis, we simulated two therapeutic effects. First, we assumed that the treatment setting would provide short-term skill training: finding a job and housing, leisure training, and so forth. At the peak of the treatment intensity, this effect increased the coping skills available to the patient fourfold. Second, we assumed that in treatment, one would also attempt to improve the acquisition rate of new coping skills. In a run not shown, we found that this therapy, with the given three-year duration and intensity, failed to produce an improvement over aversive therapy alone. The progression of the disease as indicated by our four variables was identical to that shown in Figure 36. The inability of the therapy to lead to a lasting improvement can be traced to the fact that on entering treatment, coping skills are not only insufficient to deal with stimulation, but also insufficient to serve as a basis on which to improve.

Disulfiram, Coping-Skills Training, Self-Esteem, and Social Credit Improvements

Our next test consisted of providing help so as to specifically increase the self-esteem and the social credit of the patient, in addition to the treatment described above. Figure 37 shows the dramatic increase in self-esteem, above its reference value and higher than at any time since the early teens (cf. Figure 32). As treatment subsides, however, self-esteem falls back to its previously low level. The process is repeated for social credit, though the rise is slower, the peak lower, and the decline more gradual. The treatment package, however, begins to make real inroads into the problem. The stress ratio actually improves. At age 34, consumption is down, stress is declining, self-esteem is high, and social credit has been rising. This combination often leads to the classification of being "cured." But as the patient leaves treatment, one sees in Figure 37 that previous improvements were due to exogenous factors (i.e., treatment support), and not the result of internalized

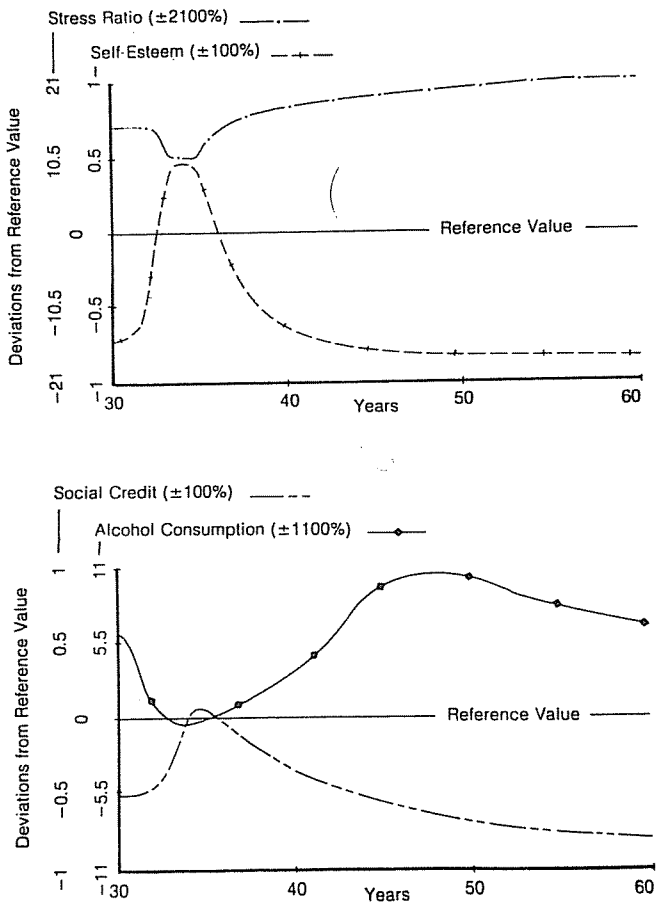

FIGURE 37. Added self-esteem and social credit improvements.

adaptive responses. Still, one notes that the average alcohol consumption curve rises much more gradually than before.

An Alcohol-Free Life

In this last treatment test, we simulated the effect of the patient being introduced to and accepting a functional alcohol replacement in addition to all other treatment. Alcohol had originally been consumed to distort the deviation between stimulation and norm. The development of the disease has ironically caused substantial increases in stimulation,

has eroded coping skills, and has thus presented the patient with a bigger problem than he or she had to begin with. Even if the physical dependency on alcohol abated during treatment, one would still expect the patient to resort to alcohol as his or her habitual response to distress. Thus, treatment must teach the patient ways to deal with stress adaptively. He or she must learn to choose behaviors that fulfill the function of suppressing stress as alcohol used to, but without the devastating consequences of alcohol. If this transition from one behavior to another is successful, the prognosis is favorable (Figure 38). Consumption remains low, and self-esteem rises as before, but it now

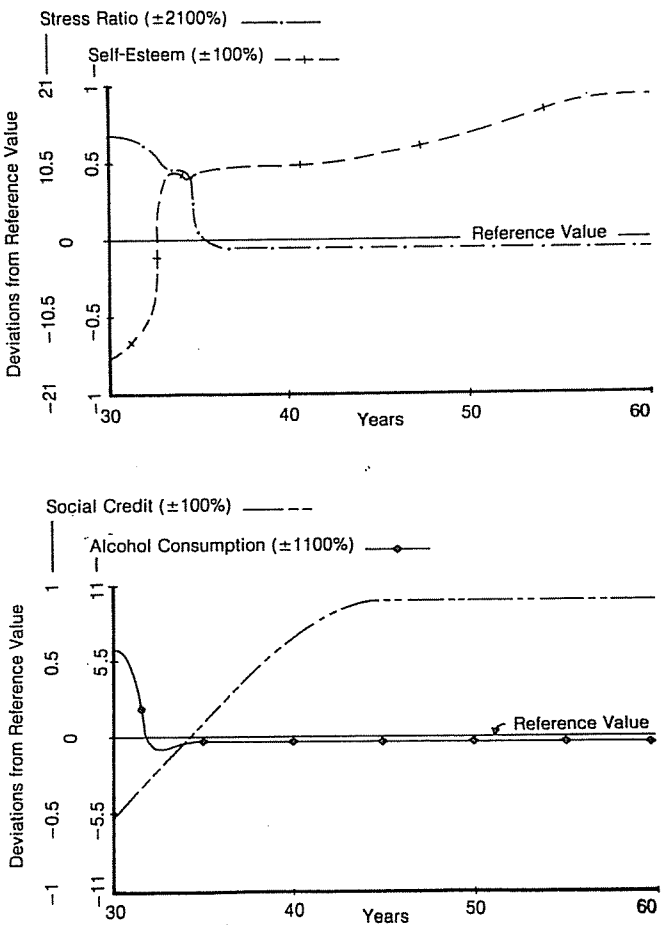

FIGURE 38. Alcohol-free life.

continues to rise, as does social credit. Effective stress is kept low for the duration of the simulation run.

CONCLUSION

Alcoholism is a unique disease. Unlike any other, it represents the tail end of the distribution of a behavior that is highly regarded in our society: social drinking. To succeed in correctly identifying alcoholics as early as possible and to treat them successfully, alcohol scientists must avail themselves of every means available. One of the current constraints in the field is the lack of a comprehensive and internally consistent framwork of explanation for drinking behaviors. Most of the pieces of the puzzle are known, but not the key to integrate them. To help overcome this difficulty, we have applied system dynamics modeling to drinking behavior.

Our theory will be criticized for being too simple, and rightly so. But "oversimplifications progressively corrected in subsequent development are the most potent or indeed the only means toward conceptual mastery over nature" (Bertalanffy, 1968, p. 178). As systems analysts, we rely on experts in each aspect of the disease to provide the empirical checks without which no theory can survive.

At present, our theory serves four functions. First, it demonstrates the applicability of simulation techniques to gaining a more comprehensive and a more consistent understanding of multifactorial diseases. As we have done with alcoholism, similar models can be built to deal with other addictive diseases, or depression, schizophrenia, or cancer. Second, the model can be instrumental in resolving many issues of contention. Its flexibility allows rapid insertion of alternative assumptions about variables, relationships, and numerical values. This flexibility means that conflicting views can be tested, and it lets their impact on overall model behavior be evaluated. Many assumptions rigorously tested in this way in a comprehensive framework will lead to untenable contradictions; yet others will not. Hence, the plausibility of some will increase at the expense of others. Third, in situations where two or more conflicting assumptions remain equally plausible after extensive testing in the model, or where precise numerical values turn out to be very significant, the model then suggests that those should be priority areas of further research. The theory can therefore be used to draw up a research agenda. Fourth, in its present form, the theory conveys large amounts of information in accessible form. Consequently, it can be used as a teaching tool.

ACKNOWLEDGMENTS

This chapter is based on work conducted within the Resource Policy Center, Thayer School of Engineering, Dartmouth College, in collaboration with Project Cork through support from the Kroc Foundation and the Commonwealth Fund.

Numerous colleagues contributed to the project, especially Barry Richmond, Ben Kissin, Ed Nace, and Fred Glaser. Peter Whybrow organized financial support; Ina Anderson gave editorial assistance; Joan Waltermire drew all the figures; Diana Golüke and Cindy Allen provided secretarial and other help.

REFERENCES

Alcaron, R. de, 1969, The spread of heroin abuse in a community, *Bull. Narcotics* 21(3):17–22.

Allport, F. H., 1955, "Theories of perception and the concept of structure," Wiley, New York.

Ashby, W. R., 1960, "Design for a brain: The origin of adaptive behavior," Wiley, New York.

Bacon, S. D., 1943, Sociology and the problems of alcohol, *Q. J. Stud. Alcohol* 4:402–445.

Baekeland, F., 1977, Evaluation of treatment methods in chronic alcoholism, *in* "The biology of alcoholism," Vol. 5, B. Kissin and H. Begleiter (eds.) Plenum Press, New York, pp. 385–440.

Bales, R. F., 1946, Cultural differences in rates of alcoholism, *Q. J. Stud. Alcohol* 6:480–499.

Barry, H., III, 1973, Cross cultural evidence that dependence conflict motivates drunkeness, *in* "Cross cultural approaches to the study of alcohol," M. Everett, J. Waddell, and D. Heath (eds.), Mouton, The Hague, pp. 249–263.

Beck, H., 1976, Neuropsychological servosystems, consciousness, and the problem of embodiment, *Behav. Sci.* 21:139–160.

Begleiter, H., and Porjesz, B., 1977, Persistance of brain hyperexcitability following chronic alcohol exposure in rats, *in* "Alcohol intoxication and withdrawal," Vol. 3b, M. Gross (ed.), Plenum Press, New York, pp. 209–222.

Bejerot, C., and Bejerot, N., 1978, Exposure factors in drug use, abuse, and addiction, *in* "Basis of addiction," J. Fishman (ed.), Abakon Verglagsgesellschaft, Berlin, pp. 89–118.

Bejerot, N., 1972, "Addiction, an artifically induced drive," Thomas, Springfield, Ill.

Bertalanffy, L. von, 1968, "General systems theory," Braziller, New York.

Bethe, A., Fischer, E., and Goldstein, K., 1931, Die Anpassungsfahigkeit (Plastizitat) des Nevensystems, *in* "Handbuch der normalen und phatologischen Physiologie," Vol. 15/2, A. Bethe, G. Bergmann, G. Embden, und A. Ellinger (eds.), Springer, Berlin, pp. 1045–1220.

Blane, H. T., 1968, "The personality of the alcoholic," Harper & Row, New York.

Bossell, H., 1977, Orientors of non-routine behavior, *in* "Concepts and tools of computer-assisted policy analysis," Vol. 1, H. Bossel (ed.), Birkhauser Verlag, Basel and Stuttgart, pp. 227–265.

Brunswick, E., 1956, Historical and thematic relations of psychology to other sciences, *Scientific Monthly* 83:151–161.

Bruun, K., Edwards, G., Lumio, M., Makela, K., Pan, L., Popham, R. E., Room, R., Schmidt, W., Skog, O. J., Sulkunen, P., and Osterberg, E., 1975, "Alcohol control policies in public health perspective," Vol. 25, Finnish Foundation for Alcohol Studies, Helsinki.

Cahalan, D., 1970, "Problem drinkers," Jossey-Bass, San Francisco.

Cahalan, D., Cisin, I., and Crossley, H., 1969, "American drinking practices: A national survey of drinking behavior and attitudes," Monographs of the Rutgers Center of Alcohol Studies, No. 6, New Brunswick, N.J.

Cannon, W., 1932, "The wisdom of the body," Norton, New York.

Cappell, H., 1975, An evaluation of tension models of alcohol consumption, *in* "Research advances in alcohol and drug problems," Vol. 2, R. J. Gibbins, Y. Israel, H. Kalant, R. E. Popham, W. Schmidt, and R. G. Smart (eds.), Wiley, New York, pp. 177–209.

Clare, A., 1977, How good is treatment? *in* "Alcoholism: New knowledge and new responses," G. Edwards and M. Grant (eds.), Croom Helm, London, pp. 279–289.

Cofer, C. N., and Appley, M. H., 1964, "Motivation: Theory and research," Wiley, New York.

de Lint, J., and Schmidt, W., 1968, The distribution of alcohol consumption in Ontario, *Q. J. Stud. Alcohol* 29:968–973.

Dochin, E., 1979, Event related brain potentials: A tool in the study of human information processing, *in* "Evoked brain potentials and behavior," H. Begleiter (ed.), Plenum Press, New York, pp. 13–88.

Dole, V. P., 1978, A clinician's view of addiction, *in* "The basis of addiction," J. Fishman (ed.), Abakon Verlagsgesellschaft, Berlin, pp. 37–46.

Edwards, G., 1977, The alcohol dependence syndrome: Usefulness of an Idea, *in* "Alcoholism: New knowledge and new responses," G. Edwards and M. Grant (eds.), Croom Helm, London, pp. 136–156.

Edwards, G., 1978, Drugs and the questions that can be asked of epidemiology, *in* "The basis of addiction," J. Fishman (ed.), Abakon Verlagsgesellschaft, Berlin, pp. 15–36.

Edwards, G., Orford, J., Egert, S., Guthrie, S., Hawker, A., Hensman, C., Mitcheson, M., Oppenheimer, E., and Taylor, C., 1977, Alcoholism: A controlled trial of "treatment" and "advice," *J. Stud. Alcohol* 38:1004–1031.

Festinger, L., 1957, "A theory of cognitive dissonance," Row Peterson, Evanston, Ill.

Fillmore, K. M., 1974, Drinking and problem drinking in early adulthood and middle age, *Q. J. Stud. Alcohol* 35:819–840.

Forrester, J. W., 1961, "Industrial dynamics," MIT Press, Cambridge, Mass.

Foster, R. O., Soeldner, J. S., Tan, M. H., and Guyton, J. R., 1973, Short-term glucose homeostasis in man: A systems dynamics model, *J. Dynam. Syst. Measurement Control* 1973:308–314.

Glaser, F. B., Greenberg, S. W., and Barrett, M., 1978, "A system approach to alcohol treatment," Addiction Research Foundation, Toronto.

Goldberg, L., 1943, Quantitative studies on alcohol tolerance in man, *Acta Physiol. Scand.* 5(Suppl. 16):5–128.

Goldstein, D. B., 1977, Temporal patterns of voluntary alcohol intake, *in* "Alcohol intoxication and withdrawal," Vol. 3b, M. Gross, (ed.), Plenum Press, New York, pp. 539–546.

Goluke, U., 1980, "A comprehensive theory of the pathogenesis of alcoholism: Appendix," Resource Policy Center, Thayer School of Engineering, Publication No 252, Hanover, N.H.

Goodman, M., 1974, "Study notes in system dynamics," Wright-Allen, Cambridge, Mass.
Goodwin, D., 1976, "Is alcoholism hereditary?" Oxford University Press, New York.
Gordis, E., 1976, What is alcoholism research? *Ann. Intern. Med.* 85:821–823.
Gross, M., 1945, The relation of the pituitary gland to some symptoms of alcoholic intoxication and chronic alcoholism, *Q. J. Stud. Alcohol* 6:25–35.
Gross, M. (ed.), 1973–1977, "Alcohol intoxication and withdrawal," 4 Vol., Plenum Press, New York.
Herz, A., and Schulz, R., 1978, Changes in neuronal sensitivity during addictive processes, *in* "The basis of addiction," J. Fishman (ed.), Abakon Verlagsgesellschaft, Berlin, pp. 375–394.
Hughes, P. H., and Crawford, G. A., 1972, A contagious disease model for researching and interviewing in heroin epidemics, *Arch. Gen. Psychiat.* 27:149–155.
Jellinek, E. M., 1960, "The disease concept of alcoholism," Hillhouse Press, New Haven, Conn.
Jessor, R., Graves, T. D., Hanson, R. C., and Jessor, S. L., 1968, "Society, personality, and deviant behavior: A study of a tri-ethnic community," Holt, Rinehart, & Winston, New York.
Johnston, V. S., 1979, Stimuli with biological significance, *in* "Evoked brain potentials and behavior," H. Begleiter (ed.), Plenum Press, New York, pp. 1–12.
Kalant, H., 1977, Alcohol withdrawal syndromes in the human: Comparison with animal models, *in* "Alcohol intoxication and withdrawal," Vol. 3b, M. Gross (ed.), Plenum Press, New York, pp. 57–64.
Katzper, H., Ryback, R., and Hertzman, M., 1976, "Preliminary aspects of modeling and simulation for understanding alcohol utilization and the effects of regulatory policies," U.S. Department of Commerce, National Technical Information Service, PB-263 023, Washington.
Kendall, R. E., and Statton, M. C., 1966, The fate of untreated alcoholics, *Q. J. Stud. Alcohol* 27:22–43.
Keshishian, J. M., 1979, Anatomy of a Burmese beauty secret, *National Geographic* 155(6):798–801.
Kissin, B., 1974, The pharmacodynamics and natural history of alcoholism, *in* "The biology of alcoholism," Vol. 3, B. Kissin and H. Begleiter (eds.), Plenum Press, New York, p. 8.
Kissin, B., 1977a, Medical management of the alcoholic patient, *in* "The biology of alcoholism," Vol. 5, B. Kissin and H. Begleiter (eds.), Plenum Press, New York, pp. 53–103.
Kissin, B., 1977b, Theory and practice in the treatment of alcoholism, *in* "The biology of alcoholism," Vol. 5, B. Kissin and H. Begleiter (eds.), Plenum Press, New York, pp. 1–51.
Kissin, B., 1979, Biological investigations in alcohol research, *J. Stud. Alcohol* Supplement No. 8, 40:146–203.
Kissin, B., and Begleiter, H. (eds.), 1971–1977, "The biology of alcoholism," 5 vols., Plenum Press, New York.
Kissin, B., and Hankoff, L., 1959, The acute effects of ethyl alcohol on the Funkenstein mecholyl response in male alcoholics, *Q. J. Stud. Alcohol* 20:696–703.
Kissin, B., Tuchfeld, B. S., Schuckit, M. A., Rosenberg, C. M., Glaser, F. B., and Matcom, A. J., 1977, Comments on "Alcoholism: A controlled trial of 'treatment' and advice," *J. Stud. Alcohol* 38:1804–1829.
Klausner, S. Z., Foulks, E. F., and Moore, M. H., 1980, "Social change and the alcohol problem on the Alaskan North Slope," Center for Research on the Acts of Man, Philadelphia.

Lashley, K., 1929, "Brain mechanisms and intelligence," University of Chicago Press, Chicago.
Lazarus, R. S., 1966, "Psychological stress and the coping process," McGraw-Hill, New York.
Lederman, S., 1956, "Alcool, alcoolisme, alcoolisation," Institut National d'études Démographique, Travaux et Documents, Cahier No. 29, Presses Universitaires de France, Paris.
Lemere, F., 1953, What happens to alcoholics? *Am. J. Psychiat.* 109:674–676.
Levi, L. (ed.), 1972, "Stress and distress in response to psychosocial stimuli," Pergamon Press, Oxford.
Levin, G., Roberts, E. B., and Hirsch, G. B., 1975, "The persistent poppy," Ballinger, Cambridge, Mass.
Lieber, C. S., 1977, Metabolism of ethanol, *in* "Metabolic aspects of alcoholism," C. S. Lieber (ed.), University Park Press, Baltimore, pp. 1–29.
Lieber, C. S., and DeCarli, L. M., 1977, Metabolic effects of alcohol on the liver, *in* "Metabolic aspects of alcoholism," C. S. Lieber (ed.), University Park Press, Baltimore, pp. 31–79.
Lumsden, D., 1975, Towards a systems model of stress, *in* "Stress and anxiety," Vol. 2, I. G. Sarason and C. D. Spielberger (eds.), Hemisphere, Washington, pp. 191–228.
Martin, W. R., and Jasinski, D. R., 1969, Physiological parameters of morphine dependence in man—Tolerance, early abstinence, protracted abstinence, *J. Psychiat. Res.* 7:9–17.
Maslow, A., 1954, "Motivation and personality," Harper & Row, New York.
McClelland, D. C., Davis, W. N., Kalin, R., and Wanner, H. E., 1972, "The drinking man: Alcohol and human motivation," Free Press, New York.
Meadows, D. L., Behrens, W., III, Meadows, D. H., Naill, R., Randers, J., and Zahn, E., 1974, "Dynamics of growth in a finite world," Wright-Allen Press, Cambridge, Mass.
Mechanic, D., 1973, "Politics, medicine, and social science," Wiley, New York.
Medicine in the Public Interest, 1979, "The effects of alcoholic beverage control laws," Washington.
Mello, N. K., 1972, Behavioral Studies of Alcoholism, *in* "The biology of alcoholism," Vol. 2, B. Kissin and H. Begleiter (eds.), Plenum Press, New York, pp. 219–291.
Mello, N. K., 1976, Some issues in research on the biology of alcoholism, *in* "Alcohol and alcohol problems," W. J. Filstead, J. J. Rossi, and M. Keller (eds.), Ballinger, Cambridge, Mass., pp. 167–192.
Menninger, K., 1938, "Man against himself," Harcourt, Brace, New York.
Menninger, K., Mayman, M., and Pruyser, P., 1963, "The vital balance," Viking Press, New York.
Mezey, E., and Tobon, F., 1971, "Rates of ethanol clearance and activities of the ethanol-oxidizing enzymes in chronic alcohol patients," *Gastroenterology* 61:707–715.
Miller, G. H., and Agnew, N., 1974, The Ledermann model of alcohol consumption, *Q. J. Stud. Alcohol* 35:877–898.
Miller, J. G., 1978, "Living systems," McGraw-Hill, New York.
Miller, W. R., and Caddy, G. R., 1977, "Abstinence and controlled drinking in the treatment of problem drinkers," *J. Stud. Alcohol* 38:986–1003.
Myrsten, A. L., 1977, Interaction of alcohol with psychological stress, *in* "Alcohol intoxication and withdrawal," Vol. 3b, M. Gross, (ed.), Plenum Press, New York, pp. 319–331.
Naitoh, P., 1972, The effects of alcohol on the autonomic nervous system of humans: Psychophysiological approach, *in* "The biology of alcoholism," Vol. 2, B. Kissin and H. Begleiter (eds.), Plenum Press, New York, pp. 367–433.
Okamoto, M., Rosenberg, H. C., and Boisse, N. R., 1977, Barbituate withdrawal in cats,

in "Alcohol intoxicavion and withdrawal," Vol. 3b, M. Gross (ed.), Plenum Press, New York, pp. 25–42.
Oppenheim, A. N., 1976, Towards a social psychology of dependence, *in* "Drugs and drug dependence," G. Edwards, M. A. H. Russel, D. Hawks, and M. MacCafferty (eds.), Saxon House, Farnborough, England, pp. 214–221.
Pattison, E. M., 1979, The selection of treatment modalities for the alcoholic patient, *in* "The diagnosis and treatment of alcoholism," J. H. Mendelson and N. K. Mello (eds.), McGraw-Hill, New York, pp. 126–227.
Polich, J. M., Armor, D. J., and Braiker, H. B., 1980, "The course of alcoholism: Four years after treatment," Rand Corporation, R-2433-NIAAA, Santa Monica, Calif.
Pomerleau, O., Pertschuk, M., and Stinnett, J., 1976, A critical examination of some current assumptions in the treatment of alcoholism, *J. Stud. Alcohol* 37:849–867.
Pribram, K. H., 1971; "Languages of the brain," Prentice-Hall, Englewood Cliffs, N.J.
Pugh, A., 1976, "Dynamo users manual," MIT Press, Cambridge, Mass.
Richmond, B., 1977, "Toward a structural theory of cancer," Massachusetts Institute of Technology, System Dynamics Memorandum D-2718, Cambridge, Mass.
Rix, K. J. B., 1977, "Alcohol and alcoholism," Eden Press, Montreal.
Roebuck, J. B., and Kessler, R. G. (eds.), 1972, "The etiology of alcoholism," Thomas, Springfield, Ill.
Roizen, R., Cahalan, D., and Shanks, P., 1978, Spontaneous remission among untreated problem drinkers, *in* "Longitudinal research on drug use," D. Kandel (ed.), Hempisphere Press, Washington, pp. 197–221.
Rutgers, 1979, "The etiology of alcoholism," J. Carpenter, Principal Investigator, and D. Lester, Scientific Director, National Institute of Alcohol Abuse and Alcoholism Grant No. 1 T50 AA03509-01.
Ryan, C., and Butters, N., 1980, Further evidence for a continuum-of-impairment encompassing male alcoholic Korsakoff patients and chronic alcoholic men, *Alc. Clin. Exp.* 4:190–198.
Ryback, R., 1971, The continuum and specificity of the effects of alcohol on memory, *Q. J. Stud. Alcohol* 32:995–1016.
Salaspuro, M. P., 1971, Influence of ethanol on the metabolism of the pathological liver, *in* "Biological basis of alcoholism," Y. Israel and J. Mardones (eds.), Wiley, New York, pp. 163–182.
Sargent, M., 1973, Theory in alcohol studies, *in* "Cross cultural approaches to the study of alcoholism," M. Everett, J. Waddel, and D. Heath (eds.), Mouton, The Hague, pp. 342–352.
Seixas, F. A., and Eggelston, S. (eds.), 1973, Alcoholism and the central nervous system, *Ann. N. Y. Acad. Sci.* 215.
Seixas, F. A., and Galanter, M., 1977–1979, "Currents in alcoholism," 6 vol., Grune & Stratton, New York.
Selye, H., 1956, "The stress of life," McGraw-Hill, New York.
Selye, H., 1974, "Stress without distress," Lippincott, New York.
Smith, C. M., 1978, "Alcoholism: treatment," Eden Press, Montreal.
Sokolov, E. N., 1963, "Perception and the conditioned reflex," Pergamon, Oxford.
Steiner, C., 1971, "Games alcoholics play," Grove, New York.
Strel'chuck, I. V., 1976, "Stages of chronic alcoholism and their correlation with defensive-protective reactions," *Int. J. Ment. Health* 5:74–84.
Tarter, R. E., and Sugarman, A., 1976, "Alcoholism: Interdisciplinary approaches to an enduring problem," Addison-Wesley, Reading, Mass.
Tintera, J. W., and Lovell, H. W., 1949, Endocrine treatment of alcoholism, *Geriatrics* 4:274–280.

Tokar, J. T., Brunse, A. J., Stefflre, V. J., Napior, D. A., and Sodergren, J. A., 1973, Emotional states and behavioral patterns in alcoholics and nonalcoholics, *Q. J. Stud. Alcohol* 34:133–143.
Ullman, A. D., 1952, The psychological mechanism of alcohol, *Q. J. Stud. Alcohol* 13:602–608.
U.S. Department of Health, Education, and Welfare, 1971, National Institute on Alcohol Abuse and Alcoholism, "First special report to the U.S. Congress on alcohol and health," DHEW Publication No. (ADM) 74-68, Superintendent of Documents, U.S. Government Printing Office, Washington.
U.S. Department of Health, Education, and Welfare, 1974, National Institute on Alcohol Abuse, and Alcoholism, "Second special report to the U.S. Congress on alcohol and health," DHEW Publication No. (ADM) 75-212, Superintendant of Documents, U.S. Government Prining Office, Washington.
Vaughan, H., 1979, A neurophysiology of mind, *in* "Evoked brain potentials and behavior," H. Begleiter (ed.), Plenum Press, New York, pp. 437–446.
Victor, M., Adams, R. D., and Collins, G. H., 1971, "The Wernicke-Korsakoff syndrome," Davis, Philadelphia.
Vince, M. A., 1949, Rapid response sequences and the psychological refractory period, *Br. J. Psychol.* 40:23–40.
Vogel-Sprott, M., 1972, Alcoholism and learning, *in* "The biology of alcoholism," Vol. 2, B. Kissin and H. Begleiter (eds.), Plenum Press, New York, pp. 485–507.
Wilder, J., 1967, "Stimulus and response: The law of initial value," John Wright, Bristol, England.
Williams, R. J., 1959, Biochemical individuality and cellular nutrition: Prime factors in alcoholism, *Q. J. Stud. Alcohol* 20:452–463.