THEORIES ON DRUG ABUSE
Selected Contemporary Perspectives

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provides a mechanism for easing the discomfort of conflict, an individual may seek out that particular drug when that conflict is reexperienced. The reinforcing quality of temporary stress reduction leads to continued reliance and utilization. The drug of choice will be the pharmacologic agent which proves harmonious with the user's characteristic mode of reducing anxiety. Furthermore, the selected drug appears to produce an altered ego state which is reminiscent of and may recapture specific phases of early child development, e.g., heroin, first year; amphetamines, second to third year.

**ADDICTIVE EXPERIENCES THEORY (p. 142)**

**Peette**

Persons use drugs, simply speaking, when they find such use to be rewarding in terms of values, needs, and overall life structure. Conceivably a drug can fulfill positive functions for an individual—such as enabling him or her to work better or to relate to others. Even in this case there is the danger that functioning in a positive sense will become dependent on continued drug use. In all cases, use of the drug will probably make it harder for the person to eliminate underlying and unresolved problems.

While the experience the drug produces for the person must provide rewards for him or her in order to maintain drug use, this is not to say that its objective impact on the user's life will not be negative. Thus narcotic or barbiturate users find the removal of pain and the absence of anxiety induced by the drug to be rewarding, even though these effects make them less sensitive to and less effective in dealing with their environment. In fact, it is this very depletion of capabilities which best guarantees continued use of the drug.

Consider the stimulant addict, such as the addicted coffee drinker, who uses caffeine to provide energy throughout the day. By masking fatigue, inadequate nutritional input, lack of exercise, etc., and all those deficiencies which force reliance on the caffeine, the drug makes the person less aware of the need to change his or her habits so as to be able to supply energy needs naturally. In this way, the caffeine perpetuates its own use.

**SOCIAL NEUROBIOLOGICAL THEORY (p. 286)**

**Prescott**

The continuation of substance usage is dependent, in part, upon the continuation of somatosensory affectional deprivation and the need to maintain friendships and social positions where those friendships and social positions are contingent upon the use of drugs or alcohol. Support for the continuing use of drugs is facilitated by the practices of modern medicine and the advertising practices of the pharmaceutical corporations. Social learning processes which operate at all levels of development (childhood to adulthood) capitalize upon the need for the body to find relief from tension and pain created in large part by
somatosensory affectional deprivation. Societal and moral values that are intrinsically opposed to somatosensory pleasure and sexual pleasure, in particular, provide support for the alternatives of drugs and alcohol. Societal opposition to massage parlors and prostitution but open acceptance and support of the alcohol industries is a case in point. Societal acceptance of addicting drugs that impair somatosensory pleasure, e.g., alcohol and methadone, and opposition to drugs that facilitate pleasure, e.g., marijuana and heroin, is another case in point.

Carstairs' (1968) classic study should be consulted in this context as a dramatic illustration of the reciprocal inhibitory relationships between drug use and behaviors that are culturally determined. Carstairs reported on the use of bhang (marijuana) and alcohol in the two highest caste groups, Rajput and Brahmin, in a village in northern India. The Rajput, the warrior class, indulged in alcohol, which facilitated the expression of sexuality and violence. The Brahmin was the religious class and indulged in bhang, which facilitated religious experiences and enhanced their spiritual life. The holy men avoided alcohol, which they considered destructive to salvation, and would not permit a Hindu who had consumed alcohol to "enter one of his temples (not even a goddess temple) without first having a purgatory bath and change of clothes" (p. 105).

The continuation of use or abuse and the choice of drug are culturally influenced. A culture will support the use of certain drugs that are consistent with and supportive of its own mores and values and will oppose the use of those drugs that interfere with these mores and values. Thus, the U.S. culture, which is predominantly an extroverted, violent, and exploitative culture (sexually and economically), supports the use of alcohol, which facilitates these behaviors. Conversely, the U.S. culture opposes the "pleasure" drugs (marijuana and heroin), which inhibit violence and exploitation and facilitate introspective and contemplative behaviors. (This statement should not be construed as supporting drug use for recreational purposes.) The issue is not whether a drug is addicting or nonaddicting—alcohol is addicting (culturally supported) and marijuana is nonaddicting (culturally opposed); heroin is addicting (culturally opposed) and methadone is addicting (culturally supported). Both the fabric and the loom of culture must be understood if the choice of specific drugs and the continuation of use and abuse are to be understood.

GENETIC THEORY (p. 297)

Schuckit

Once someone does try a drug, the decision to continue using the substance probably involves a combination of social and biological factors. While genetically mediated reactions to the drug may play a larger role here than in the initiation of use, social factors still hold great influence.

Genetically influenced biological factors may be important in the balance of pleasant and unpleasant effects seen with almost all substances on their first try. Constitutional factors may determine the incidence and severity of adverse problems, such as coughing, nausea, or vomiting, and may mediate the intensity of pleasant effects as well. Thus, the individual's personality, usual level of anxiety, the mechanisms and
Somatosensory Affectional Deprivation (SAD) Theory of Drug and Alcohol Use

James W. Prescott, Ph.D.

The somatosensory affectional deprivation (SAD) theory of drug and alcohol use is a developmental psychobiological theory that is proposed to account for the common ground of the many and diverse theories of substance abuse. The first basic proposition of this theory is that the neurobiology of our behavior is not only inseparable from, but is in fact largely shaped by, culture. The shaping process of culture upon the developing brain (the organ of behavior) is accomplished through our various sensory modalities and through the sensory processes of deprivation and stimulation.

With few exceptions, the developing mammalian brain, particularly the primate brain, is highly immature at birth and is dependent upon sensory stimulation for its normal growth, development, and functional and structural organization. The richness or paucity of dendritic structures of the neurone (brain cell), for example, is largely influenced by the sensory processes of stimulation and deprivation during the formative periods of brain development. The complexities and possibilities of neuronal communication (and thus behavior) are dependent upon the complexity of dendritic structures of brain cells (Greenough 1975; Greenough and Juraska 1979; Rosenzweig 1979; Floeter and Greenough 1979; Riesen 1975; Globus et al. 1973; Coss and Globus 1979; Coleman and Riesen 1968; Horn et al. 1979; Spinelli and Jensen 1979; Blakemore and Cooper 1970; Hirsch and Spinelli 1970; and Hubel and Wiesel 1970). Dendritic structures are analogous to telephone cables that interconnect various telephone centers (brain cells) with one another. These dendritic structures of brain cells form the structural basis of interneuronal communication. Another major element in the story of interneuronal communication is neurochemical transmitter substances which are present at synaptic junctions between dendrites and which make possible the transfer of "information" from one brain cell to another. These events are accompanied by electrophysiological activity, which is another manifestation of interneuronal communication. The point of this synoptic overview of interneuronal communication is to emphasize that the morphological (structural) and the neurochemical and electrophysiological (functional) processes of interneuronal
communication are all strongly influenced by the sensory processes of stimulation and deprivation. Thus, the effects of the social, physical, and cultural environment are ultimately transformed into perceptual experiences through the encoding and decoding of sensory processes. Further, whether certain perceptual experiences can ever be realized will be dependent upon the quality and quantity of our sensory experiences, as structured by our social, physical, and cultural environment during the formative periods of brain development (Prescott 1967, 1971a,b, 1972a,b, 1973, 1975, 1976a,b, 1977, 1978, 1979b).

The second basic proposition of SAD theory is that certain sensory modalities and processes are more important than others in accounting for emotional/social disturbances and substance abuse. Specifically, it is the emotional senses of somesthesia (touch), vestibulation (movement), and olfaction (smell), that are the primary mediators of our emotional/affective behaviors. Substance abuse that alters primarily our emotional/affective state must be understood within the context of our emotional senses. It is the deprivation of our emotional senses and not our cognitive (visual-auditory) senses during the formative periods of brain development that can account for and predict our emotional/affective social behaviors, which include not only substance abuse but abusive social behaviors in general. Thus, the question of destructive and exploitive behaviors toward ourselves and others becomes a question of whether affectional bonds are formed or not formed during the formative periods of brain development. Within an evolutionary context, it should be noted that olfaction assumes a greater role in lower mammals, and vestibular functions assume a greater role in higher mammalian forms, specifically the primate, in the formation of affectional bonds (Prescott 1976a, 1977). Similarly, substance abuse that alters primarily our cognitive state (e.g., hallucinogens) must be understood within the context of our cognitive (visual-auditory) senses. It should be noted that movement (vestibulation) is often involved in altered cognitive states and it has been proposed that the vestibular-cerebellar neuraxis may be a master integrating/regulating system of sensory-emotional and motor processes. Thus, the vestibular-cerebellar system may serve as a "bridge" between our "emotional" and "cognitive" senses (Prescott 1976a, 1977; Erway 1975).

In previous studies, the SAD theory has been successful in predicting physical violence (high and low) in 100 percent of 49 primitive cultures distributed throughout the world. This was made possible by evaluating the degree of physical affection (touching, holding, carrying) of the infant by its mother or caretakers and by the degree of physical affection that was permitted to be expressed through the acceptance or rejection of premarital sexuality (Prescott 1975, 1977, 1979b).

The issue of violence, i.e., the failure of nurturance and the failure to form affectional bonds, is strongly related to the issue of substance abuse in several aspects. First, in a very general sense, the body needs and "searches" for a state of harmony, contentment, and in higher life forms (homo sapiens), an altered and transcendental state of conscious "being." A necessary condition for the attainment of this "state of being" is the experiencing of physical (somatosensory) pleasure that is essential for the formation of affectional bonds. When somato-sensory pleasure and affectional bonds are denied, then compensatory behaviors to reduce tension, discomfort, and "anomie" become imperative. The common compensatory behaviors are physical violence (toward others and oneself), alcoholism and drug abuse, and perseverative stimulus-seeking behaviors that attempt to provide the sensory
stimulation that was deprived early in life. The stereotypical rocking behaviors of isolation-reared Harlow monkeys and of institutionalized children is a case in point. The "quieting" effect of stimulant drugs upon some hyperactive children is another illustration of a "need for neural activation" that is met by pharmacological stimulation rather than by sensory stimulation. The chronic stimulus-seeking behaviors, particularly of a sexual and violent nature, in the American culture (evidenced, for example, by massage parlors, pornography, violent films, rape) are also illustrative of this basic principle of stimulus-seeking behaviors consequent to early somatosensory deprivation (Prescott 1972a, 1973, 1975, 1976a,b). Additional studies that relate early sensory experiences to later behaviors, particularly aberrant sensory behaviors, can be usefully consulted (Ainsworth 1972; Cairns 1966, 1972; Bowlby 1969; Harlow 1971; Harlow et al. 1963; Dokecki 1973; Lichstein and Sackett 1971; Lynch 1970; Mason 1968, 1971; Mason and Kenney 1974; Mason and Berkson 1975; Fuller 1967; Freedman 1968; Friedman et al. 1968; Melzack and Burns 1965; Melzack and Thompson 1958; Melzack and Scott 1957; Mitchell 1968, 1970, 1975; Mitchell and Clark 1968; Sackett 1970; Riesen 1960, 1961a,b, 1965; Schaffer and Emerson 1966a,b; Spitz 1945, 1965; Suomi and Harlow 1972; Zubek 1969).

The self-mutilation and pain agnosia of children characterized by psychosocial dwarfism consequent to somatosensory affectional deprivation and child abuse reported by Money et al. (1972), is a classic verification at the human level of the same behaviors (self-mutilation and pain agnosia) found in animals reared under conditions of somatosensory affectional deprivation (social isolation) (Lichstein and Sackett 1971; Melzack and Burns 1965; Melzack and Scott 1957; and Mitchell 1968, 1970, 1975). The pain agnosia of children subjected to physical restraint and immobilization reported by Friedman et al. (1968) is another demonstration of these relationships at the human level.

Another important dimension to these early experiences and behaviors is the neurochemical and neuroendocrine mediators of pain hypersensitivity and pain hyposensitivity (pain agnosia) consequent to somatosensory deprivation. Harvey and Yungcr (1973) have shown that decreases in brain serotonin (5-HT) result in an increased sensitivity to pain, and Coleman (1971) has shown that isolation-reared monkeys who are characterized by both tactile hypersensitivity and hyposensitivity (Lichstein and Sackett 1971) have significantly decreased levels of platelet serotonin.

A number of investigators have also shown that there is significant reduction in growth hormone (GH) and adrenocorticotropic (ACTH) in psychosocial dwarfism (reversible hyposomatropism) (Patton and Gardiner 1975; Powell et al. 1967a,b; Wolff and Money 1973; Money and Wolff 1974; Brown 1976). Significant to these findings is the report that endogenous opioids are involved in the regulation of serum growth hormone (GH) and prolactin (PRL). Specifically, naloxone depresses basal serum concentration of GH and PRL. Related to the above are the well-known phenomena that stress elicits an increase of endogenous opioids in the brain; and of ACTH and β-endorphin in the systemic circulation; and that serotonin increases prolactin, growth hormone, and adrenocorticotropic (Meites et al. 1979).

These observations are made to suggest that psychosocial dwarfism may well be characterized by abnormal endorphin mechanisms which may be responsible for the observed abnormalities of GH and ACTH in
psychosocial dwarfism. Thus, these speculations suggest that endorphin mechanisms may assume a much greater role and significance in somatosensory affectational deprivation phenomena than has heretofore been realized.

The findings of Behling (1979) highlight the relationship between alcohol abuse, child abuse, and failure of nurturance, showing that in 69 percent of 51 instances of child abuse, at least one parent had a history of alcohol abuse.

In the context of the SAD theory, it is not surprising to find the compensatory behaviors of violence in the primitive culture study cited above or the finding of Barry (1976) that the single greatest predictor of drunkenness in 13 primitive cultures was the large amount of crying during infancy ($r=0.77$). Drunkenness was also significantly correlated with low general indulgence during infancy ($r=0.48; N=26$) and low duration of bodily contact with caretaker during later stages of infancy ($r=0.42; N=23$). Significant relationships between deprivation of parental physical affection and use of drugs and alcohol have been reported for college students (Prescott 1975), for prisoners (Prescott and Wallace 1978), for institutionalized alcoholics, and for participants in a drug treatment program (Prescott and Wallace 1976). Significant relationships between high drug and alcohol usage with attitudes rejecting premarital and extramarital sex have also been reported for college students (Prescott 1975).

An interpretive statement of these relationships with respect to somatosensory pleasure seeking, isolation rearing (somatosensory affectational deprivation), altered neuronal communication, and altered states of consciousness" appears necessary. Briefly, the SAD theory postulates that somatosensory deprivation from isolation rearing leads to impaired brain neuronal systems that mediate pleasure which now lack the neuronal structural bases to interact with and influence higher brain (cognitive) centers (neocortex). This impairment prevents an integration of somatosensory pleasure with higher brain centers and precludes the normal development of altered states of consciousness or states of "transcendent being." (See Teilhard de Chardin's 1933 essay "The Evolution of Chastity" on the role of pleasure in achieving states of "transcendent being."). Consequently, most of the somatosensory pleasure-stimulus-seeking behaviors of contemporary Western civilization (not just America) appear to be "nonintegrative" in nature, i.e., primarily "reflexive." This means the "pleasure experience" is a momentary and transitory phenomenon that produces a temporary reduction of physiological tension and discomfort but does not represent a true positive state of "integrative pleasure" that is essential for experiencing an "altered state of consciousness." Thus, anomic remains, a high need for another "pleasure fix" remains, and the complex of perseverative behaviors remains. Drugs and alcohol "bypass" the somatosensory process and provide a direct route to higher brain centers that alter "states of consciousness" which simulate states of "transcendent being." It should be noted that somatosensory affectional deprivation from social isolation results in an aversion to touch and thus constitutes a barrier to the "touch therapy" that is essential for rehabilitation, namely, the establishment of emotional/affective-social relationships.

Within the context of SAD theory, three basic groups of substance abusers are proposed to exist and need to be evaluated and treated differently. These are (a) pleasure seekers (marijuana, heroin, etc.),
(b) pleasure avoiders (alcohol, depressants, tranquilizers), and (c) "altered states of consciousness" seekers (hallucinogens).

A factor-analytic study involving items of drug and alcohol usage produced orthogonal (independent) factors for alcohol and marijuana usage (Prescott and Wallace 1976). Unfortunately, time and space do not permit review of these data or an elaboration of SAD theory of drug typologies and their implications for research and therapy. It is suggested, however, that a sensory process orientation would be highly heuristic. Special attention should be given to evaluating vestibular-cerebellar processes in alcoholics, somesthetic-cerebellar processes in pleasure-seeking drug users, and visual/auditory neocortical processes in hallucinogen users. It should be recognized that these suggestions are highly speculative and have many limitations, but they may, nevertheless, have some merit in attempting to identify specific neurobiological brain processes with specific choices of substance use and abuse.

Evidence that social isolation rearing alters neurochemistry of brain function has been partially reviewed elsewhere (Prescott 1971a, 1976a; Lal et al. 1972; Essman 1974, 1979; Essman and Casper 1978; Welch and Welch 1969; Valzelli 1967; De Feudis and Marks 1973; Rosenzweig 1979; Rosenzweig et al. 1968). Certain studies, however, deserve special commentary, and recent developments with respect to the endorphins are especially relevant to somatosensory affective deprivation theory and data, as is the basic alteration of the CNS's response to drugs that is induced by SAD of isolation rearing.

In this specific social-neurobiological context, Lal et al. (1972) have demonstrated that social isolation rearing of mice (somatosensory affective deprivation) significantly altered the pharmacological effects of hexobarbital, pentobarbital, chloral hydrate, barbital, and chlorpromazine. Specifically, social isolation enhances stimulant drug effects and reduces CNS depressant effects.

Bonnet et al. (1976) reported that mice reared in social isolation (somatosensory affective deprivation) for 20 weeks showed a significant reduction in narcotic agonist and antagonist binding. No differences could be found in stereospecific binding between the rearing groups with 15 weeks of differential rearing, but were found at 17 and 21 weeks. These authors also reported a significant reduction of the number of opiate binding sites in the brains of isolation-reared mice compared to aggregation-reared mice. This loss of opiate receptor sites in isolation-reared mice may be analogous to the loss of dendrites consequent to social isolation rearing.

Panksepp et al. (1978) and Herman and Panksepp (1978) reported a significant decrease in distress vocalizations of puppies which were briefly separated from their mothers (15 minutes) with an injection of 0.125 mg/kg of oxyphormine, and they found that naloxone increased group vocalization of two- to five-day-old white Leghorn chicks briefly separated from their mother. These authors discuss the parallels between the biological nature of narcotic addiction and the formation of social bonds, and their theoretical position is similar to SAD theory and my belief that the brain endorphin systems may be one of the most important neurobiological systems mediating the development of affectational bonds, including sexual affectional bonds.
The role of endorphins in sexual behavior has been studied by Gesa et al. (1979), and they have reported the following findings from their rat study:

a) DALA (D-Ala²-Met-enkephalinamide) given intracerebroventricularly at a dose of six micrograms completely inhibited copulatory behavior and the ability to ejaculate in sexually active rats. Naloxone (four mg/kg) given intraperitoneally completely reversed this effect.

b) Naloxone does not enhance sexual behavior in sexually active rats.

c) Naloxone (four mg/kg) given intramuscularly significantly enhances mounting, intromission, and ejaculation in sexually inactive rats.

These authors suggest that endorphins may mediate sexual disorders and that opioid antagonists "might become potentially useful therapeutic agents for sexual disturbances in man" (p. 204). A similar statement might be made for the treatment of alcoholics whose somatosensory pleasure system is dysfunctional and often inoperable. Whether pleasure-inducing drugs, such as marijuana and the opioids, may prove to be a useful first step in a program of somatosensory rehabilitation for alcoholics and other somatosensory impaired individuals remains to be demonstrated. Different therapeutic strategies appear indicated, however, for differing classes of substance abusers.

Veith et al. (1978) have also reported the effects of endorphin compounds upon emotional and sexual behaviors in rats. They examined the consequences of a single intraperitoneal injection of 100 mg of α-endorphin (β-LPH 61-76), γ-endorphin (β-LPH 61-77), and β-endorphin (β-LPH 61-91), and a [D-Ala²] analog of Met-enkephalin upon several measures of open field behavior compared to saline controls.

In brief, these authors found that β-endorphin enhanced grooming behavior; γ-endorphin and its analog [D-Ala²] increased emotional responses (ran to the wall faster and greater defecation); and α-endorphin [D-Ala¹] increased sexual arousal (penile erection and seminal discharge). The selective behavior effects of these various peptides were emphasized, and it was suggested that each peptide may be coded to act upon receptor rates in a differential manner to mediate the differing behavioral effects.

From this writer's perspective it is sufficient to emphasize the social, emotional, and pleasure (sexual) behaviors that are induced by endorphin compounds. In this context, it is heuristic to note the findings of Houck et al. (1980) who reported two β-endorphinlike materials in human placenta from three patients undergoing natural childbirth. These authors speculate upon the possible role of placental endorphins "as a natural antidote to the pain and stress of parturition." This writer cannot help but speculate further that the positive emotional state toward pregnancy of women electing natural childbirth may be reflected in a "positive intrauterine state" that is characterized by the presence of placental endorphin. This raises additional questions whether "stressful" pregnancies or "unwanted" pregnancies are characterized by a significant decrease or lack of placental endorphins.

Finally, does the presence or absence of placental endorphins reflect, in any way, the integrity of fetal endorphin mechanisms or the future developmental integrity of neonatal/infant/child endorphin mechanisms? Does obstetric medication have any adverse effect on fetal endorphin
mechanisms? Do such events have any long-term developmental implications for how pain and pleasure are experienced, the quality of development of emotional-social relationships, and whether and what coping/compensatory behaviors may be adopted as a consequence of dysfunctional psychobiological affectational mechanisms?

These studies are cited because of the increasing evidence that has linked affectional variables and early social isolation to (a) violence, drug and alcohol abuse, and sexual dysfunctioning; (b) altered neurochemistry, electrophysiology, and dendritic structures (neuronal communication) in somatosensory and motor cortex and cerebellar cortex; (c) altered narcotic agonist and antagonist binding; and (d) altered CNS response to stimulant and depressant drugs. The role of sexual functioning and sexual pleasure in the developmental continuum of affectional bonding and its relationship to endorphins, drug and alcohol use, and violence, particularly alcohol-induced violence, brings a convergence of theories and experimental evidence that were heretofore considered disparate entities and phenomena. The report of Pradelles et al. (1979) that visual deprivation decreases Met-enkephalin in various amygdaloid and striatal structures provides further support for linking sensory deprivation phenomena to enkephalin neurotransmitter or neuroregulatory processes.

The findings of Gesa et al. (1979) and of Panksepp (1978), however, appear contradictory and inconsistent with this proposed convergence.
In the former study, stimulation of opiate receptors induced pleasure-deficit behaviors (failure to copulate and ejaculate), whereas in the latter study, stimulation of opiate receptors induced pleasure-enhancement behaviors (decrease in distress vocalizations). Similarly, in the Gesa study naloxone enhanced pleasure behaviors (increased copulation and ejaculation), whereas naloxone decreased pleasure behaviors (enhanced distress vocalization) in the Panksepp study. These apparent fundamental contradictions are, it is proposed, resolvable within SAD theory and Cannon's Law of Denervation Supersensitivity (Cannon 1939; Cannon and Rosenbleuth 1949; Collier 1968; Sharpless 1975), which is an integral and essential neurophysiological mechanism of SAD theory (Prescott 1971a, 1972b).

Briefly, fundamental distinctions must be made between CNSs that are characterized by or not characterized by denervation supersensitivity, which is induced by deafferentation, i.e., a loss of afferent input. Sexual inactivity, like social isolation rearing, involves somatosensory deprivation that constitutes a special case of functional deafferentation. As reported by Struble and Riesen (1978), primate isolation rearing results in loss of dendrites in somatosensory cortex. The loss of opiate receptor sites, reduced narcotic agonist and antagonist binding, enhancement of stimulant drug effects, and inhibition of depressant drug effects are also all consequent to social isolation and thus share, in my view, a common explanatory mechanism, namely, Cannon's Law of Denervation Supersensitivity. It is within this context that it is relevant to emphasize that opioid substances act on their receptors to depress the activity of cells bearing these receptors and, consequently, are classed as inhibitory neurotransmitters (Frederickson and Norris 1976). The enhancement of these inhibitory neurotransmitters through the mechanism of denervation supersensitivity might account for the inhibition of copulatory and ejaculatory behavior as reported by Gesa et al. (1979). Similarly, the absence of denervation supersensitivity in Panksepp's experimental subjects could account for his endorphin stress-reducing (pleasure-enhancing?) effects.
The findings of Gispen et al. (1976) that low doses of β-endorphin (0.01-0.3 micrograms) induced excessive grooming behavior in rats, and of Meyerson and Terenius (1977) that "higher" doses of β-endorphin (one and three micrograms) significantly reduced mounting and copulatory behavior in Wistar rats exposed to estrous females support the "bidirectionality" (prosocial versus asocial behaviors) of endorphin mechanisms. Naltrexone given subcutaneously 30 minutes before the peptide blocked the effect of one microgram β-endorphin, thus confirming that impaired sexual functioning was mediated via opiate receptors. It should be noted that one microgram β-endorphin did not interfere with sexual exploratory behavior that included active pursuit and investigation of the anogenital area of the female.

These reports of bidirectionality of endorphin activity as a function of dosage level, the endorphin antagonistic effects, and the naloxone agonistic effects concerning sexual behaviors are not unrelated to the naloxone agonistic effects concerning pain perception.

Levine et al. (1978), in a study of human clinical pain (tooth extraction), found that naloxone produces analgesia at low doses (0.4 and 2 mg) and hyperalgesia at high doses (7.5-10 mg) for a placebo-respondent group. Interestingly, naloxone had little effect on placebo nonresponders. Questions must be raised whether placebo responders and those experimental preparations that manifest naloxone agonistic effects (bidirectionality) could be characterized by SAD or other forms of induced denervation supersensitivity. These questions are relevant to the findings of Buchsbaum et al. (1977), who divided their subjects into pain-sensitive and pain-insensitive groups as determined by their ratings of an electric shock. They found that only the pain-sensitive subjects reported a naloxone (2 mg) analgesic effect and that pain-insensitive subjects showed naloxone hyperalgesia.

Although the studies of Levine et al. (1978) and Buchsbaum et al. (1977) are not directly comparable since Levine employed multiple doses of naloxone and Buchsbaum employed a single naloxone dose, it is of interest to contrast the two naloxone hyperalgesia groups with respect to the issue of placebo responding. Levine et al. reported a naloxone bidirectional effect for placebo responders, whereas Buchsbaum's pain-insensitive bidirectional responders (naloxone hyperalgesia) were characterized as placebo "nonresponders" since their placebo response was less than half that of the pain-sensitive group. These "inconsistencies" require further experimental study.

These observations only complicate an already very complicated set of issues and phenomena of endorphin-related behaviors. However, the bidirectionality phenomena of naloxone and the naloxone agonist effects and endorphin antagonist effects involving not only pain phenomena but also sexual-social and motor behaviors (Oese et al. 1979; Meyerson and Terenius 1977; Gispen et al. 1978; Bloom et al. 1976; Jacquet and Marks 1976) suggest an extremely complex role of modulation, regulation, and integration of sensory, social, emotional, and motor behaviors by the endorphin system.

A theory of cerebellar regulation and integration of sensory, social, emotional, and motor behaviors within the context of SAD theory has been previously elaborated (Prescott 1971a, 1976a, 1978). Heath and his coworkers (Heath 1972, 1975a,b, 1976, 1977; Heath et al. 1978, 1979) have established a wealth of data describing cerebellar-limbic
relationships, which were postulated by SAD theory. They have further dramatized how cerebellar stimulation can modulate extreme states of emotional expression (positive and negative) in human subjects. According to SAD theory, the cerebellum is not itself the site of these behaviors, but it exerts a regulatory influence on limbic, reticular, and frontal cortical structures to modulate these behaviors. Cerebellar modulation of limbic-endorphin activity would be a natural extension of SAD theory and could be tested in both animal and human studies. It would be expected, for example, that endorphin/naloxone behaviors would be altered with chronic cerebellar electrical stimulation that resulted in profound changes in emotional behavior, as described by Heath et al. In particular, since Heath (1972, 1975a,b) has documented abnormal electrical spike discharges in the limbic and cerebellar structures of isolation-reared primates, and Saltzberg and colleagues (Saltzberg et al. 1971; Saltzberg and Lusick 1975; Saltzberg 1976) have developed signal analysis methods to detect these deep brain spike discharges from scalp EEG recordings, it is now possible to undertake studies that could link a known history of somatosensory affectional deprivation to abnormal deep brain spike activity and to specific patterns of endorphin/naloxone-induced behaviors associated with dysfunctional behaviors, e.g., alcohol-induced violence and impaired sexual functioning. Effective therapies should be reflected in elimination of spike discharges, altered endorphin/naloxone behaviors, development of affectional emotional behaviors, and elimination of drug and alcohol dependence.

The role of the cerebellum in somatosensory affectional deprivation has been given support by Berman et al. (1974); and Floeter and Greenough (1979), who reported significant increases in spiny branchlets of Purkinje cells in the para floculus and the nodulus of the cerebellum in monkeys reared in colony conditions compared to isolated-reared and socially experienced animals (environmental variation of SAD). The finding of opiate receptors in the cerebellum should be noted in this respect (Meunier and Zajac 1979). Although denervation supersensitivity mechanisms inherent in somatosensory affectional deprivation are offered as a major explanatory process in accounting for the variety of diverse and often apparently inconsistent and contradictory findings from the endorphin/naloxone behavioral literature, it is recognized that other factors, e.g., neonatal anoxia, can induce denervation supersensitivity (Berman and Berman 1975; Burch et al. 1975) and that the "family" of endorphins and their antagonists are additional factors that can contribute to the complexity of findings reported in the literature and their interpretation.

The major theoretical orientation of this paper is to emphasize that any study of endorphin/naloxone behaviors or drug/alcohol behaviors must take into account the developmental history of the organism to determine whether the CNS of that organism is characterized by denervation supersensitivity, whether induced by somatosensory affectional deprivation or other etiological developmental factors.

The phenomenon of "hyperendorphinism" of affective disorders (Buchsbbaum et al., in press), which may well be an expression of "neurotransmitter density" due to denervation supersensitivity, is an example of a construct that might be benefited by a developmental perspective. (Neurotransmitter density in neurochemistry is analogous to current density in electrophysiology and expresses the relationship of the amount of released neurotransmitter substance available to the number of available receptors.)
Since isolation rearing results in a reduction of the number of opioid receptors, a state of "hyperendorphinism" may not reflect a change in the absolute volume of released endorphin but rather a change in the number of opioid receptors (endorphin density). The converse could also occur (increased volume of endorphin with receptor number remaining constant) for different etiological reasons. This is mentioned for the purpose of suggesting that "hyperendorphinism" may not be a unitary phenomenon since different mechanisms and etiologies could mediate this effect.

It would be a serious omission not to mention the classic theoretical system developed by Petrie (1976), which has unusual relevance to the issues of substance abuse and to somatosensory affectional deprivation theory. Briefly, Petrie has proposed a theoretical system that postulates CNS processes of reduction and augmentation of the sensory environment to describe an individual's "reactance" to pain and sensory deprivation. The "CNS augmenters" are characterized by an intolerance for pain and a tolerance for sensory deprivation. This pattern of reactance occurs because the CNS of these individuals acts to augment reactance occurs because the CNS of these individuals acts to reduce or inhibit the impact of a given sensory event upon the CNS. Thus, the "CNS reducers" are characterized by a chronic state of insufficient afferent stimulation (stress of sensory insufficiency or sensory deprivation) and engage in behaviors that are designed to maximize afferent stimulation of the CNS. Consequently, these "CNS reducers" are those who engage in a variety of stimulus-seeking behaviors, e.g., when punished with solitary confinement, delinquents who are CNS reducers will frequently engage in self-mutilative behaviors, such as cutting themselves with razors or burning themselves with cigarettes (note self-mutilation of isolation-reared animals).

Petrie (1976) described the response of reducers, moderates, and augmenters to alcohol and found that augmenters were most affected by dramatically changing from an augmenting reactance mode to a reducing reactance mode. Similar but less strong reducing effects were obtained with reducers. Comparable results were obtained with other drugs, such as aspirin and chlorpromazine. Thus, augmenters as a group were shifted away from pain intolerance to pain tolerance. Buchsbaum (1978) has provided a review of a number of neurophysiological studies from his laboratory and others on reducers and augmenters. Without reviewing all of his findings, suffice it to point out that he reported that reduction of the amplitude of sensory-evoked potentials to increases in stimulus intensity was associated with pain tolerance and analgesia, and that augmentation was linked to substance abuse. The studies of Buchsbaum and Ludwig (in press) and von Knorring and Oreland (1978) are also relevant to these issues.

It has been previously suggested that somatosensory affectional deprivation of isolation rearing is a major contributing factor in the developmental neuropsychobiological substrate of Petrie's typology of reducer and augmenters (Prescott 1967). Chronic or perseverative stimulus-seeking behaviors and impaired pain perception, for example, are predominant characteristics of somatosensory affectional deprivation (denervation supersensitivity) and the "CNS reducer." There are, however, significant differences in the communality of the two theoretical systems in which SAD is characterized by "paradoxical" behaviors.
e.g., simultaneous supersensitivity to touch and impaired pain perception that are not accounted for by Petrie's typology. Zuckerman's (1979) theory of sensation seeking is also intrinsically related to the theories of Petrie (1976) and Prescott (1967, 1971a,b, 1972a,b, 1973, 1975, 1976a,b, 1977).

This writer has attempted to link these basic developmental neurobiological processes of SAD to cross-cultural characteristics of child-rearing practices; to social and religious mores and customs that regulate sexual behaviors; and to personality characteristics of authoritarianism, exploitation, and narcissism in contrast to egalitarianism, nurturance, and altruism. Further, it is postulated that these contrasts in personality characteristics, considered at the microsocial level, constitute the bases for the political structure of a culture, namely, egalitarian-democratic societies versus authoritarian-fascist societies (Prescott 1975, 1976, 1977). It is of some significance that Petrie (1976) draws exactly the same parallels from her theory to the characteristics of both personality and culture with her typologies of "compassion" (augmenter) versus "callousness" (reducer) (pp. xii-xiv).

In concluding this theoretical essay it hardly needs to be emphasized that the social-emotional dysfunctions of the individual in society, in whatever form it may be expressed, is not only an intrinsic aspect of neurobiological functioning of the individual but also of the social-psychological forces of culture that shape the individuality of neurobiological functioning through the formative developmental processes of sensory stimulation and deprivation, and through a culture of chemical and physical environments that influence fetal, neonatal, and postnatal development. Maternal habits of chemical ingestion, e.g., alcohol, drugs, food/spice preferences, or exposure to certain chemical environments during gestation, may well "imprint" upon the developing fetus certain "sensitivities" and "predispositions" for use or avoidance of those chemical agents during postnatal life with all the implications that this has for behavior.

It necessarily follows that preventive and therapeutic programs cannot be restricted to molecular biological strategies that are directed at the individual organism. The reconstruction of the individual requires also the reconstruction of society and culture.

The elements of societal and cultural reconstruction involve not only shaping a safe, beneficent physical environment but also a nurturant, caring, and affectionate environment of human relationships. The latter touches deeply upon philosophical and religious ideologies that regulate the morality of pain and pleasure in human relationships and the role of women in society.

The matrilineal/patrilinial structure of human cultures and their relationship to nurturance in human relationships, as well as the construction of the supernatural in human cultures, are a logical extension of SAD theory. However, it is beyond the scope of this essay to develop these topics and relate them to what has been reviewed herein.


Robbins, E.S.; Frosch, W.A.; and Stern, M. Further observations on untoward reactions to LSD. American Journal of Psychiatry, 124:393-395, 1967.


