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Sensation seeking: A comparative approach to a human trait

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Abstract: A comparative method of studying the biological bases of personality compares human trait dimensions with likely animal models in terms of genetic determination and common biological correlates. The approach is applied to the trait of sensation seeking, which is defined on the human level by a questionnaire, reports of experience, and observations of behavior, and on the animal level by general activity, behavior in novel situations, and certain types of naturalistic behavior in animal colonies. Moderately high genetic determination has been found for human sensation seeking, and marked strain differences in rodents have been found in open-field behavior that may be related to basic differences in brain neurochemistry. Agonistic and sociable behaviors in both animals and humans and the trait measure of sensation seeking in humans have been related to certain common biological correlates such as gonadal hormones, monoamine oxidase (MAO), and augmenting of the cortical evoked potential.

The monoamine systems in the rodent brain are involved in general activity, exploratory behavior, emotionality, socialization, dominance, sexual and consummatory behaviors, and intracranial self-stimulation. Preliminary studies have related norepinephrine and enzymes involved in its production and degradation to human sensation seeking. A model is suggested that relates mood, behavioral activity, sociability, and clinical states to activity of the central catecholamine neurotransmitters and to neuroregulators and other transmitters that act in opposite ways on behavior or stabilize activity in the arousal systems. Stimulation and behavioral activity act on the catecholamine systems in a brain–behavior feedback loop. At optimal levels of catecholamine systems activity (CSA) mood is positive and activity and sociability are adaptive. At very low or very high levels of CSA mood is dysphoric, activity is restricted or stereotyped, and the organism is unsocial or aggressively antisocial. Novelty, in the absence of threat, may be rewarding through activation of noradrenergic neurons.

Keywords: activity, arousal; behavior genetics; bioamines; catecholamines; emotionality; evoked potentials; extraversion; individual differences; personality; psychopathology; sensation seeking; socialization

> Novelty is always the necessary condition of enjoyment. The development of men up to now does not seem to me to need any explanation differing from that of animal development.
>
> Sigmund Freud (1948, pp. 43, 52)

A biological approach to human traits must of necessity be a comparative approach. It is, of course, possible to treat human personality as strictly a phenomenon of socialization, which is determined at the broadest level by culture and society, and many social theorists do so. Individual differences are accounted for by variations in the way societies' norms for behavior are translated within the family, and by subsequent social influences. Although most learning theorists such as Bandura (1977) and Skinner (1953) acknowledge the importance of biological factors they feel that the investigation of human behavior is more practically limited to the study environmental influences. [See *BBS* special issue on the canonical papers of B. F. Skinner, *BBS* 7(4) 1984.]

It is this author's conviction that a comparative biosocial approach is feasible at this time, although it must be conceded that our progress will be paced to future advances in the neurosciences. Recent contributors to this journal such as Gray (1982) and Panksepp (1982) agree with this optimistic view and have presented us with biological models for a specific emotional trait, anxiety (Gray), and the primary emotions in general (Panksepp). I would like to describe a biosocial model of a personality trait labeled "sensation seeking," with some reference to the related traits of "extraversion" and "impulsivity."

A model for a comparative approach. Among comparative biosocial personality theorists there are two modes of approach determined largely by our training and experience. Theorists like Jeffrey Gray prefer to define basic dimensions of individual differences in behavior at the animal level, where more elegant and direct experimentation is possible, and then to extrapolate their findings to dimensions defined by tests and behavioral observations at the human level. Theorists like Hans Eysenck (1967) and me (Zuckerman 1979b) work in the opposite direction by studying humans and attempting to extrapolate down the phylogenetic scale. Regardless of which direction we take, we are faced with the problem of linking the two levels of observation. As long as the links between human and animal behavior rest only on the judged similarities of behavior we run the risk of anthropomorphism (generalizing inappropriately from humans to animals) or zoomorphism (generalizing inappropriately from animals to humans).

I have proposed a method that goes beyond the metaphorical comparisons of human and animal behavior (Figure 1). The essence of this is the search for common

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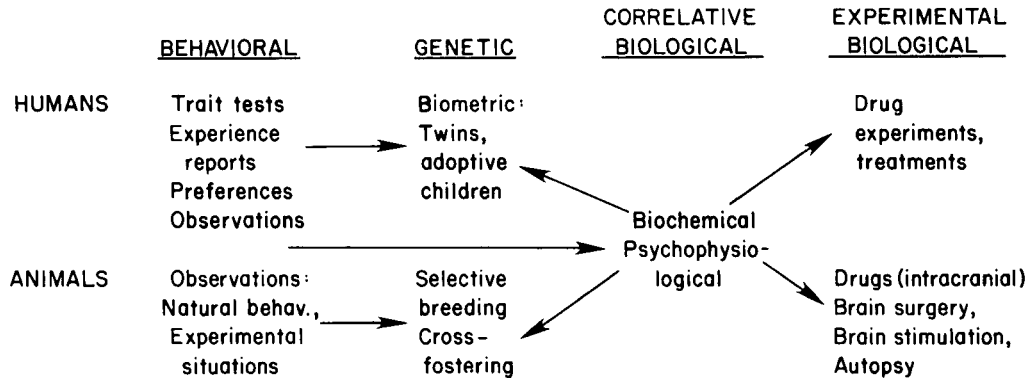

Figure 1. A model for comparative study of a personality trait showing the methods available for humans and animals (nonhuman species).

biological correlates of the behaviors seen in humans and animals. If we can show that the same biological systems are related to similar behaviors in humans and other animals, the results of experimentation on those systems in animals assumes greater significance for our understanding of humans. If we can further show that variations in the function of these biological systems are in some part genetically determined in humans and other animals, we have a potential way out of the ambiguity of causation in brain–behavior correlations with the egg (and sperm) preceding the chicken.

Another way out of the uncertainty of direction of causation in correlational results is to alter the physiological systems involved. In normal humans we are limited to nonharmful, transient alterations of physiology, usually by drugs such as amphetamine, caffeine, and nicotine that change arousal levels for a limited period. Since general cortical arousal and arousability have been significant constructs in many of the theories (Eysenck 1967; Zuckerman 1969) such experiments may be useful. An alternative way of influencing arousal is by varying the intensity of stimulation, short of extremely noxious levels, to observe the effects on psychophysiological or behavioral indicators of activation.

Once we get beyond models of arousal and address ourselves to specific neurotransmitter systems (Gray 1982; Zuckerman 1979b) such drugs are less useful because of their nonspecificity and short duration of effect. The study of the effects of drugs given to clinically disturbed humans over long periods is another source of knowledge. However, there is a problem here in establishing baselines of behavior and physiology that preceded the manifestations of the clinical state. Ideally, it would be preferable to study the effects of the drugs on normal humans whose personality and physiological status had been assessed before administering the drug, but such experiments have been difficult to do outside special medical research settings.

Using other species we have the full range of biological techniques available such as neurological and chemical lesioning and direct measure of brain levels (from autopsy) of the crucial neurotransmitters and enzymes. In humans our measures of brain neurochemistry are indirect, depending largely on assays of metabolites found in urine, blood, or cerebrospinal fluid. The results of one such study involving all three sources of these measures in the normal human is reported in the section "Monoamines and human personality."

Sensation seeking in humans

The sensation-seeking scales (SSS). The first sensation-seeking scale (Zuckerman, Kolin, Price & Zoob 1964) was developed in an attempt to find a personality predictor of individual variation in response to the experimental condition of sensory deprivation. The test was based on the constructs of optimal levels of stimulation (OLS) devoted to maintaining optimal levels of arousal (OLA). Both Zuckerman's (1969) theory of sensory deprivation and sensation seeking and Eysenck's (1967) theory of introversion–extraversion suggested that the typical activities associated with these traits represented attempts to increase or reduce stimulation, in order to maintain an *optimal level of arousal.* In Eysenck's (1967; 1981) theory extraverts are seen as chronically underaroused individuals who seek stimulation to increase their levels of arousal to a point that is hedonically positive for them. The first sensation-seeking scale (SSS, form II) was developed specifically around the OLS concept. Items loading on a broad general factor included those reflecting an interest in exciting and risky sports and activities (now called "thrill and adventure seeking") and an interest in stimulating the senses through music, travel, food, and drugs (now known as "experience seeking").

Later factor analyses (Zuckerman 1971, American subjects; Zuckerman, Eysenck & Eysenck 1978, English subjects) rotated the factors and consistently revealed four factors using expanded forms of the test:

1. *Thrill and adventure seeking* (TAS): the seeking of sensation through risky but exciting sports and other activities such as fast driving.
2. *Experience seeking* (ES): seeking sensation through the mind and the senses and through a nonconforming life-style.
3. *Disinhibition* (Dis): the seeking of sensation through social stimulation and disinhibition through social drinking.
4. *Boredom susceptibility* (BS): an aversion to monotonous, invariant situations and restlessness when exposed to such situations.

The most recent form (V) of the SSS (Zuckerman et al. 1978) contains 10 items from each of the factors, with a total score that includes all 40 items. Prior to this a general scale was developed in the first form (II) and was also used in form IV (Zuckerman 1971) in addition to the four factor-derived scales.

Subsequent factor analyses in Australia (Ball, Farnill &

Wangeman 1983) and America (Stewart & MacGriffith 1975) have revealed essentially the same four factors. S. B. G. Eysenck and H. J. Eysenck (1977) have adopted many of the items from the TAS scale in a scale they call "venturesomeness," and Schalling, Edman, and Åsberg (1983) have constructed a scale called "monotony avoidance" that resembles the BS scale.

SSS internal and retest reliabilities are high, especially for the total score for which internal reliabilities are about .85, and 3-week retest reliability is .94 (Zuckerman 1979b). Subscale internal reliabilities range from .6 (for the BS scale) to .8 (for the TAS scale).

Despite the fact that the scales were derived from orthogonal factors, shared item loadings produced scales that are low to moderately (.2 to .4) intercorrelated. This was the basis for constructing an additive total score in Form V.

Relationships to primary dimensions (Eysenck's) of personality. Eysenck (1967) sees personality as organized along three primary dimensions: extraversion (E) versus introversion, neuroticism (N) versus emotional stability, and degree of psychoticism (P) (tough-minded aggressiveness). He regards narrow traits such as impulsivity and sociability as components of the higher order traits, such as extraversion. Impulsivity can, in turn, be divided into a narrow impulsivity trait (acting quickly on impulse) and venturesomeness (sensation seeking). The actual correlations between the sensation-seeking scales and Eysenck's higher order factors reveal something more complex than a simple hierarchical arrangement. Sensation seeking has a low to moderate correlation (.3 to .4) with the E and P dimensions and with impulsivity and does not correlate at all with the N dimension (Eysenck & Zuckerman 1978). Sensation seeking seems to constitute a dimension lying between two of Eysenck's primary dimensions, E and P (perhaps closer to P) rather than representing a component trait of either. The question of which are the primary orthogonal dimensions of personality cannot be answered by factor analyses of tests alone but must involve comparative alignments of test-defined dimensions with biological dimensions of personality (Gray 1983; Zuckerman, Ballenger & Post, 1984).

Validity of the SSS: Phenomenal correlates. The development of reliable scales for a personality dimension is only a first step. The second is to demonstrate that the test has significant relationships to behavioral experience in natural environments and predicts response in experimental situations. Twenty years of research have amply demonstrated the validity of the SSS in these respects (Zuckerman 1979b). Concurrent validity studies have shown the test to be related ($r = .55$) to peer evaluation of the trait (Carll 1980). Studies have shown that the test-measured trait is also related to: variety of sexual experience; use of illegal drugs, and to variety of drugs used within drug-using populations (Zuckerman 1979b; 1983c); food preferences and driving habits (Zuckerman & Neeb 1980); engaging in risky sports (Zuckerman 1983b) and some aspects of risk taking in those sports (e.g. accident proneness among skiers); cognitive, perceptual, and aesthetic preferences such as preference for complexity, tolerance for ambiguity, originality, and creativity; richness of imagery and dreams; vocational interests and choices; and liberal, permissive, and nonconforming attitudes (see Zuckerman 1979b; 1983a for current summaries of this research).

Predictive validity studies have shown that in any kind of confined monotonous condition high sensation seekers become measurably restless compared to low sensation seekers (Zuckerman, Persky, Hopkins, Murtaugh, Basu & Schilling 1966). In short-term deprivation experiments, sensation seekers pressed buttons at a high rate for stimulation (Lambert & Levy 1972) but, when given the choice, preferred activity to viewing dull slides as a reward (Hocking & Robertson 1969). In a more stimulating confinement with another person and with access to stimulation materials, it was the *low* sensation seekers who reported stress and showed it in biochemical measures (Zuckerman, Persky, Link & Basu 1968).

The SSS also predicts volunteering for many kinds of unusual experiences or activities such as sensory deprivation and hypnosis (Zuckerman, Schultz & Hopkins 1967), drug research (Bone, Cowling & Choban 1974; Zuckerman 1979b, 1983c), encounter groups (Stanton 1976), meditation training (Myers & Eisner 1974), alpha control training, sensitivity groups, and gambling classes (Zuckerman 1974), or simply for unspecified "research" (Kohn, Hunt, Davis & Cowles 1982).

Although the trait of sensation seeking is not related to social anxiety or neuroticism, the thrill and adventure seeking subscale in particular is negatively related to fear of physical harm. In one study, the TAS score predicted actual behavior in three potentially phobic situations involving exposure to heights, a snake, and darkness; the low sensation seekers demonstrated greater fearfulness in these situations (Mellstrom, Cicala & Zuckerman 1976). In studies of anticipated risk the low sensation seekers showed high risk appraisals of a variety of situations and anticipation of greater fear and less pleasure if they were actually exposed to those situations (Zuckerman 1979a).

Arousal. As mentioned earlier, Eysenck's (1967) model for extraversion and an earlier model for sensation seeking (Zuckerman 1969) assigned a central role to individual differences in arousal and arousability as regulated by the sensitivity of the reticulocortical activating system. These theories were built on the concept of the reticulocortical system acting as a "homeostat" (Lindsley 1957), regulating the tonic level of arousal in the cortex to accommodate performance requirements (Hebb 1955) and maintain a positive hedonic state (Berlyne 1971).

The concept of a single general nonspecific arousal system is an oversimplification since there are a number of arousal systems that directly or indirectly have the ultimate effect of cortical activation (Zuckerman & Como 1983). Besides the ascending reticulocortical system there are the diffuse thalamocortical system, the sympathetic branch of the autonomic system, the pituitary-adrenocortical system, and the monoamine systems of the limbic brain. Previously it was assumed that the limbic system affected cortical arousal only through collaterals to the reticulocortical system, but Routtenberg (1968) suggested that a "second arousal system," originating in the reward areas of the limbic brain, had its own pathways of cortical arousal. Anatomical and behavioral studies (Aston-Jones & Bloom 1981) have supported this hypothesis.

Sensation seeking has not generally been shown to have any relation to tonic levels of arousal, as assessed by EEG (Cox 1977) or skin-conductance levels (Cox 1977; Neary & Zuckerman 1976; Ridgeway & Hare 1981). As Gale (1981) has pointed out, it is difficult to assess the characteristic tonic level of arousal since the procedures and instructions of any experiment are actually stimuli that affect arousal and may interact with the personality trait evaluated.

For a number of years we have questioned basing sensation seeking on individual differences in arousal. Apart from the failure to find differences between high and low sensation seekers in tonic arousal levels, other kinds of phenomenal correlates raised questions about the theory. Although high sensation seekers use a variety of drugs, such as amphetamine and cocaine, that raise arousal levels, and even show some evidence of favoring these drugs (Carrol & Zuckerman 1977), the fact remains that they also use drugs, such as opiates and barbiturates, that lower arousal levels. Furthermore, high and low sensation seekers do not show a differential response in mood or performance in reaction to stimulant or depressant drugs (Carrol, Zuckerman & Vogel 1982).

Orienting, defensive, and startle reflexes. When we speak of arousal as a phasic response we must further clarify the term. Simple tones may elicit *orienting*, *defensive*, or *startle* types of heart-rate responses depending upon their novelty, intensity, expectedness, and rise time (Graham 1979; Sokolov 1963). Each of these "arousal" reactions has distinct physiological and behavioral characteristics that are adaptive to the stimulus characteristics. Neary and Zuckerman (1976) found that high sensation-seeking males gave stronger electrodermal (skin-conductance) responses to novel visual and auditory stimuli of moderate intensity. Differences were not found for females. Although some replication of these electrodermal results was reported by Feij, Orlebeke, Gazendam, and van Zuilen (in press) and Stelmack (1981), failures of replication were reported by Cox (1977) and Ridgeway and Hare (1981) and in several unpublished studies including a recent one by Como (1984).

The electrodermal response is poor for differentiating these three types of reflex. The heart-rate response is better for this purpose because of its biphasic nature. Heart-rate deceleration defines the orienting response (OR), and acceleration is part of the defensive reflex (DR) and startle reflex (SR). Further distinctions between DR and SR can be made on the basis of the rise time and intensity of the stimulus and the habituation of the response (SRs show rapid habituation).

Three studies (Como 1984; Orlebeke & Feij 1979; Ridgeway & Hare 1981) indicate that high and low disinhibition-type sensation seekers show two kinds of cardiac arousal in response to moderately high intensity stimuli: The highs tend to show ORs and the lows DRs or SRs, or some mixture of these responses to the same stimuli. Figure 2 shows the responses of high and low scorers on the SS disinhibition to an 80 db tone in the Orlebeke and Feij study.

The orienting reflex is thought to be related to interest in and attention to the stimulus and to reflect an attitude of "stimulus acceptance" (Lacey 1959). The DR is typically elicited by intense or noxious stimuli and is believed to indicate an attitude of "stimulus rejection."

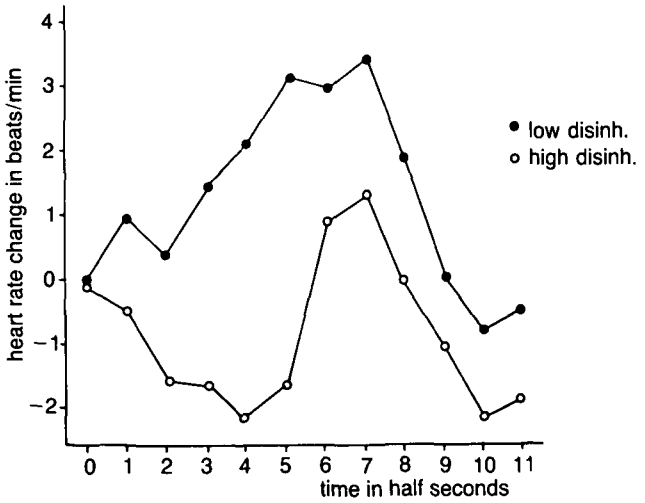

Figure 2. Phasic heart-rate response to an 80 db tone for subjects classified as low and high disinhibitors, averaged over the first three trials (from Orlebeke & Feij 1979; copyright 1979 by Lawrence Erlbaum Associates).

Schneirla (1959) suggested that the tendencies of many species to approach a retreating object and withdraw from a rapidly approaching one are based on the augmented arousal produced by the increasing retinal image and the reduced arousal produced by the diminishing image of the retreating object. Gale, Spratt, Chapman, and Smallbone (1975) have shown that cortical arousal increases as a function of the approach of another person. Bernstein, Taylor, Austen, Nathanson, and Scarpelli (1971), using nonmeaningful stimuli, found that apparent movement of an object toward the observer produced stronger and more persistent ORs than either movement away or simple change in size. Movement away produced stronger initial ORs than simple changes in size, but these differences habituated quickly.

It must be emphasized that in all studies comparing high and low sensation seekers the differences in OR are found only on the first one or two trials when the stimulus is relatively novel. At some level, the differences may reflect the biological propensity of novel stimuli to elicit interest and "seeking" in high sensation seekers, and lack of interest and avoidance of novel stimuli, persons, or situations in the low sensation seekers. To understand the meaning of these differences, however, we must examine the early stages of information processing at a higher level in the nervous system, the cortex.

Cortical evoked potentials. Buchsbaum and Silverman (1968) developed a procedure using cortical evoked potentials (EPs) to measure a dimension of personality called *augmenting–reducing* (Petrie 1960). Petrie had used a psychophysical procedure, the kinesthetic aftereffect (KAE), in an attempt to measure individual differences in the relative amplification or inhibition of somatosensory effects produced by tactual estimates of width before and after rubbing a block of wood. Buchsbaum's method (Buchsbaum & Pfefferbaum 1971) consists of administering a range of stimulus intensities

(usually visual or somatosensory stimuli) and measuring the amplitudes of the P_1–N_1 component of the EP at each intensity. The slope of the least squares plot between stimulus intensity and EP amplitude for an individual constitutes one of the measures used for the EP measure of augmenting-reducing. High positive slopes define the augmenting end of the continuum, and high negative slopes define the reducing end. Typically the major differences between augmenters and reducers occur at the highest stimulus intensities where augmenters continue to show increased EP amplitudes whereas reducers show a marked reduction in EP amplitude relative to the level of the next lower intensity.

Buchsbaum (1971) first reported some evidence of a positive relationship between sensation seeking as measured by the SSS and augmenting: High sensation seekers tended to be augmenters, and lows tended to be reducers. Zuckerman, Murtaugh, and Siegel (1974) compared high and low sensation seekers on augmenting of the EP in response to stimulation of the visual system (flashing lights) with the results shown in Figure 3. The highest correlation of augmenting was with the disinhibition subscale, the one that was also most highly related with the heart-rate deceleration and acceleration (see previous section).

Since these two studies were done, four more studies have replicated the relationship between sensation seeking and augmenting of the EP: Como (1984), Coursey, Buchsbaum, and Frankel (1975), Lukas (1982), and von Knorring (1981). Coursey et al. and Como found the relationship between augmenting of the auditory EP and sensation seeking; all but Coursey et al. found the relationship between the visual EP and SS. Von Knorring, like Zuckerman et al. (1974), used form IV of the SSS and like them found the strongest relationship between the disinhibition subscale and augmenting, although augmenters and reducers were also significantly different on the general scale which in the Coursey et al. study was related to sensation seeking too. Lukas and Como used only the disinhibition subscale to define their independent groups. The von Knorring study was done in Sweden, using a Swedish translation of the scale (an indication of the robustness of the finding across populations). Barratt and Patton (1983) also found a relationship between EP augmenting and impulsivity, a trait related to disinhibition. Both EP augmenting and high scores on the disinhibition scale have been found in sociopaths, delinquents, and drug users and in persons disposed to hypomanic states (Zuckerman 1978b; Zuckerman, Buchsbaum & Murphy 1980; Zuckerman & Neeb 1979).

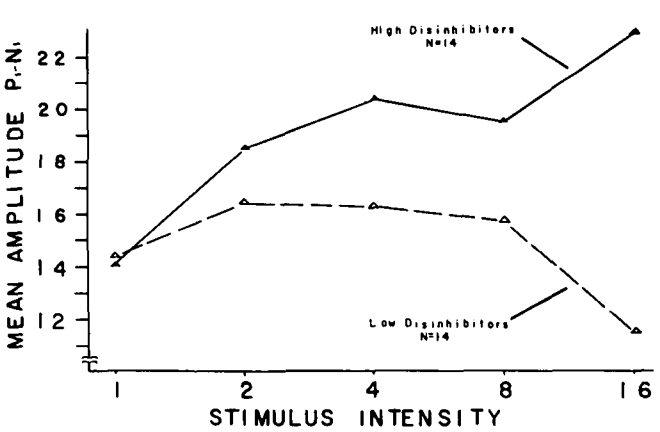

Figure 3. Mean evoked potential amplitudes (in arbitrary mm deflection units, each unit = .42 V) for low and high disinhibition scorers at each level of stimulus intensity (from Zuckerman, Murtaugh & Siegel 1974; copyright 1974 by The Society for Psychophysiological Research, reprinted with permission of the publisher).

The augmenting-reducing measure qualifies as a biological marker suitable for a comparative analysis of sensation seeking. Hall, Rappaport, Hopkins, and Griffin (1970), and Lukas and Siegel (1977) have measured augmenting-reducing in cats and have related individual differences in the biological trait to behavioral reactions, such as responsivity to novel stimuli, that resemble the reactions of sensation-seeking humans.

Augmenting seems to represent the capacity of the cortex to respond to high levels of stimulation. Reducing resembles what Russian investigators have called "transmarginal inhibition"; in their terms augmenting is an indicator of a "strong" nervous system and reducing represents the "weak nervous system."

The convergent relationships of augmenting-reducing and heart-rate acceleration or deceleration in their relation to the disinhibition scale suggested that they might be biologically connected. Como (1984) used both the EP and heart-rate measures in his study and found a significant correlation between evoked potential augmenting and heart-rate deceleration responses. The high disinhibitors showed evidence of continuing to orient when the low scorers on this subscale were showing a predominance of defensive reactions. The low disinhibitor seems to be characterized by a protective cortical mechanism that is congruent with his stimulation-avoidance behavior. The reaction is most obvious where stimulation is both novel and intense.

Gonadal hormones. Sex differences and age declines in sensation seeking, particularly on the disinhibition and thrill and adventure seeking subscales (Zuckerman et al. 1978; Zuckerman & Neeb 1980), first suggested that gonadal hormones might play some role in the trait. The association of sensation seeking with sexual experience and traits of dominance and impulsivity and aggressiveness also suggested a relation with gonadal hormones since testosterone seems to play some role in these traits, at least in other species.

Daitzman, Zuckerman, Sammelwitz, and Ganjam (1978) correlated plasma androgen levels with the SSS form IV in two samples of males and estrogen levels with the SSS in one male sample and one small sample of female college students. The disinhibition subscale correlated significantly with androgen levels in both male samples. Estrogen correlated significantly with both the general and disinhibition scales in the male sample and with disinhibition in the female sample.

The disinhibition scale was used to select extreme high and low male scorers in a second study (Daitzman & Zuckerman 1980). This study also included a variety of other personality scales and scales for sexual attitudes and experience, and used specific assays for plasma testosterone, estradiol, estrone, and progesterone. The high dis-

inhibitors had significantly higher levels of testosterone, 17-beta-estradiol, and estrone than low scorers, confirming the findings of the first study showing significant relationships between disinhibition and both androgen and estrogens.

Testosterone, estradiol, and estrone also correlated with many of the other scales given to the subjects. To examine the broader dimensions of personality associated with gonadal hormones a factor analysis was done that included the personality and gonadal hormone variables. The disinhibition scale was not included in the factor analysis because it was used to select subjects; the general SS scale was included instead. Testosterone loaded on the positive end of a dimension labeled *stable extraversion versus neurotic introversion* and correlated positively with most of the sociability, impulsivity, sensation-seeking, and heterosexual-experience scales, defining the positive pole of the dimension, and negatively with introversion and neuroticism scales, defining the negative pole. Estradiol loaded positively on the second factor labeled *social deviancy versus social conformity.* This factor was defined at the positive end by scales from the Minnesota Multiphasic Personality Inventory (MMPI) like F (general response deviancy), schizophrenia, psychopathic deviate, and hypomania. The negative end of the factor was defined by scales measuring achievement need, conformity, and the need to make a good impression on others. Although the new form of the Eysenck's scale (EPQ) that contains the P scale was not used, this dimension seems to resemble their P dimension of personality. Both Eysenck and Gray have suggested that the P dimension might be related to testosterone, but the results of this study show a relationship with estradiol in males. Testosterone was related, instead, to extraversion.

The results of the factor analysis tend to support Gray's (1973; 1983b) hypothesis that the biologically significant dimensions of personality lie along two of the diagonal quadrants in Eysenck's dimensions, at least for this analysis of gonadal hormones. The results of a similar analysis of psychological and bioamine metabolite and enzyme variables are presented in the section "Monoamines and human personality."

Apart from sensitizing specific sexual control mechanisms and playing a role in aggressiveness and dominance traits in other species, the gonadal hormones probably regulate the metabolism of several neurotransmitters in the brain and thereby affect the general activity level. One influence on the monoamine system is through their reducing effects on monoamine oxidase (MAO), a vital enzyme involved in the degradation of the monoamines norepinephrine, dopamine, and serotonin (Broverman, Klaiber, Kobayaski & Vogel 1968). Klaiber, Broverman, Vogel, and Kobayaski (1979) found that estrogen therapy for depressed female patients lowered the elevated MAO levels in these women to 63% of their mean pretreatment levels. As is shown in the following section, lower MAO levels are associated with greater activity, sociability, impulsivity, and sensation seeking.

Monoamine oxidase. As we move into the area of brain chemistry we come up against the great "blood–brain barrier" to research on living humans; I am speaking figuratively about the methodological barrier, as well as the chemical one. In humans we must rely on peripheral indices that have uncertain relationships to the levels of the putative sources in the brain. MAO in the brain is an enzyme contained in the mitochondria of neurons in the monoamine neurotransmitter systems. MAO is a neuroregulator that controls levels of the monoamines in these neurons by degrading the neurotransmitters when they are taken up in the presynaptic neuron after being released. MAO inhibitors increase levels of the monoamines in rodent brains and increase behavioral activity (Murphy 1977).

In living humans MAO is measured from blood platelets. The relationship of platelet levels of the enzyme to concentrations in the brain is unknown; however, platelet MAO is used clinically to monitor the effects of MAO-inhibiting drugs and platelet MAO levels have been shown to be related to diagnosis and prognosis for the bipolar affective disorders and chronic schizophrenia. In addition, platelet MAO levels have been related to a variety of personality trait and behavioral variables (described in this section). It is therefore unlikely that platelet MAO levels have no relation to brain MAO and the bioamines it regulates. For the present, however, the data of platelet MAO must be regarded as an uncertain index of brain MAO, and theorizing from the findings relating the peripheral index to personality and behavior must be regarded in nearly the same way that behavior is used by some theorists as an index of hypothetical brain states. The difference is that the brain MAO is not a hypothetical variable and the actual functional relationship of platelet to brain MAO is potentially discoverable.

Platelet MAO level is a reliable trait in humans that does not change much with radical changes in clinical states, like the shift from depression to mania or from a calm state to an anxious one. Variations do occur within individuals but they are small compared to between-subject differences (Murphy, Wright, Buchsbaum, Nichols, Costa & Wyatt 1976). Human twin studies have provided evidence that MAO is strongly under genetic control (Murphy 1973; Nies, Robinson, Harris & Lamborn 1974), and the range in newborn infants (Sostek, Sostek, Murphy, Martin & Born 1981) is comparable to the range in normal adult populations (Murphy et al. 1976). Females have *higher* levels of MAO than males at nearly all ages between 18 and 75 (Murphy et al. 1976; Robinson, Davis, Nies, Ravaris & Sylvester 1971), consistent with the higher scores of males on sensation seeking and the greater incidence of depressive disorders in females and of antisocial personality types in males (also related to low MAO levels). MAO levels increase with age in human brain, platelets, and plasma (Robinson, Davis, Nies, Colburn, Davis, Bourne, Bunney, Shaw & Coppen 1972), although Murphy et al. (1976) failed to find an increase of platelet MAO with age. These sex and age differences are consistent with the sex and age differences in sensation seeking and the relation of this trait to low MAO levels.

Significant negative relationships have been found between the general SSS and MAO in two groups of males and one of two groups of females (Murphy, Belmaker, Buchsbaum, Martin, Ciaranello & Wyatt 1977; Schooler, Zahn, Murphy & Buchsbaum 1978). These investigators also found negative correlations between plasma amine oxidase (AO) and some of the sensation-seeking scales.

Unlike the previously discussed biological correlates of sensation seeking, MAO is most consistently related to the general SS scale rather than the disinhibition subscale. Johansson, von Knorring, and Oreland (1983), using a Swedish translation of the SSS with chronic pain patients, found that low-MAO patients were higher than high-MAO patients on the general and experience-seeking SS scales. Schalling et al. (1983) found near significant negative correlations between a Swedish translation of the SSS and MAO. These investigators constructed their own sensation-seeking type scale called "monotony avoidance," and this scale did correlate significantly and negatively with MAO levels. Perris, Jacobsson, von Knorring, Oreland, Perris, and Ross (1980) also found significant negative correlations between the monotony-avoidance scale and MAO in a sample of depressed patients.

In the largest study done on personality and MAO, von Knorring, Oreland, and Winblad (1983) studied 1,129 consecutive 18-year-old men drafted into the Swedish army in an enlistment center. Most studies use volunteers, introducing questions of selection, but this was a total population study: 97% of the men consented to take personality questionnaires and 99% allowed blood samples to be obtained for determination of platelet MAO. The questionnaires included a modified version of the Swedish translation of the SSS V (excluding the disinhibition subscale), other personality scales, and an intelligence test. An experience questionnaire containing items about the use of drugs and alcohol and smoking habits was also given to the recruits. Low MAO subjects were significantly higher than high MAO subjects on the SSS modified total score, and on boredom-susceptibility scales and the Karolinska Hospital Personality Inventory (Schalling & Åsberg, in press) impulsivity and monotony-avoidance scores. Eysenck's extraversion score did not differentiate the MAO groups. Low MAO subjects showed more signs of alcohol and marijuana dependence. A discriminant analysis, which correctly classified 71% of the subjects into high- and low-MAO types, revealed that the SSS was the only personality test contributing significantly to the discrimination. In a stepwise multiple regression analysis the sensation-seeking scales were the most important variables in the relationship with MAO.

Despite the negative findings relating extraversion to MAO, some other studies have found significant relationships in Swedish male students and schizophrenics (Gattaz & Beckmann 1981) and middle-aged German men (but not women) in a rural population (Demisch, Georgi, Patzke, Demisch & Bochnik 1982).

The fact that MAO is less consistently related to sensation seeking and extraversion in females than males might be attributable to the effect of estrogen on MAO discussed previously. Since estrogen levels in women vary markedly as a function of the menstrual cycle, the MAO values obtained from women at different points in their cycles may be less reliable and comparable. However, the finding of the relationship in men from diverse national, age, socioeconomic, and diagnostic groups using translated scales suggests some robustness of the correlational findings.

The relationships between the traits of extraversion and sensation seeking and MAO suggest that low-MAO types are more sociable than high-MAO types. This is confirmed by Coursey, Buchsbaum, and Murphy (1979) who found that low-MAO college students of both sexes reported more time spent in social activities than high-MAO types. The low-MAO males also were much more likely to report using drugs and smoking cigarettes, as was found in the von Knorring et al. (1983) study. Low-MAO males were also more likely to have convictions for criminal offenses than the high-MAO males. All of these activities are phenomenal correlates of sensation seeking, as reported in an earlier section, and the relation to criminal convictions is consistent with the association between sensation seeking and the primary type of psychopathy (Blackburn 1978; Emmons & Webb 1974; Zuckerman & Neeb 1979) and psychopathic traits (Zuckerman 1978b).

Monoamines and human personality. The work relating platelet MAO to sensation seeking suggested the involvement of the monoamine systems, but the question of which systems are involved, in what direction they might be related to sensation seeking, and whether the crucial factor was the regulation by MAO, the levels of the bioamine itself, or the enzymes involved in its production, could not be answered. The hypothesis (Zuckerman 1979b) was: "Sensation-seeking trait is in some part a function of the catecholamines norepinephrine and dopamine in the reward areas of the limbic system, as well as the neuroregulators that control their availability at the synapse" (p. 372).

Actually, the prediction followed the Stein (1974; 1978; 1983) and Crow (1977) hypothesis, in that dopamine was thought to sensitize the high-activity and exploratory tendencies, whereas NE was thought of as related to the expectation of positive reinforcement in novel situations and the consequent willingness to take risks.

The first encouraging finding came from a study by Buchsbaum, Goodwin, and Muscettola (1981) who found a positive and significant correlation between levels of urinary MHPG, a metabolite of NE, and sensation seeking in a sample of 10 normal subjects. The positive correlations were found during both a baseline and a stressful period. Trait depression did not relate significantly to MHPG in either period, but was related to the change from baseline to stress. No correlation between the SS trait and MHPG was found in a group of 12 patients with affective disorders.

A study by Umberkoman-Wiita, Vogel, and Wiita (1981) examined the relationships between sensation seeking and several blood measures, including dopamine-beta-hydroxylase (DBH), the enzyme that converts dopamine to norepinephrine in the NE neurons in the brain. Serum DBH was negatively and significantly related to sensation seeking. Of course, the relationship between serum DBH and DBH in NE neurons in the brain is unknown, but if there is a relationship, and if plasma NE reflects the concomitant release of DBH along with NE when the neuron fires, we would have to conclude that high sensation seekers had either a low NE production rate or a low rate of activity in the system. This finding was incongruent with the direction of the relationship proposed by the theory, and the Buchsbaum et al. (1981) finding regarding urinary NE. It assumed greater significance as a consequence of the next study.

An interdisciplinary study done at the National In-

stitute of Mental Health is probably the most ambitious investigation of bioamines in normal individuals to date (results reported in Zuckerman, Ballenger, Jimerson, Murphy & Post 1983). A variety of neurotransmitters or their metabolites and various neuroregulators were obtained from cerebrospinal fluid (CSF), blood, and urine in a psychiatrically screened group of normal male and female subjects. Diet and activity factors were controlled prior to the lumbar puncture and blood and urine collections. Subjects were given a battery of tests that included the SSS and Eysenck's EPQ.

High correlations between CSF and plasma measures of MHPG (r = .74) and DBH (r = .71) as well as moderate correlations (about .5) between most of these variables and CSF-NE suggested that all of these variables were assessing a common system, perhaps that originating in the locus coeruleus (LC) of the brain. Such an assumption would be consistent with findings that stimulation of the LC in rats produces increases in lumbar CSF and plasma MHPG (Crawley, Roth & Maas 1979). CSF-NE may represent the results of release from the LC since tracts from these neurons descend directly into the spinal cord. Maas and Leckman (1983), in a review of the recent literature showing significant correlations between central and peripheral levels of NE and MHPG, have suggested that central and peripheral adrenergic neurons function as an interactive unit.

Correlations with the SSS, partialing out the influences of age and body size (height and weight), replicated the Umberkoman-Wiita et al. (1981) negative correlation between plasma DBH and sensation seeking in the total group, and in males and females separately. The study did not replicate the Buchsbaum et al. (1981) correlation between urinary MHPG and sensation seeking. Although the SSS did not correlate significantly with other NE metabolite (MHPG) measures in CSF or plasma, it was significantly correlated with CSF-NE in the total group and in males and females separately. Figure 4 shows a scatterplot of this important correlation. It is apparent that this linear relationship is *not* produced by a few extreme cases.

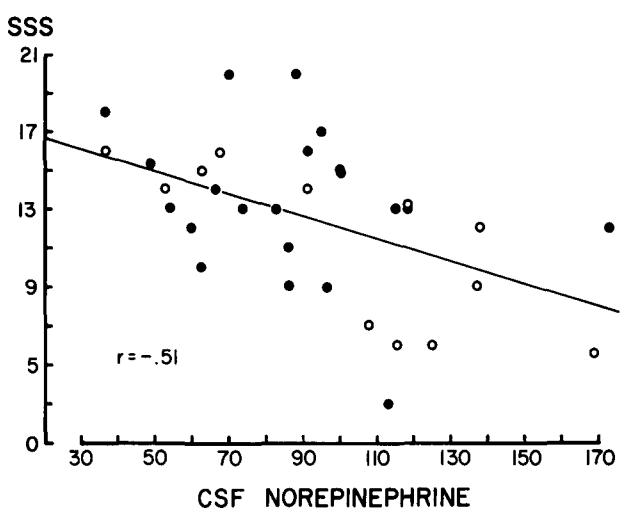

Figure 4. Scatterplot of scores on the general sensation-seeking scale (SSS) vs. CSF norepinephrine. Solid circles are males and open circles are females (from Zuckerman et al. 1983; copyright 1983 by Lawrence Erlbaum Associates).

Extraversion-introversion did not correlate with measures of activity of the monoamine systems but did correlate with a CSF calcium measure. The extraversion scale from the EPQ correlated positively and the social introversion scale from the MMPI correlated negatively with CSF calcium. However, the N scale from the EPQ, as well as neuroticism scales from the MMPI, also correlated negatively with CSF *and* serum calcium. A factor analysis (Zuckerman et al., 1980) revealed that the dimension defined by calcium measures went from stable extraversion to neurotic introversion. Again (like the hormonal data) these data confirm Gray's idea that significant biological dimensions are more likely to be found at the diagonal between Eysenck's two dimensions of extraversion and neuroticism.

The results of this essentially exploratory study raise more questions than they answer. The replicated findings regarding plasma DBH and the new findings relating CSF-NE to sensation seeking certainly implicate the NE system originating in the locus coeruleus in sensation seeking, but the direction of the relationship was not anticipated. To what extent is CSF-NE a biological trait or state? CSF DBH seems to be a little of each in relation to anxiety, with those *low* in trait anxiety responding on the day of the lumbar puncture with *high* levels of DBH activity, and those independently *high* on state anxiety that day also showing correlated *high* DBH levels. CSF-NE has been observed to react to changes from depression to mania (high NE) in patients, but in the current study it did not correlate with the trait or state anxiety, making it unlikely that either trait or state anxiety influenced the relationship between sensation seeking and CSF-NE.

Neuroregulators, bioamines, and augmenting–reducing of the cortical evoked potential. Negative correlations between platelet MAO levels and augmenting of evoked potentials (EPs) have been found in patient groups (Buchsbaum, Landau, Murphy & Goodwin 1973) and in students showing signs of affective disorder (Haier, Buchsbaum, Murphy, Gottesman & Coursey 1980), but such correlations were not found in normal subjects in the Haier et al. study.

Von Knorring and Perris (1981) have found relationships between certain transmitter metabolites and enzymes and cortical augmenting–reducing. Augmenters are characterized by low levels of serum DBH and CSF 5-HIAA (serotonin metabolite), HVA (dopamine metabolite), and endorphins. MOPEG, a norepinephrine metabolite measured in CSF, was not related to the cortical arousability measure. The results are interesting because of the negative relationships between sensation seeking and DBH, MAO, and CSF endorphin levels (Johansson, Almay, von Knorring, Terenius & Åström 1979) and the positive relationship of sensation seeking to augmenting of the EP.

Genetical analyses. A substantial genetic contribution has been found for the trait of sensation seeking (Fulker, Eysenck & Zuckerman 1980). The biometrical method of Jinks and Fulker (1970), applied to the data from 422 pair of twins, yielded a heritability figure of 58%, or 69% of the reliable variance. The data fit a polygenic model of inheritance. Detailed analyses of these data in relation to

Eysenck's dimensions are discussed by Eysenck (1983) and Martin, Eaves, and Fulker (1979). Analyses of the data suggested that subscale profiles are largely under the control of different genes in the two sexes although the general or common factor is under the control of the same genes in both sexes. Genetical influences are highest for the experience seeking and lowest for the boredom-susceptibility scale in both sexes. In males the disinhibition scale also shows a predominantly genetic influence.

Using smaller twin samples, substantial heritabilities have been shown for platelet MAO (Murphy 1973; Nies et al. 1974), plasma MHPG (Jimerson, Nurnberger, Post, Gershon & Kopin 1981), DBH (Gershon, Kessler & Bunney 1977), augmenting of the cortical evoked potential (Buchsbaum 1974) and most components of the EP (Rust 1975), and the orienting reflex (Lykken 1982). Thus, many of the biochemical and psychophysiological correlates of sensation seeking show strong genetic determination. The heritability of the behavioral trait would depend on the heritability of the biological systems governing the trait. Buchsbaum (1974; data reported in Zuckerman 1979b) has shown that the monozygotic twins who are most alike on augmenting–reducing of the EP are also most alike on the trait of sensation seeking.

Animal models

Schneirla (1959) proposed that tendencies toward approach and withdrawal constitute two broad basic behavioral tendencies in all species. In species evolving earlier and possessing simpler nervous systems these tendencies are automatic (reflexive or instinctual), as illustrated in tropisms such as attraction or repulsion to light. In organisms higher on the evolutionary scale, for whom learning and even cognition may play a larger role, the tendencies might be called *seeking* and *avoidance*. The theory of sensation seeking represents an attempt to describe individual differences in the strength of the generalized approach tendency in humans exposed to novel situations.

The open-field model. The response to novelty constitutes the heart of the definition of sensation seeking: "a trait defined by the need for varied, novel and complex sensations and experiences and the willingness to take physical and social risks for the sake of such experience" (Zuckerman 1979b, p. 10).

The open-field arena and other tests of reaction to novel environments and stimuli might provide a useful animal model for the human trait. Older rodents show less exploration of the open field than younger ones despite the fact that there are no decreases in emotionality as a function of age. Sensation seeking in humans is one of the few personality traits that declines with age. Costa, McCrae, and Arenberg (1980) have shown little or no decline in other traits over a wide age span. Behavior in the open field and similar situations depends on two dispositional traits: exploration and fear. Although factor analysis (Royce 1977; Whimbey & Denenberg 1967) and genetic studies have established the *relative* independence (in rodents) of these two tendencies, they do tend to be negatively correlated (Gray 1979), particularly after the first day of exposure to the maze (Whimbey & Denenberg 1967). The two traits clearly interact in determining behavior in novel environments. Sensation seeking and anxiety states in humans show a similar interaction in risky situations (Zuckerman 1979a). The role of fear in the open-field situation somewhat vitiates its usefulness as a model of sensation seeking since at the phenomenal level the *trait* of sensation seeking is not correlated at all with tests of social anxiety (most anxiety tests) or neuroticism, and there are only small to moderate negative correlations with tests assessing fear of *physical* harm.

Genetics of open-field behavior. Marked strain differences have been found in mice in reactions to the open field. Similar differences have been shown between inbred strains of rats (Driscoll & Bätig 1982). Figure 5 shows the different reactions of two strains of mice, BALBs and C57BLs (DeFries, Gervais & Thomas 1978). BALBs show strong fear reactions (behavioral inhibition and defecation), whereas the C57BLs show less fear and stronger exploratory (activity) tendencies in this novel situation. The defecation and activity scores of derived F_1, backcross, and F_2 and F_3 generations are also shown in this figure. The percentage of genes from the C57BL parental strain is linearly and positively related to mean activity levels and negatively related to emotionality (defecation) levels. McClearn (1959) demonstrated that strains are highly consistent in their responses to other kinds of apparatus that expose the mouse to a novel situation. Bovet (1977) showed that the great fearfulness of the BALB/c strain is also seen in its greater success in avoidance learning in a shuttle box than the C57BL/6 mice.

Mice have been selectively bred for open-field activity with considerable success. Starting with a cross between the BALB/c (low activity) and C57BL/6 (high activity) strains, DeFries et al. (1978) selected and bred separate high- and low-activity strains for 30 generations. The final generations of the two strains showed no overlap in open-field activity. As in an earlier study by Broadhurst (1975), there was a correlated response to selection for defecation

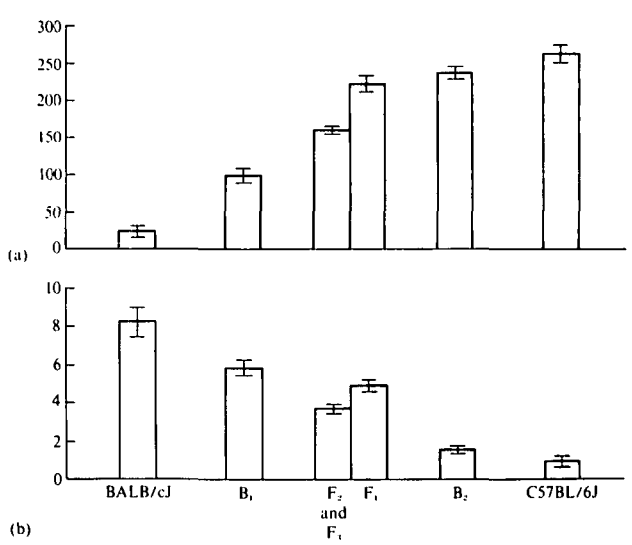

Figure 5. Mean open-field (a) activity and (b) defecation scores (± twice the standard error) of BALB/cJ and C57BL/6J mice and their derived F_1, backcross (B_1 and B_2), F_2, and F_3 generations (from DeFries, Gervais & Thomas 1978; copyright 1978 by Plenum Publishing Co.).

(emotionality) in the open field, indicating a genetic relationship between these two traits. These findings could also be used to support Gray's hypothesis that one of the fundamental biological dimensions underlying personality represents combinations of emotionality (neuroticism) and exploratory activity (extraversion). In rodents, at least, these two tendencies are negatively correlated at the genotypic and phenotypic levels.

Cross-fostering studies of the BALB/c and C57BL/6 strains, particularly those using ovary transplants to separate intrauterine and social environmental effects, have shown no effects of maternal postnatal environment on either open-field activity or defecation (Plomin, DeFries & McClearn 1980, pp. 249–53).

Mouse strain comparisons on monoamine systems. Neurochemical comparisons between exploratory and nonexploratory emotionally reactive strains of mice offers us one way to test some inferences about differences in brain chemistry related to behavior in the open field. Ingram and Corfman (1980) have provided a useful bibliographic entree into the literature. Focusing on comparisons between the exploratory, nonreactive C57BL strain and the nonexploratory, emotionally reactive BALB/c strain, we can compare levels and uptake of the monoamines in brain.

It is difficult to summarize the complex findings because of differences related to brain loci and age of the animals. What the literature suggests is that tyrosine hydroxylase, an enzyme involved in the early stages of dopamine and NE synthesis, is higher in the corpus striatum, pons-medulla, and hypothalamus of the BALB strain (Tiplady, Killiam & Mandel 1976), but levels of NE are higher in C57s in whole brain, cortex, and some limbic structures including the hippocampus (Berger, Herve, Dolphin, Barthelemy & Tassin 1979; Bernard, Finkelstein & Everett 1975; Sudak & Maas 1964). However, higher levels of NE are found in the BALBs in the hypothalamus (Eleftheriou 1974) and brain stem (Sudak & Maas 1964). The BALBs also show higher serotonin levels in the brain stem.

Perhaps the most interesting finding is that of a 36% higher number of catecholaminergic cell bodies in the locus coeruleus of the C57s compared to the BALBs (Berger et al. 1979). If we identify the BALBs with a strong anxiety or general emotionality tendency (they are also more irritable and aggressive; Bernard et al. 1975; Moisett, Hendley & Welch 1975), then Gray's (1982) hypothesis of anxiety proneness being positively related to activity of the dorsal noradrenergic system is not supported by these strain comparisons. The hippocampus, also involved in the anatomical model of Gray's system, yields higher levels of NE in C57s than in BALBs. Dopamine, in contrast to NE, is higher in the BALBs, at least in the whole brain (Bernard et al. 1975).

A higher rate of uptake of catecholamines is found in the BALBs (Moisett 1977; Moisett et al. 1975), and the inverse relation of uptake to exploratory activity (Moisett 1977) is consistent with the idea that availability of NE at receptor sites is a determinant of exploratory activity.

Obviously, these kinds of neurochemical comparisons of exploratory and nonexploratory strains of mice cannot give any definitive basis for establishing the actual role of the monoamines in the inherited traits of exploratiVeness or emotionality. The differences in neurochemistry might be produced by the activity of the mice before autopsy rather than determining that activity. Alternatively, the correlation of open-field activity and neurochemical status of brain might be produced by a third variable related to both. The experimental neurophysiological approaches discussed later provide a sounder basis for conclusions about the role of the monoamines in behavior. An optimal approach would be to study different strains in experiments in which neurophysiological variables are also varied.

Naturalistic observation in animal colonies. The open-field test may not be the best model for sensation seeking in nonhuman species for reasons alluded to earlier. Similar behavioral genetic studies of sociability, social dominance, sexuality, and other social traits may be more appropriate to an understanding of the genetic mechanisms underlying sensation seeking and could provide answers to the basic question of what is inherited when one inherits a disposition toward the behavioral trait.

Certainly this particular animal model does not incorporate all of the features of human sensation seeking; it seems to describe the thrill and adventure seeking aspect more than the others. Much of human sensation seeking occurs in social activity, as embodied in the disinhibition subscale and related traits such as dominance, sexuality, and playfulness. Ellison (1977) has studied such behavior in rats living in large colonies, noting such natural phenomena as time spent in burrows as opposed to time in areas of social interaction, and dominance, as assessed by order of precedence through a tunnel leading to food rewards. Redmond, Murphy, and Baulu (1979) have studied behavior in a monkey colony, noting naturally occurring social interaction, grooming, play, aggression, sexuality, and so forth. These investigators found that low-MAO monkeys observed in a colony tend to be more active, make more social contacts, and engage more in play activities than high-MAO monkeys. The low-MAO male monkeys are also more prone than the highs to engage in aggressive and sexual activities. MAO inhibitors that reduce MAO levels have been shown to increase general hyperactivity and to increase activity in a novel environment in rodents (Murphy 1977). MAO type B increases in the rat brain with age (Benedetti & Keane 1980; Mantle, Garrett & Tipton 1976) as it does in humans. These studies show that platelet MAO levels constitute another marker linking *similar* kinds of behavior in different species to a common biological correlate.

Although most comparative theorists are wary of discontinuities in behavior between lower and higher species, it is often assumed that the biological mechanisms are quite similar. In the case of MAO this assumption is unwarranted. On the basis of rodent studies, some investigators have claimed that the MAO type A, rather than the MAO type B found in blood platelets, is responsible for deamination of monoamines in the brain. If this were true for humans and primates the relevance of the data on platelet MAO (B type) for inferences about brain-behavioral mechanisms would be dubious. However, Garrick and Murphy (1980) have shown that the nonhuman primate brain contains predominantly (85%) MAO-B activity in contrast to the nearly equal activity of MAO-A (55%) and MAO-B (45%) in the rodent brain. Deamina-

tion of dopamine by specific MAO inhibitors for the type A (clorgyline) and B (deprenyl and paragyline) was compared in vervet, human, and rat brains. The type A MAO inhibitor accounted for a preponderance of inhibition in the rat brain whereas the type B MAO inhibitors resulted in a higher percentage of inhibition in primate brains. Since MAO in the brain is a major regulator of three monoamine systems (adrenergic, dopaminergic, and serotonergic) these data raise some questions about the validity of the vast literature studying the functions of these systems through comparative experiments done almost exclusively with rodents. There may be other discontinuities in the biological mechanisms of these systems that are still unknown. But sheer economic factors prevent most research on nonhuman primates so we must proceed warily down the comparative trail watching for pitfalls.

Animal personality. To use animal models we must be sure that there are reliable dimensions of animal behavior and that these dimensions resemble those found in humans. The dimensions of exploration–activity and emotionality–inhibition found in the open-field situation could correspond to extraversion (or sensation seeking) and neuroticism respectively. However, the open-field situation is a solitary one, and sociability, the major component of extraversion, cannot be assessed if we limit our observations to this one situation.

Chamove, Eysenck, and Harlow (1972) studied the behavior of Rhesus monkeys. Between 9 and 12 months of age monkeys were periodically observed in a cage containing 4 animals. The coded behaviors were intercorrelated and factor analyzed. The major factors found were:

1. A factor consisting of both social and nonsocial play and positive physical contact with other animals was identified by Eysenck as a *sociability factor*, although the presence of nonsociable play suggests that it could also be described as *sensation seeking*.
2. A combination of nonsocial, inappropriate, and appropriate fear responses (withdrawal) plus social exploration (anxious watching of other monkeys) constituted a factor identifiable as *fearfulness* or *neuroticism* in Eysenck's terms.
3. Both contact and noncontact hostility constituted an *aggresiveness* factor corresponding to the psychoticism or P dimension, in Eysenck's system.

The results were more complicated when animals were suddenly exposed to a single other monkey in a more controlled situation. Not surprisingly, behavior becomes more situation specific when we narrow the situation and observe a limited sampling of behaviors.

Although the results showed that "dimensions" of personality similar in form to those found in humans can be reliably identified in monkeys, the next step must be to identify common biological correlates of the traits in the two species. Playfulness, the main component of the first factor, was found to be related to low platelet MAO levels in the Redmond et al. (1979) studies of monkeys previously described, and low MAO is found in sensation seekers and extraverts.

Ethological versus experimental observation. Ethological approaches are important in understanding the role of biological factors in behavior that is natural to the species. Experimental studies of learning and extinction in response to rewards and punishment are also important but can sometimes be less enlightening, because they impose on organisms situations that are not found in their natural environments and demand responses that are not part of the species-specific repertoire for a situation (Bolles 1970). Pressing bars to obtain rewards or avoid electric shock are unfamiliar situations for most species, including the human one. Although the use of well-controlled laboratory situations is obviously of value, their *exclusive* use in behavioral biology may lead to models of behavior that distort the actual behavioral interactions with the environment and are ultimately less predictive of the natural phenomena.

Anxiety, as a human trait, is most relevant to uncertainty or expectations of negative outcomes in social situations. Pain conditioning seems largely irrelevant to human anxiety. The open-field paradigm incorporates the element of uncertainty of outcome in a potentially hostile environment but not the social element. File and Hyde (1978) have developed a model for anxiety that combines both elements of situations that typically elicit anxiety in humans. They contrast the time rats spend in social interaction in familiar and unfamiliar environments. A further degree of unfamiliarity is introduced by varying the light levels in the unfamiliar condition since rats tend to be somewhat photophobic. Social interaction is highest when rats are placed in a familiar environment under low levels of illumination and lowest in an unfamiliar environment under high light conditions. The visual gradient of decrease from the familiar to the unfamiliar is affected by ACTH and anxiolytic drugs (studies discussed in the section "Sociability and dominance") in a manner consistent with the model. What is ignored in the model is the possibility that sociability per se may be a reliable trait in rats and may interact with the anxiety trait or experimental treatments to affect the reaction of a particular rat. Although the sociability factor is somewhat controlled by contrasting the levels of social interaction in the familiar and unfamiliar environments, the initial levels in the familiar one would be bound to affect the degree of decrease produced in the unfamiliar one.

Despite its drawbacks, the experimental model seems to be isomorphic with the human situation and yet respects the species-specific response characteristics of the animal used.

Studies of the monoamine systems in animals

Apart from the problems in extrapolating from platelet MAO to brain MAO, the problem of establishing the functional relationship of the monoamine neurotransmitters to adaptive behavior mechanisms is formidable. It is unlikely that there is a single trait, such as fearfulness or sensation seeking, that is associated with a single specific neurotransmitter. To the extent that a neurotransmitter such as norepinephrine or serotonin mediates general arousal or inhibition it could play a role in several behavioral systems. There is also the fact of interaction between systems at the behavioral level (e.g. fear vs. exploration) and at the biochemical level (norepinephrine vs. serotonin). To quote Goldberg and Silbergeld (1977):

The separation and assignment of precise roles for each

> of the monoamine neurotransmitters in control of behavior [are] very difficult. The use of chemical agents has helped to distinguish these processes, but investigation of these treatments has revealed that in some cases the neurochemical depletions of the various transmitter compounds are not as specific as originally supposed. No clear interactive system prevails and indeed the relationship among these systems probably depends upon the level of functioning of these systems. (p. 377)

One further complication is that the neurotransmitters may have different behavioral effects in different loci of the brain. Plaznik and Kostowski (1983), for instance, have discussed the dissociation of noradrenergic and dopaminergic neuronal systems and the dissociation of the dorsal noradrenergic (NA) bundle and ventral noradrenergic bundle in their interactions with other monoamines and their behavioral relationships. Although NA effects often complement those of dopamine and are reciprocal to those of serotonin, this is mainly true for the dorsal bundle NA tract and the locus coeruleus. Ventral bundle lesions seem to show an opposite function for this branch of the NA system: a complementary influence on serotonin and a reciprocal influence on dopamine function. Studies that have applied a chemical lesioning agent like 6-hydroxydopamine (6-OHDA) nonspecifically may indiscriminately affect all of these catecholamine systems and therefore not tell us much about which system is really mediating behavioral changes. Specific applications of such lesioning agents to particular areas may be better.

In a previous paper (Zuckerman et al., 1984) I discussed the different theories hypothesizing one or several functions for each of the monoamine systems (norepinephrine, dopamine, and serotonin) considered separately. An approach that examines the relevant animal behaviors in relation to the monoamines may have greater value in clarifying the interactions between the biological systems. Here I focus primarily on behavioral characteristics that are likely models for sensation-seeking behavior in humans: general activity, exploratory activity, sociability, dominance, sexuality, consummatory behavior, and cortical self-stimulation.

General activity. General activity level has been suggested as a basic dimension of human temperament (Buss & Plomin 1975). Both direct and indirect evidence indicates that activity level is a component of sensation seeking. Zuckerman et al. (1966) found that high sensation seekers in restricted environmental confinement were distinguished by spontaneous activity that was not related to subjective stress. Given a choice of visual stimulation or opportunity for activity in a sensory deprivation experiment, high sensation seekers, in contrast with lows, tended to choose activity (Hocking & Robertson 1969). Infants born with low platelet MAO levels exhibited more activity during the first 72 hours after birth than high-MAO infants. Hyperactivity is associated with two clinical syndromes that are characterized by extreme sensation-seeking behavior: minimal brain dysfunction (MBD) in children and episodes of mania in bipolar affective disorders. The latter clinical condition has been directly related to sensation seeking (Zuckerman & Neeb 1979) and to augmenting of the EP (Buchsbaum et al. 1973) and low MAO levels (Murphy & Weiss 1972). A perusal of the SSS items shows several indicating extreme restlessness in monotonous conditions.

Goldberg and Silbergeld (1977) suggest that "hypermotility may be associated with increased functioning of one or more of the monoaminergic pathways and decreased functioning of cholinergic and GABA-nergic systems" (p. 377). One type of evidence supporting this view is that isolation hypermotility in rats is accompanied by increased excretion of urinary MHPG (Speiser & Weinstock 1976). Taken alone, this finding might simply indicate that activity stimulates release of NE, but other studies show that intracerebral (Benkert 1969) and intraventricular (Segal, Geyer & Weiner 1975) injections of NE stimulate activity. Dopamine or d-amphetamine injected into nucleus accumbens and tuberculum olfactorium parts of the mesolimbic dopamine system also increases activity, but no increases are seen when dopamine is injected into the striatum (Pijnenburg, Honig, van der Heyden & van Rossum 1976). These last data suggest that the behavioral effects of dopamine, and perhaps other monoamines, probably vary with locus in the monoamine systems. The effects may also vary with genetic dispositions. Segal et al. (1975) found marked differences between two strains of inbred rats in response to intraventricular injections of NE and d-amphetamine, suggesting that one strain showed greater release and receptor sensitivity.

Other evidence for the hypothesis is that amphetamine in *low doses* increases activity and is thought by some to release and block reuptake of dopamine and NE. These findings are consistent with the effects of low doses of amphetamine in humans and the relative attractiveness of stimulant drugs for high sensation seekers (Carrol & Zuckerman 1977). Chronic amphetamine users, however, often pass through this phase of amphetamine effect, becoming seclusive and showing a very restricted range of activity. Some develop paranoid psychoses with anxiety, delusions, and hallucinations (Griffith, Cavanaugh & Oates 1970). Eysenck (1963) has hypothesized, in line with his arousal hypothesis of introversion–extraversion, that chronic amphetamine use tends to increase introversion. Ellison (1979) has shown in an animal model that the effects of amphetamines on behavior and the dopamine systems depend on the phase of amphetamine use. Using slow-release silicone pellets planted under the skin that release amphetamine continually for 10 days, they found four stages of effect: the first stage (0–6 hours) is characterized by enhanced locomotion in rats, more frequent tendency to approach humans, and more time spent out of burrows; in the second stage (6 hours to 3 days) exploratory behavior becomes more circumscribed and stereotyped; in the third phase (4–5 days) the rats are back in their burrows and tend to show spontaneous startle reactions (we might say that they have gone from impulsive extraversion to anxious introversion); in the final phase (5–7 days) they come out of their burrows and engage in excessive and violent aggressive behavior, often picking a particular victim in a kind of "pair-bonded aggression." The changes in the effects of amphetamine were found to be accompanied by damage to dopamine terminals and a reduction of tyrosine hydroxylase in the caudate nucleus. Thus, whereas the initial enhanced activity following low-dose amphetamine stimulation is

most probably a function of increased catecholamine release, the later reduction and stereotyping of activity may be based on a depletion of dopamine resulting from damage to dopamine terminals.

These findings in rats show a similarity to the effects of administering small frequent doses of dextroamphetamine to human volunteers over a five-day period (Griffith et al. 1970). A period of mild euphoria and sociability was followed by one of reduced activity and sociability, hypochondriacal symptoms, depression, and irritability. Six of the nine subjects developed a blatant paranoid psychosis characterized by ideas of reference and delusions of persecution, and two others were taken off the drug when the prepsychotic symptoms first appeared.

Goldberg and Silbergeld (1977) have used a similar explanation for the data showing a transitory hyperactivity produced in rats through destruction of catecholamine pathways by 6-OHDA or electrolytic lesioning of dopamine cell bodies in the substantia nigra. They suggest that the transmitter is first released from damaged terminals, then exhausted in the system; this is followed by supersensitivity of receptors and eventually neural regeneration. Clearly, the lesioning studies may produce contradictory results as a function of when the animal is studied after lesioning.

Ellison (1977) has used lesioning of NE (using 6-OHDA injected into the lateral ventricles) and serotonin systems (5, 6 dihydroxytryptamine in the ventricles) to examine effects on the behavior of rats in a colony. Ellison claims that multiple small injections produce more specific effects on the target systems; levels of amines in the brain after sacrifice confirmed this conjecture. NE-system-lesioned animals tended to be quite inactive, spending most of their time in their burrows, whereas serotonin-system-lesioned animals were quite aroused, exploratory, and active in their natural habitat, spending much of their time out of their burrows and interacting with other rats in the "commons." However, dramatic reversals of typical behaviors were found for the same animals in a novel environment (the open field); these are discussed in the next section.

Depletion of serotonin using electrolytic or chemical lesioning has not produced consistent effects on spontaneous motor activity in familiar environments or running wheels (reviewed in Davis 1979). More naturalistic observations suggest an *anxious* type of hyperactivity. Kostowski, Giacalone, Garattini, and Valzelli (1968) describe their electrolytically lesioned rats as showing "increase of spontaneous motor activity including running around the cage, aimless rotatory or across cage movements, occasionally stereotyped sniffing and licking, slight tremor and rigid posture of the extremities. Hypersensitivity to external stimuli such as clicks and touching was also observed" (p. 373). File (1975) describes serotonin-depleted male rats as "typically jumpy, aggressive and hypersexual" (p. 982). Soubrie (in preparation) suggests that serotonin mediates behavioral inhibition but is not related to central anxiety states. Low 5-HIAA levels (CSF) in humans are associated with aggressiveness *or* suicide attempts, but not with lowered anxiety.

Panksepp (1982) and Ellison (1977) regard NE and serotonin as antagonistic in their effects on arousal of all emotional systems. However the behavioral effects of arousing a fear system are typically inhibitory (Gray 1982; see also multiple book review, *BBS* 5(3) 1982) in contrast to the arousal of expectancy (reward) systems. If these neurotransmitters activated all emotional systems we would expect to see mixed effects on activity depending on levels of arousal and the situational provokers. Perhaps fear and reward systems have different thresholds of response to general arousal by catecholamines, moderate levels being associated with reward and higher levels with fear.

Both Crow (1977) and Stein (1974; 1978) have drawn a distinction between the roles of dopamine and NE in their effects on behavioral arousal. These authors regard the dopaminergic system as one that energizes behavior directed toward primary biological rewards, activating search, and exploration. NE is seen as guiding response selection in line with previously rewarded behaviors. Crow (1977) views serotonin as a general inhibitor of behavior, pointing out that lesions of dorsal and median raphe nuclei, which reduce serotonin, increase activity. Both Crow and Stein, as well as Gray (1982), see serotonin as subserving the behavioral inhibition associated with signals of punishment. However, Gray also sees the NE system as involved in sensitization to threatening (fear) stimuli, whereas Crow and Stein view serotonin as the sole mediator of the fear response. Redmond (1977; see also Redmond & Huang 1979a), like Gray, regards the dorsal noradrenergic system as an "alarm system," functioning as a "novelty detector, stimulus enhancer or attention provider" at low levels of activity and a mediator of fear at high levels of activity. Redmond's conclusions are based in part on studies involving direct stimulation of the locus coeruleus in monkeys which produced typical fear reactions, otherwise seen in monkeys in response to threat. It should be noted that the intensities of neuronal stimulation used may be in excess of what would be produced by natural stress. Also, these animals were necessarily restrained, a condition that usually enhances fear and inevitably prevents exploration.

Mason, Roberts, and Fibiger (1978) have found that lesions of the dorsal bundle increase fear reactions in rats in certain behavioral situations. Mason and Fibiger (1979) have argued that NE only mediates response to novel stimuli and is associated with fear only to the extent that the stimuli presented have some association with fear. Their view is supported by the work of Aston-Jones and Bloom (1981) who recorded neuronal activity in the locus coeruleus in *unrestrained* animals. They found that the locus coeruleus responds to novel *nonnoxious* stimuli and habituation of response with repetition of stimuli. Furthermore, spontaneous activity in this NE center varies with stage of arousal, being lowest in non-REM sleep and highest in waking, alert, active states. Their results suggest a reconciliation of the reward versus fear hypotheses of NE function. As a general arousal system alerting the organism to significant (novel) environmental cues the system may serve either fear or reward functions. If there is any intrinsic relation between level of arousal and emotions, as suggested by optimal level of arousal theory, then high activation of the system might produce fear whereas moderate arousal might be rewarding. Similarly, high levels of arousal may activate the "behavioral inhibition system" (Gray 1982) associated with fear, and low to moderate levels might activate centers impelling the

organism *toward* immediate sources of stimulation or provoke a search for stimulation (exploration).

Activity in novel environments and response to novel stimuli. Gray (1982) has maintained that the reaction to novelty is a function of the behavioral inhibition system (BIS). The BIS is neurologically identified as the septo-hippocampal system together with its neocortical inputs from the entorhinal area and prefrontal cortex and ascending inputs from noradrenergic and serotonergic systems. Parts of the Papez circuit are also involved. From this model it might be predicted that organisms with a strong BIS should not move much in the open field and should show strong autonomic arousal, orienting responses, and avoidance responses to novel stimuli. Since Gray has suggested that NE is one of the neurotransmitters activating the BIS response through the ascending dorsal noradrenergic bundle, NE depletion should result in increased exploration and weakened autonomic arousal, the latter because of the dampening of activity in the descending noradrenergic fibres of the locus coeruleus. Those like Crow and Stein who maintain that dopamine activates search and exploratory behavior and that NE mediates positive reinforcement would argue that catecholamine depletion would reduce exploration in novel environments and bias the organism against novel stimuli which are more likely to be reacted to as aversive.

Mason and Fibiger (1979) destroyed the dorsal NE bundle in rats using 6-OHDA injections and tested them in a novel maze. The NE-depleted rats took longer to leave the start box and to consume food pellets in the novel environment. They also drank less of novel-flavored saccharine, saline, or chocolate milk solutions than controls. Once the depleted rats did make contact with novel stimuli placed in the maze, they took longer to habituate responses to these stimuli.

A different kind of result was reported by Ellison (1977). NE-lesioned rats, although inactive and socially submissive in their home environments, locomoted more and reared less (rearing is assumed to be a behavioral orienting response) than controls in the novel environment. In contrast, serotonin-depleted animals in the open field tended to "freeze," showed hypervigilance (rearing) and stayed near the walls. Ellison assumes that the NE-depleted animals were "depressed" and inattentive to their surroundings rather than "fearless." Serotonin-depleted animals were assumed to be extremely "anxious" in the novel environment. The interaction of NE and serotonin, as revealed by the behavior of doubly depleted animals, was not a simple cancellation of the effects produced by NE and serotonin depletion alone. The doubly depleted animals were the most helpless in the open field, neither rearing nor moving around but merely huddling near the walls.

In contrast to Ellison's results with serotonin-depleted rats, those of Plaźnik, Kostowski, Bidzinski, and Hauptmann (1980) showed that electrolytic lesions of the dorsal and median raphe nucleus produced increased activity in the open field. Geyer, Puerto, Menkes, Segal, and Mandell (1976) reported that rats with selective lesions of median raphe nuclei (but not those with lesions of dorsal or lateral nuclei) were more active than controls during the first 24 minutes in the open field, but differences between groups disappeared by the end of an hour. The results suggested that serotonin depletion affects activity or exploration in novel environments but not in familiar ones. Lorens, Sorensen, and Yunger (1971) electrolytically lesioned the dorsal and centralis superior raphe nuclei and found that lesioned rats showed more spontaneous crossing in a shuttle box than controls before conditioned avoidance training began. Even after shock conditioning reduced the crossings in both groups during intertrial intervals, the lesioned group continued to show significantly more spontaneous crossings. Brody (1970) used p-chlorophenylalanine (pCPA) to deplete serotonin. Post mortem assays showed selective depletion of 5-HT without effects on NE. Rats were habituated to the open field by two exposures prior to the injections of pCPA in the experimental groups. Half of the subjects in both experimental and control groups were exposed to the open field with light or noise stimulation while the other rats were reexposed to the open field without additional stimulation. There were no differences between lesioned and control rats in the first three minutes in the open field without the new stimulation. During both light and noise stimulation the serotonin-depleted rats showed more activity than controls, but the lesioned rats without stimulation showed *less* activity than their controls. The lack of difference between lesioned rats and controls in the initial moments in the open field probably resulted from the fact that both groups had habituated to the initially novel situation. The introduction of new elements of novelty in the form of light and noise increased activity in the lesioned rats. In the absence of such novelty the lesioned rats showed a relatively greater decrease of activity.

The import of these open-field findings, taken together with the inconsistent findings on general activity in familiar environments, is that one of serotonin's functions is to reduce activity in response to novel stimuli or environments rather than activity per se. Soubrie (in preparation) suggests that in conflict situations in which there are opposing reinforcement possibilities and a choice between action and inhibition, serotonin weights the likelihood of inhibition of behavior. The depletion of serotonin may produce an organism that is likely to act impulsively *in spite of anxiety* rather than because anxiety has been reduced. If activity in novel environments is regarded as a measure of exploratory tendencies, and *if* exploratory tendencies (rather than the absence of fearfulness) are regarded as a rat model for sensation seeking in humans, then we might expect high sensation seekers to have low serotonin levels. On the other side, the greater reactivity to novel stimuli in the lesioned rats could represent a greater sensitization to stimulation of a novel and intense nature and the greater activation by such stimuli might represent emotional or fearful rather than exploratory behavior. The findings on the effects of lesioning on the startle response would support the last interpretation.

Electrolytic lesions of median raphe nuclei result in increased amplitude of startle response to auditory (Davis & Sheard 1974) and tactual (air-puff) (Geyer et al. 1976) stimuli. Lesioned and control groups do not differ much in reaction to the first stimulation, but the lesioned group shows an exaggerated reaction on the immediately following trials, or a pattern of sensitization. Eventually all

groups habituate at about the same rate. Similarly, using pCPA lesioning, a transitory increase in the magnitude of the startle response to auditory stimuli is seen (Carlton & Advokat 1973; Conner, Stolk, Barchas & Levine 1970). In these studies there is some evidence that the lesioning does slow habituation relative to the rate in controls. File (1975) found that pCPA *reduces* the initial orienting response to a stimulus. However, there was evidence of greater sensitization to a second novel stimulus after habituation to the first.

Superficially, the results of these startle and OR studies can be compared with results with humans reviewed earlier. The lesioned rats resemble low sensation seekers who show increased startle (or defensive) reflexes and reduced OR responses to tones (Como 1984; Orlebeke & Feij 1979; Ridgeway & Hare 1981), and this would suggest the possibility that high sensation seekers should have higher brain serotonin levels than low sensation seekers. However, it should be remembered that heart-rate acceleration and deceleration were used to define startle and OR in humans whereas skeletal motor responses and interruption of ongoing activity were used for startle and OR definitions in rats. Although these two types of responses might show some correlation if assessed together, they do involve two different autonomic expressions: transient cardiovascular and skeletal-motor responses. Another difference is that the startle (heart-rate acceleration) is almost always greatest on the first trial when differences are found between high and low sensation seekers. In rats the differences in startle (motor response) are usually seen after the first trial which acts to sensitize the animals to the stimulus. Whether or not the models are appropriate to humans is still an open question since we have not as yet been able to find a relationship between the serotonin metabolite 5-HIAA and sensation seeking in humans.

Ellison's results showing a dramatic reversal of behavior of animals in their home colonies and the open-field test suggest that neurotransmitters may have different influences in social-interactive and novel environmental behaviors. Perhaps social behavior in the rat colony provides a better model for human sensation seeking, particularly since gonadal hormones and MAO seem to relate most strongly to traits like sociability, aggressiveness, and sexuality.

Sociability and dominance. At the trait level sensation seeking is positively related to an impulsive type of extraversion characterized by dominance, surgency, changeability, and exhibitionism (Zuckerman 1979b). In groups high sensation seekers speak more and tend to be selected as group leaders (Ozeran 1973). Highs are attracted to dissimilar types of persons whereas lows avoid persons with different attitudes (Williams, Ryckman, Gold & Lenney 1982). Low-MAO humans spend more time in social activities than high-MAO types, and low-MAO monkeys spend more time in agonistic social interactions characterized by dominance and aggressiveness (Redmond et al. 1979). For these reasons one might expect the monoamines to be related to these traits.

Ellison (1977) found that NE-lesioned rats spent more time in their burrows and less time in the behavioral arena where social interactions took place. They also dropped in the dominance hierarchy and tended to end up on the bottom in typical "wrestling matches." They recovered from these effects about 30 days after lesioning, probably indicating regeneration of the NE system. Potentiation of catecholamines, particularly dopamine, initially produced an increase in time spent in the behavior arena, although rats rarely engaged in social behavior, instead spending their time in environmental exploration (Ellison 1979). In later phases of chronic amphetamine stimulation the rats, like human amphetamine addicts, showed a tendency toward seclusiveness. In the final phases aggressive social interactions increased.

Crow, Deakin, File, Longden, and Wendlandt (1978) found that 6-OHDA lesions of the dorsal NE bundle in rats did not affect time spent in social interactions in familiar *or* novel environments, and they use this as one of their arguments against the anxiety hypothesis for locus-coeruleus–NE-system function. The dissimilarity of their findings to those of Ellison (1977) may arise from the fact that their 6-OHDA lesions were more specifically placed than those of Ellison. File and Vellucci (1978) have shown that adrenocorticotrophic hormone (ACTH) reduces social interaction in rats in both familiar and unfamiliar environments. The behavioral effects of ACTH, particularly in the unfamiliar environment, were counteracted by ethanol and chlordiazepoxide, two anxiolytic drugs. Since these anxiolytic drugs also *decreased* the turnover of serotonin (5-HT) in the midbrain, hypothalamus, and cerebral cortex, in contrast to the *increased* 5-HT turnover produced by ACTH, the authors hypothesize that anxiety may result from the action of ACTH on 5-HT pathways in the midbrain and hypothalamus.

Sexual behavior. Consistent findings have been reported of relationships between sensation seeking and variety of heterosexual experience and partners (Zuckerman 1979b), and similar findings have been reported for extraversion (Eysenck 1976). The findings relating the monoamines to sexual behavior have been summarized by Gessa and Tagliamonte (1974; 1975) and Meyerson, Palis, and Sietnieks (1979). Increasing dopamine levels in male rats increases mounting, intromission, and ejaculation and decreases latencies of these reactions; decreasing dopamine levels has opposite effects. However, in female rats dopamine decreases sexual behavior in the form of lordosis (presenting). Serotonin has effects opposite to dopamine in the male, inhibiting sexual activity, and in the female it also decreases sexual activity. NE seems to play little role in male sexual activity. In the female, NE turnover is high and DA turnover low in the median eminence during the proestrous stage when sexual receptivity is present. Electrolytic lesions to the dorsal NE bundle decrease sexual presenting in estrogen treated females, suggesting a causal role of NE in female sexual receptivity. Meyerson et al. (1979) have suggested how gonadal hormones may affect sexual response by sensitizing or inhibiting monoamine pathways in the brain. They claim that estrogen decreases serotonergic and dopaminergic activity in the female and that testosterone decreases serotonergic and increases dopaminergic activity in the male (possibly after conversion to estrogen).

The findings relating the monoamines to sexual activity in the rat are interesting, but, as with other areas of brain–behavior relationships, we must be wary of simple extrapolation to human mechanisms. The catecholamine

releaser amphetamine has varied effects in humans, producing increased sexual activity in some and reduced activity in others (Bell & Trethowan 1961). Whereas estrogen plays a primary role in sexual arousal in the female rat, there is evidence that androgens are the arousal hormone in the human female as well as the male, and that estrogen has little or no role in sexual arousal or arousability. The data are suggestive since, as reported in a previous section, sex hormones are positively related to sensation seeking.

Consummatory behavior (appetite). Sensation seeking has been related to a preference for stimulating (spicy, sour, and crunchy) foods as opposed to less stimulating (bland, sweet, and soft) foods (Kish & Donnenwerth 1972). Gourmets, who also prefer stimulating food, scored higher on sensation seeking than vegetarians (Back & Glasgow 1981).

Mason and Fibiger (1979) found that NE depletion inhibits consummatory behavior in a novel environment and produces reduced consumption of novel-flavored substances. Ellison (1977) reports that the NE-depleted rat shows poor appetite (also a symptom of melancholia in humans). The low-NE rats underconsumed sucrose, even when deprived of food, whereas the serotonin-depleted rats overconsumed sucrose even when not food deprived. Zigmond and Stricker (1977) found that catecholamine-lesioned rats do not increase food intake when given large doses of insulin, and do not increase water intake when made hypotensive. More severely lesioned rats (90% or more destruction of ascending dopaminergic neurons) show aphagia and adipsia and may perish. However, if aroused by stimulant drugs or pain, or tempted by very palatable food (e.g. chocolate milk or cereals), they will eat and drink. The authors state: "In each case, a higher threshold of arousal appears to be required in order to initiate the appropriate [survival] behavior" (p. 418).

Serotonin depletion through lesions of dorsal and median raphe nuclei causes transient increases in food and water intake, suggesting an inhibitory role of serotonin on appetite, but most studies have not shown any effects of destruction of the dorsal NE bundle on food and water intake (Crow 1977). Ellison (1977), however, reports that animals depleted of both NE and serotonin were underresponsive to all positive reinforcers. Rats selectively depleted of serotonin, however, showed closely related increases of consumption of mash and a 50% dextrose solution (Diaz, Ellison & Masuoka 1974).

The data generally support a facilitation of consummatory behavior by catecholamines and an inhibitory effect of serotonin on consummatory behavior. However, there is one paradoxical finding that provides an exception to this conclusion. Amphetamine, a catecholamine releaser, has a general appetite-*suppressant* effect. Hoebel (1979) has suggested that this effect may be specific to the ventral noradrenergic bundle and is not found in the dorsal bundle. Chemical or electrolytic lesions of this ventral bundle produce increased eating and weight gain (Ahlskog & Hoebel 1973), in contrast to lesions of the dorsal bundle, which do not seem to affect consummatory behavior. As with many of the monoamine effects, there may be an optimal-level effect of nonspecific depletion or potentiation of neurotransmitters: either severe depletion *or* too much release of catecholamines may depress appetite. In extrapolating to sensation seeking at the human level we must be wary of a breakdown of the analogy. Sensation seekers tend to be gourmets, not necessarily gourmands; they seek novel and stimulating foods. This could result as much from NE depletion as from a moderately high level of NE since it has been shown that aphagic catecholamine-lesioned rats can only be tempted by more palatable foods not characteristic of their normal diet.

Intracranial self-stimulation. Although electrical self-stimulation of the brain has been a useful method for exploring the locus of reward and punishment in the brain, it is not a natural method, and many of the results are discrepant with those using natural rewards (Gray, Owen, Davis & Eleftheria 1983). However, it does have one reasonably close analogy in human sensation-seeking behavior, namely, the use of stimulant drugs such as amphetamine and cocaine. The rewards produced by these drugs have little to do with the activity of ingesting them, although their illegal and risky nature may be involved in their appeal for sensation seekers (Zuckerman 1983c). Initially these drugs provide a euphoric type of arousal related to their catecholamine releasing or potentiating effects. Stein (1974; 1978; 1983) has in fact argued that catecholamine release is the basis of (intracranial) self-stimulation (ISS). The drugs, such as amphetamine, that increase ISS are those that release catecholamines. Drugs that deplete NE or block NE receptors decrease ISS. Largely on the basis of the ISS studies both Stein and Crow have proposed the following hypothesis:

> Dopamine neurons mediate the incentive or activating response to rewarding environmental stimuli whereas the locus coeruleus noradrenaline system mediates the "reinforcing" or behavior-modifying aspects of such stimuli. Thus dopamine neurons may be expected to be active in appetitive, and the locus coeruleus in consummatory, behaviors. The ventral bundle is envisaged as a satiety mechanism which can terminate a positively rewarded behavioural sequence. (Crow 1977, p. 171).

Stein (1978) also sees a role for the endorphins in the drive-reduction aspects of reward.

The recent theory of sensation seeking (Zuckerman 1979b) hypothesized that these reward systems, mediated by the brain catecholamines, might be the basis for the sensation-seeking disposition, a sensitivity to reward in Gray's terms. However, Gray (1982; Gray et al. 1983), because of his view of the role of the dorsal noradrenergic bundle in anxiety, behavioral inhibition, and sensitivity to signals of punishment, has argued forcefully against the Stein-Crow theory concerning the role of NE in reward. Gray has speculated that the dopaminergic system may serve this role, but his investigations have so far focused on anxiety.

The ISS studies have clearly shown that self-stimulation can be maintained by electrodes placed in various dopaminergic cell sites, including the substantia nigra. Although some studies have found self-stimulation in the locus coeruleus NE site, others have not, and it has been suggested that earlier studies were stimulating other areas in addition to the LC (Wise 1978). It has also been noted that LC ISS produces little behavioral activation

compared to stimulation of the posterior lateral hypothalamus and is harder to obtain and train (Olds & Fobes 1981). Olds and Fobes somewhat cynically state that "current investigation in the field includes those who favor NE mediation [of reward], those who favor DA [dopamine] mediation, and those who favor a critical role for both amines" (p. 554). Perhaps it is safe to say the *catecholamines* support the ISS type of reward at some sites and at some intensities of stimulation.

Inescapable shock has been used as an animal model of depression and "learned helplessness," and this treatment has been shown to reduce catecholamines in the brain (Weiss, Glazer, Pohorecky, Bailey & Schneider 1979). A recent study by Zacharko, Bowers, Kokkinidis, and Anisman (1983) has shown that exposure to uncontrollable shock, in contrast to controllable or no shock, produced marked reductions of ISS response in mice in the medial forebrain bundle and nucleus accumbens, but not in the substantia nigra. The treatment did not affect general activity in the self-stimulation chambers. The authors interpret their results as due to alterations of dopamine activity affecting either the motivation to respond or the reinforcement value of the stimulation.

Summary of findings

Table 1 summarizes the findings showing behavioral and trait correlates of the biological variables discussed in this article. Admittedly, many of the sensation-seeking and behavioral correlates are based on only one or two studies and most of the animal findings are limited to one species. Of necessity, most human correlative studies of bioamines and neuroregulators are based on metabolites and indices measured in cerebrospinal fluid (CSF) or blood, whereas most animal findings are based on experimental operations that directly alter brain levels. Despite these limitations, the total picture may provide the basis for a preliminary comparative model of the role of biological factors in the human trait of sensation seeking. This is attempted in the final section.

New theoretical formulations

If we interpreted CSF-NE as a stable indicator of characteristic (trait) activity of the NE system originating in the locus coeruleus, we would have to say that sensation seekers are persons with characteristically low tonic levels of activity in this system. The possibility exists that high sensation seekers either engage in risky activities and seek high levels of activity and stimulation to compensate for low tonic levels of NE activity and stimulation and low levels of DBH that would slow production rates of NE, or are hypoarousable in this system, needing more stimulation to produce an optimal amount of NE release. This would also explain their attraction to stimulant drugs like amphetamine and cocaine that stimulate catecholamine release or block reuptake.

This explanation is like the one Eysenck postulated for extraversion; extraverts seek stimulation because they are chronically underaroused and consequently need more intense and varied stimulation than aroused introverts to reach an optimal level of arousal. Like Eysenck's theory our earlier theory of sensation seeking (Zuckerman 1969; Zuckerman et al. 1974) attributed these differences in personality type to the sensitivity of the reticulocortical arousal system. In regard to extraversion, considerable evidence for underarousability of sensorymotor neurons has been found, even in the brain stem and lower (Stelmack & Plouffe 1983; Stelmack & Wilson 1982). The findings of the NIMH study (Zuckerman et al. 1983) suggest that levels of calcium, distributed in neurons throughout the body, could be one basis for the hypoexcitability of extraverts as well as hyperexcitability in anxious introverts. However, there has been no evidence of hypoarousal of the cortical neurons of sensation seekers, who in contrast to extraverts actually seem hyperarousable to novel stimuli in orienting reflex studies. The current data indicate the possibility that the specific sensation-seeking behavior may be in reaction to the state of NE and (still possibly) dopamine systems.

It is with great reluctance that I am beginning to reentertain the idea of an optimal-level theory, but one that points to the catecholamine systems of the brain rather than the reticulocortical system. Optimal-level theories are difficult to demonstrate or more substantively refute (Popper 1959), although Eysenck (1981) has suggested ways of doing so. Carrol et al. (1982) rejected the optimal level of arousal theory of sensation seeking on the basis of a drug experiment in which differential effects were predicted for high and low sensation seekers. Although no drug and personality interactions were found, there is no way of knowing what might have happened with higher doses of the stimulant and depressant drugs. Fairly high levels of stimulation intensity are needed to elicit the cortical augmenting–reducing phenomena since the major differences between augmenters and reducers typically appear only at the higher intensities of stimulation. Ethical restraints on human research simply limit the extent to which we can go in assessing optimal-level characteristics in humans.

Psychopathology, mood, and the monoamine systems. The literature relating human psychopathology to levels of metabolites and neuroregulators of the monoamine systems presents a somewhat confusing picture, and this is understandable in view of the complicating factors:

1. CSF, plasma, and urinary measures of the monoamines and their regulators are limited indices of brain neurochemistry; they are influenced by rates of peripheral release, metabolism, and excretion as well as by activity in the brain systems.

2. Activity and mood changes in patients may influence many of the measures, and previous and current drug treatments can change the biochemical systems from the state that existed prior to the illness.

3. Complex interactions between the biochemical systems themselves may obscure the simple relationships between activity of any one neurotransmitter and behavior or mood.

Despite these problems of interpretation, the data from correlational studies, human and animal drug studies, and experimental studies of animals can be put together to form a preliminary model that may be useful as a guide to future studies based on more precise methods, such as neurochemically selective emission tomography. In the

Table 1. *Biological correlates of sensation seeking and their behavioral correlates in humans and animals*

Biological correlates	Human trait and behavioral correlates	Animal correlates	Species
Psychophysiological			
+ orienting reflex	+ attention	+ rearing, interrupting	rodents
	− anxiety	of ongoing behavior	
− startle reflex	+ high arousal in chronic anxiety	− serotonin levels	rats
+ augmenting of evoked potential	+ manic-depressive disorder	+ exploratiyeness	cats
		+ activity	
	+ drug use	+ aggressiveness	
	+ delinquency	+ emotional reactivity to novel stimuli	
Biochemical			
+ testosterone	+ sexual arousability (both sexes)	+ sexual arousal	male rats
	+ sociability	+ aggressiveness	varied
	+ dominance	+ dominance	
	+ activity		
Bioamines & neuroregulators			
− MAO (platelets & plasma)	− manic-depressive disorder		
	− sociability	− sociability	monkeys
	− criminal behavior	− agonistic behaviors (sexual & aggressive)	
		− play behavior	
		− dominance	
− DBH (plasma & serum)	− trait anxiety		
	+ state anxiety		
(serum)	− augmenting of EP		
− NE (CSF)		***NE (brain)***	rats
(+0) MHPG (urinary)	+ bipolar (manic state)	+ activity	
		+ arousability	
	− bipolar (depressive state)	+ activity	
		+ exploratiyeness	
		+ fear (high levels)	rats, monkeys
		+ sexual behavior	rats
		+ appetite	
		(+0) intracranial self-stimulation	
		Brain serotonin	
0 5-HIAA(CSF)	− suicidal disposition	+ behavioral inhibition in novel environments	rats
(serotonin metabolite)	− aggressive behavior	− startle, OR	
	− augmenting of EP	− behavioral reactivity to novel cues	
		− consummatory behaviors	
		− sexual behavior	
	Low-moderate levels of release (brain dopamine)		
0 HVA (CSF)	+ euphoria	+ activity (low-intermediate levels)	rats
(dopamine metabolite)	+ sociability	− activity (high levels)	

Table 1. (*Continued*)

Biological correlates	Human trait and behavioral correlates	Animal correlates	Species
	High or chronic levels of release	+ sexual behavior ♂	
		− sexual behavior ♀	
	+ anxiety	+ intracranial self-stimulation	rats
	− euphoria		
	− sociability		
	− augmenting of EP		
(−0)endorphins (CSF)	− augmenting of EP		

+ = positive relationship − = negative relationship 0 = no significant relationship (+0) = mixed findings (unreplicated positive relationship) (−0) = mixed findings (unreplicated negative relationship) ♂ = males ♀ = females

model proposed no attempt is made to distinguish the effects of the catecholamines dopamine and NE, since this is difficult at the present time. Both are clearly activating systems, but their roles in depression, mania, and other disorders are not yet clearly distinguishable. The general term *catecholamine systems activity* (CSA) is used to summarize the net effect of production, rate of release, metabolism, disposal, and receptor sensitivity on the general level of activity in these systems.

Figure 6 presents the model. Adaptability in general is postulated to be a function of CSA activity, neuroregulators such as MAO, and neurotransmitters such as serotonin and endorphins that generally seem to regulate actions of the catecholamines. There is a tonic level of CSA (at point C) that is adaptively optimal for mood (positive hedonic tone), general activity, and social interaction. Drugs or environmental stimulation may cause transient changes in mood and behavior along the CSA dimension (e.g. from B to C for a normal individual, or between further separated points for abnormal personalities). Figure 7 from Snyder, Taylor, Coyle, and Meyerhoff (1972) shows the effect of amphetamine dosage on the activity of rats. Activity increases with dose up to some point and is then reduced. D-amphetamine, which is a much more potent agent in inhibiting catecholamine uptake, is also more potent than l-amphetamine in stimulating behavioral activity.

Extremely low CSA is associated with primary depression and secondary anxiety, social withdrawal, and inhibition of activity, but far above the optimal level (points E and F) positive feelings (points C and D) can change to dysphoric ones characterized primarily by anxiety, and secondarily by depression. Pervasive endogenous depression (melancholia) would be more characteristic at point A, reactive and neurotic depressions at points E and F. Gray (1982) has postulated a similar relationship of anxiety and depression to overactivity or depletion of the NE system. However, in Gray's model generalized reward expectation would lie on a separate dimension rather than at an intermediate optimal level of catecholamine activity. Furthermore, I believe the clinical and psychometric evidence does not suggest the absence of anxiety in endogenous depression, but a correlated anxiety that is secondary to the depression. Clinically, depression and anxiety seem to go together far more than

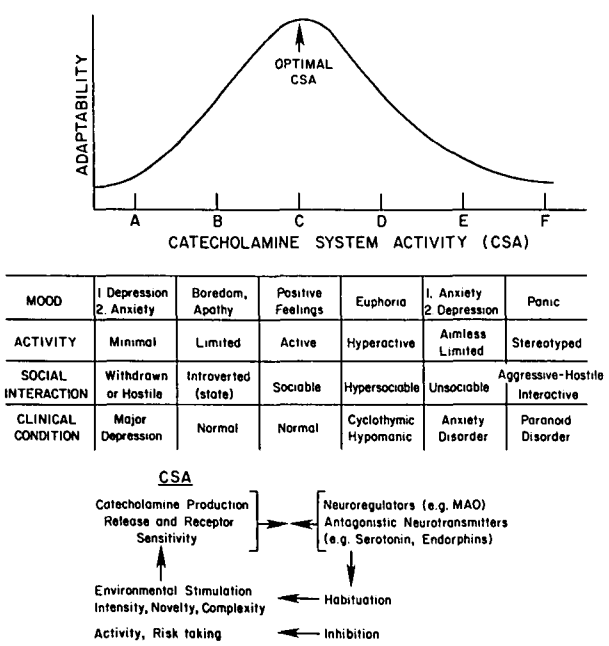

Figure 6. A model for the relationships of mood, activity, social interaction, and clinical conditions to catecholamine system activity (CSA).

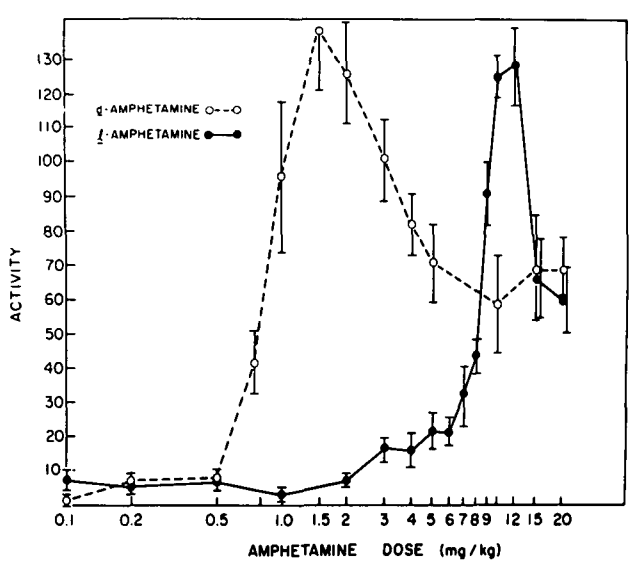

Figure 7. Relationship between locomotor activity of rats and dosages of d- and l-amphetamine (from Snyder, Taylor, Coyle & Meyerhoff 1972).

they diverge. Bipolar affective disorders, because of low levels of one or more of the neuroregulators such as MAO, may move more rapidly between extreme points such as A to D, or D to A, as a function of extreme shifts of CSA.

Discrepancies between the characteristic states of the CSA and the optimal level may dispose persons toward types of stimulation seeking and activities that most affect CSA. If the CSA is at point B, for instance, but the optimal level is at point D, an individual would need to seek intense and novel stimuli, engage in risky and even fear-provoking activities, or take drugs such as amphetamine and cocaine that temporarily increase CSA from point B to points C or D.

The model reconciles the theories suggesting that the NE system mediates alarm or anxiety with those claiming that NE (and dopamine) release are associated with rewarding effects. At optimal levels of CSA the latter is true; at high levels the former effect would be seen. The natural histories of amphetamine and cocaine addiction, and the animal model for the former (Ellison 1979), suggest a movement from C to F, as a function of increasing doses of the stimulant drugs, increasing sensitivity or proliferation of CA system receptors, or both. If Ellison is right in his hypothesis that in the final stages of amphetamine intoxication dopamine is depleted because of damage to the system, then the model presented in Figure 6 should actually be circular; it may be possible to go from point F to point A with chronic overstimulation of the catecholamine systems. The model might also explain the paradox in the results from Ellison's (1977) lesioning studies, in which serotonin-depleted animals were "aroused and exploratory" and active and social in the familiar colony environment but "frightened and paranoid," vigilant, and inactive in the novel environment of the open field. By removing serotonin antagonism of NE and thereby increasing CA levels, he brought them to point D in the model, but the additional CSA produced by the extreme novelty of the open arena temporarily moved the rodents to point E where the noncharacteristic behaviors were observed. The same situation may have moved the NE-lesioned animals from point C (describing their typical behavior in the colony) to D (fearless). Although these kinds of interpretation are post hoc, new methodology that enables us to monitor neurotransmitter activity continuously in free-moving animals could be used to test the deductions from the model.

Further research may reveal that dopamine, NE, and serotonin are differentially related to the behaviors listed. NE may be more relevant to mood and dopamine may be more related to social interaction and general activity, as suggested by the Stein and Crow model. Certainly we must eventually be more specific about which neurotransmitters and what parts of these systems mediate the specific actions.

The relation of sensation seeking to NE or dopamine CSA in the brain cannot be asserted with any confidence, given the limited data on humans and the questions raised about the animal models. The relevance of the data showing low DBH and NE in sensation seekers could mean that there is less conversion of dopamine to NE in NE neurons, allowing for rapid depletion of NE, more dopamine, and a greater dependence on high levels of external stimulation for NE release. The idea of a behavioral trait as compensatory to reduced activity in some neuronal systems works well for extraversion, and somewhat for the clinical trait of psychopathy, but whether it will prove heuristic in understanding sensation seeking remains to be seen.

According to this idea of negative feedback between brain and behavior, the sensation seeker might be at point B of the CSA continuum when in an unstimulated state and would require intense or novel stimulation or risky activities to increase CSA to an optimal level at point C or even D. If the low sensation seeker starts at a higher level of CSA activity, additional stimulation may move him into the anxiety range. For this reason the low sensation seeker might develop an aversion to activities or substances that increase CSA.

The relation between anxiety and CSA will depend on what range of the CSA continuum we examine since anxiety is characteristic at both extremes, being found in a relatively pure state at the high end and mixed with depressive affect at the low end. If we look at normal individuals ranging from A to C in their current states we should find a negative correlation between anxiety and CSA just as has been found between trait anxiety and MHPG in the Ballenger, Jimerson, Lake & Zuckerman (1984) studies of normal subjects (also see Zuckerman et al. 1983). However, if we study patients who might range in state anxiety from D to F, we might expect to find a positive correlation between CSA (CSF-NE) and anxiety, as in the study by Sweeney and Maas (1979), which found increases in urinary MHPG associated with increases in state anxiety in depressed patients stressed by changes in activity demands.

As with the animal models, this human model could be more easily tested if we could track levels of neurotransmitter or metabolite activity in blood during changing conditions of negative stress and positive arousal. The model acknowledges the brain–behavioral feedback, and this creates difficulties in distinguishing state and trait and "cause and effect." Which comes first, the change in CSA or the behavioral changes correlated with it? The experimental work on animals and human clinical and experimental studies suggest that drastic behavioral changes can be initiated by a change in the biochemical status of the organism. Perhaps psychopathological extremes of behavior are a result of deficits of the more stable and genetically determined neuroregulators, such as MAO, that allow neurotransmitter levels to fluctuate widely in response to stress. This kind of model has been proposed by Haier et al. (1980). According to these investigators the combination of low MAO and a strong augmenting pattern of response to stimulation produces an overaroused condition and a behavioral disposition toward sensation seeking. When this is unmodulated by inhibitory capacities of the brain (reducing) there is a high risk for bipolar affective disorder. The model has little to say about the role of the monoamines regulated by MAO or the biochemical basis of the augmenting–reducing tendency.

Pleasure and intrinsic reward. Pleasure (an emotion associated with reward and positive reinforcement) is just as much a biological reality as fear. Its function is to ensure

that adaptive behaviors that ultimately trigger it are repeated. The connections between pleasure and some specific sensory qualities, such as the taste of sweetness, are "wired in"; they do not have to be learned. Whether or not there is an optimal quantitative level of stimulation or arousal that is intrinsically pleasurable is more problematic. Stein (1978) has suggested that there may be two kinds of reward: one related to increasing arousal produced by release of catecholamines in reward areas of the limbic brain, and another produced by the reduction of arousal through release of the endogenous opiates. Many polydrug users (also usually high sensation seekers) seem capable of enjoying both catecholamine releasers like cocaine and amphetamine and opiates that bind to endorphin receptors in the brain. But the opiates and endorphins seem to act in antagonism to the catecholamines, particularly dopamine (Volavka, Davis & Ehrlich 1979). Are these drug users simply trying to maintain an optimal level of arousal that is intrinsically rewarding or are they trying to stimulate both types of brain receptors in order to "have it all"?

The level of arousal produced by these neurotransmitters may not be relevant to the pleasure produced except insofar as it serves situational needs. Stimulant drugs enhance interest in the environment and energize the person for activity. They are also reported to both prolong and enhance the pleasure of sexual orgasm (Bell & Trethowan 1961). Depressants reduce anxiety and inhibition that may interfere with full enjoyment of social or sexual interactions. But persons ingest these drugs and rats self-stimulate certain brain regions when there are no situational needs, and the preoccupation with the chemically or electrically produced sensations may supersede attention to "natural rewards" like food or sex. To paraphrase Edna St. Vincent Millay: the question of *what* they are seeking "is irrelevant biologically speaking." They have found a short-cut for the more circuitous behavioral paths to pleasure.

What is the explanation for the seeking of novel experience which is the heart of the definition of sensation seeking? Aston-Jones and Bloom (1981) have shown that such stimuli do increase activity of cells in the locus coeruleus and therefore release norepinephrine. But is response to such novel stimuli intrinsically rewarding, as Stein's theory would suggest, or fear provoking, as Gray's theory proposes? Humans spend a lot of time and money in the pursuit of novel stimulation and experience, such as travel, art, music, literature, films, and so on, that stimulate the senses, cause mild increases in arousal, but are not instrumentally linked to primary rewards or punishments. Could the brain cells that detect novelty have some kind of link to cells that mediate reinforcement? Such intrinsic reward would reinforce interest in change in the environment which would be of obvious adaptive advantage.

Social influences

Social psychologists and sociologists may rightly ask, Where is the "social" in this "biosocial" theory of sensation seeking? I must confess to a neglect of research on social determinants of the trait. It is difficult to advance on two fronts simultaneously, and I have had much to learn about biology in the last 10 years. Hypotheses concerning environmental influences have been formulated but not extensively tested.

In my earlier theory (Zuckerman 1969) I suggested that the optimal levels of stimulation and arousal might be set or changed by prolonged exposure to levels of environmental stimulation, particularly during early periods of development. Shorter periods of under or overstimulation may result in transient changes of optimal levels as shown in sensory-deprivation research (Zubek 1969). A deduction from this theory is that parents who make a generally stimulating and varied environment for their children are more likely to have high sensation-seeking offspring than parents who provide minimal stimulation in the home. There is ample evidence that monkeys reared in social isolation and invariant sensory conditions do not show the normal preferences for complex stimuli seen in normally reared monkeys and human neonates (Sackett 1972; Suomi & Harlow 1976). Monkeys reared in isolation are also excessively fearful of very novel stimuli.

Among humans higher SSS scores are found in firstborns and only children than in later-born children (Bone, Montgomery & McAllister 1973). This difference might be because parents are more likely to spend more time and devote more attention to only children and firstborns until their attention must be divided. But older siblings are also a source of stimuli, and Suomi and Harlow (1976) have shown that rearing with peers even without parents prevents the detrimental effects of isolation rearing.

Farley's (1973) theory suggests that whereas the basic sensation-seeking tendency is due to constitutionally low levels of arousal, the form that high sensation seeking takes depends on social-learning factors. Thus, the working-class or unemployed delinquent finds stimulation in criminal activities or drugs, partly because of the boredom of nonemployment or the dull, monotonous kinds of labor available to this class, and partly because these kinds of excitement are the only ones modeled by peers. The middle-class sensation seekers may also turn to delinquency, but they also may have access to activities such as travel or sports that may satisfy the need for novel experience.

This theory raises obvious questions about the influence of education and social class on SS subscales or total scores. Zuckerman and Neeb (1980) found remarkably little influence of social class on the SSS scores for men and only some weak influences, particularly on the experience-seeking subscale, for women. Among college males, blacks scored lower than whites on the general thrill and adventure seeking and boredom susceptibility scales, but there were no differences on disinhibition (Kurtz & Zuckerman 1978). These differences might be related to class since the blacks came from a predominantly black state college drawing students largely from lower- or lower-middle-class backgrounds, whereas the white students came mostly from middle- or upper-middle-class families. Although education and social background may play some role in the types of risky thrill and adventure-seeking sports and the desire for travel and alternative life-styles in the experience-seeking scale

items, disinhibition seems to be a culture-free form of sensation seeking rather than a favored expression in lower socioeconomic classes.

The genetic analyses of the SS subscales (Eysenck 1983; Fulker et al. 1980) indicate that genetic factors play a major role in the patterning of the subscale scores as well as the general factor that runs through them. In other words, genetic factors may play a role in the specific forms of sensation seeking as well as in the general disposition. A new sensation-seeking scale, which separates actual experience from desired experience, is now being developed. This form of the SSS may prove more useful in studying the effects of social and familial environment factors on typical experience.

Farley's theory suggests a social-modeling type of learning for the phenomenal expressions of sensation seeking. Kish's (1973) theory is more of a reinforcement model. He proposes that fearful or overprotective parents may discourage or punish sensation-seeking behavior in their children and reward conformity. High sensation-seeking parents may encourage and reward sensation-seeking proclivities in their children. Although both modeling and reinforcement views of parental influences have some plausibility, they pose difficult methodological problems. Since parents who model or reinforce sensation-seeking behavior are likely to be sensation seekers themselves, how do we separate their genetic and environmental influences? Longitudinal studies of adopted children provide the only feasible method for separating the nature and nurture influences, but the SSS has not yet been used in such studies.

It is absurd to deny *some* role for learning exposure in any human behavioral trait. Gray (1973) and Eysenck (1967) have proposed that biological influences determine the susceptibilities to learning effects involving positive and negative reinforcement. The psychopath learns little from punishment and much from tangible reward. Conversely, the introverted neurotic is oversensitized to signals of punishment relative to signals of reward. The high sensation seeker may be characterized by a high expectation of reward in novel experience, whereas the low sensation seeker is sensitized to the possibilities of negative outcomes (Zuckerman 1979b). Risk appraisals and imagined reactions of high and low sensation seekers to new experiences support the postulated cognitive differences (Zuckerman 1979a). Like Gray, I believe that these differences in sensitivity or expectation depend in large part on constitutional differences as well as past outcomes of risk-taking behaviors in life situations.

Cattell (1982), in discussing the high heritability found for the trait of "surgency" (highly correlated with the SSS, particularly disinhibition), tells how these results have changed his theory of the causal influences on the trait. Previously he felt that low surgency was the outcome of punishing, depriving, and inhibiting influences in the family. Now he regards the trait as one of "susceptibility to inhibitory pressures" (p. 337).

These biological views of interaction are opposed to the idea that basic personality traits are shaped entirely by specific environments. I would argue that genes create a type of nervous system that leads us to seek specific kinds of experience. We cannot choose our parents, but what we learn from observing them and others and the outcomes of our experiences with them, depend in some significant part on the way we are constructed.

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A balanced emphasis on environmental influences

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For 15 years, Zuckerman has been making outstanding contributions to our knowledge of sensation seeking, and this paper is no exception. Zuckerman is developing a sophisticated biological model that interweaves multiple causal factors, allows the importance of each factor to be balanced relative to other factors in an empirically defensible manner, and provides a flexible framework open to additions and changes. In his final section, Zuckerman states that the weakest part of his theory is the treatment of social influences. I would like to offer some ideas for developing the weaker side of his model.

In order to construct a truly balanced biosocial theory of sensation seeking, we need to focus considerably more attention on all relevant environmental influences, social and nonsocial. I agree with Zuckerman's suggestion that reinforcement theory and social learning theory can be useful in analyzing environmental influences. Although Zuckerman appears to treat them as separate theories, reinforcement theory and social learning theory are completely compatible (Baldwin & Baldwin 1981a). Social learning theorists fully acknowledge the importance of reinforcement (Bandura 1969; 1971). When people observe real or symbolic models, both the acquisition and performance of behavior are influenced by differential reinforcement. People's choice of models to observe and their likelihood of imitating a modeled behavior are a function of both social and nonsocial reinforcement.

All types of reinforcers and punishers can influence the acquisition and performance of sensation-seeking behavior. Social reinforcers and punishers shape people's choices of models to attend and imitate, causing one person to turn to juvenile deliquency for TAS (thrill and adventure seeking) and another to athletics. Nonsocial reinforcers and punishers have their impact too. After two nearly fatal auto accidents, a 35-year-old auto racer may decide to stop racing and turn to safer sources of sensory stimulation that involve less TAS. Finally, sensory reinforcers are obviously important in shaping sensation-seeking behavior: People tend to learn forms of sensation seeking that bring optimal CSA (catecholamine systems activity) and avoid activities that produce nonoptimal CSA (cf. Ellis 1973). Sensory reinforcers play a very important role in promoting behavioral development all through life: They reinforce the experience-seeking activities that facilitate the development of knowledge, skills, and social adjustment (Baldwin & Baldwin 1981b:179ff.).

To produce a balanced biosocial model, Zuckerman needs to incorporate some insights from sociology and learning theory. For example, estimates of heredity may not be as meaningful as Zuckerman seems to imply. Jencks (1980) demonstrates that estimates of heritability (h^2) "set no upper limit on the explanatory power of environmental variation" (p. 723). High values of

h^2 do not preclude enormous environmental influences on behavior. Even the expression of highly heritable traits – such as PKU (phenylketonuria) – can be influenced significantly by the environment. Children with PKU who grow up in societies with good medical care will suffer few of the deleterious effects experienced by PKU children in societies with inadequate medical care. Environmental conditions almost completely determine the expression of the trait. Likewise, a moderately high estimate of h^2 for sensation seeking does not preclude considerable environmental input in the development and learning of the behavior.

Although Zuckerman analyzes sensation seeking in terms of trait theories, there is reason to be cautious about this strategy, especially given his admitted lack of data on social and learning variables. Mischel's (1968) analysis of trait theories applies to sensation seeking. Trait theories usually presume considerably more behavioral stability and inflexibility than is warranted. Traits are often inferred from psychological tests that measure behavior, not traits; and the structure of questionnaires and tests can generate an artificial sense of structure that is often imputed to the person rather than to the questionnaire.

In actuality, most behavior is much more flexible and malleable than trait theories imply. Learning theory helps clarify both short-term and long-term variations in behavior. Short-term variations tend to reflect changes in context stimuli. Each person has a large repertoire of sensation-seeking behavior, with much more diversity than is suggested by trait theories; and a change in context cues can set the occasion for a person to switch from one type of sensation seeking to a radically different type. Long-term variations in behavior can reflect major changes in context or the learning of different styles of behavior. As people learn new activities that involve different types of sensory experience, they expand their repertoire of behavior and engage in types of sensation seeking not seen at earlier ages.

Zuckerman must be especially cautious in positing a sensation-seeking trait, because (1) he has little data on the extent of environmental influences on sensation-seeking behavior (as he admits in his final section), and (2) a moderately high h^2 does not preclude considerable environmental influences on the behavior. Perhaps at this time it is safer merely to discuss sensation-seeking *behavior*, rather than assuming that the presence of a trait has been adequately demonstrated.

The relative importance of biological and environmental influences on sensation-seeking behavior is an empirical question that requires much future research. As a first step in evaluating the importance of each, we need to build both biological and environmental factors into our biosocial models. A theory that includes all the relevant variables will help alert researchers to control for crucial variables that might not be included in studies based on purely biological or purely social models. This means that biologists need to be sensitive to learning theory, social factors, and other environmental influences in order to control for them in their research; and social psychologists need to attend to biological theories such as Zuckerman's. In addition, we need a balanced biosocial theory to provide a framework for coordinating and integrating the work of researchers in various disciplines. Balanced biosocial theories are within our reach, and they promise to increase the power of the behavioral sciences considerably (Baldwin & Baldwin 1981b).

Personality traits: Causation, correlation, or neo-Bayesian

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Zuckerman's empirical data and theoretical formulations are both convincing and provocative in suggesting a catecholamine arousal hypothesis for sensation seeking. Having been involved for several decades in a similar approach to the study of impulsiveness (Barratt 1959; 1972), I appreciate the amount of effort that is involved in this type of multidisciplinary personality research. I also appreciate the frustrations as evidenced by Zuckerman when he notes that it took him almost 10 years to learn the "biology" relevant to his research on sensation seeking; the result was less emphasis on the study of social variables related to sensation seeking. In addition, there is the very difficult task of interrelating a wide range of data that present the "causal" bases for sensation seeking. It is this last point that I address in this commentary.

Several writers (Barratt, in press; Rorer & Widiger'1983) have emphasized recently that the individual differences model per se has limitations in defining personality concepts. Rorer and Widiger (1983) provide an excellent review of these arguments. Their "thesis is that psychology is burdened with an outmoded philosophy and a distorted view of science" (p. 344). Within this context, they state that they "have tried to use assessment (individual difference) models that are inappropriate to the task, which is more complicated than had been realized. The complications are conceptual, and understanding them requires a significant change in orientation." As they suggest, cause and effect models have their limitations in helping us understand personality theory. Let me briefly relate this suggestion to Zuckerman's theory.

At a general level, Zuckerman presents in his Figure 6 a model that "causally" relates catecholamine metabolism to mood, activity, social interaction, and clinical conditions. In Table 1, he outlines some of the biological *correlates* of sensation seeking or sensation-seeking-"like" behavior in humans and lower animals. Consider just a few questions about the inferred causes and relationships. How do these biological markers relate to other personality traits? For example, let us consider one marker, MAO (monoamine oxidase). MAO is a complex enzyme that has been related to a wide range of neurological and psychiatric disorders including schizophrenia (Murphy, Belmaker & Wyatt 1974). It has a wide range of endogenous inhibitors (Becker, Giambalvo, Fox & Macho 1983) exists in several forms (Denney, Fritz, Patell & Abell 1982), and has extraneural as well as intraneural metabolic functions. What is its unique role in "causing" sensation seeking? Is it that MAO is involved in a wide range of brain activities, and, hence, could potentially be related to predispositions to a broad range of psychopathologies, personality traits, and personality disorders? The same questions have to be asked for every marker in Zuckerman's Table 1. Sensation seeking as a personality trait must interact with other personality traits in predisposing behaviors. For example, what biochemical state exists when an individual reports that he is anxious *and* a sensation seeker? Our own research has indicated, for example, that impulsiveness and anxiety interact to influence behavior on a wide range of information-processing tasks (Barratt 1983; in press; Barratt & Patton 1983). Can Zuckerman's model define a unique profile of markers for sensation seeking? He discusses multiple correlation analyses of a self-report questionnaire that psychometrically define sensation seeking. He then presents the empirical results of laboratory and field studies to arrive at biological markers that are summarized in a neo-Bayesian compilation of predictors (Table 1) to arrive at a causal model of sensation seeking (Figure 1). This is the typical vicious circle of "lifting oneself by one's bootstraps" that has characterized much personality research. What is needed is an additional step – namely, the development of a systems model that allows for meaningful interrelationships of data from different disciplines. It is helpful to obtain data from many disciplines, including using comparative phylogenetic approaches when studying personality traits. The need is for a model that interrelates these data. As I have noted recently (Barratt 1983, p. 387), "The study of the biological bases of

individual differences in personality suffers from the lack of a generally accepted, broad model that interrelates behavioral, cognitive, biological and environmental (including social) concepts." It is especially important when using a comparative approach in studying personality traits to use a model that allows one to compare possible differences in the unique role of biological as well as social and psychological markers at the different phylogenetic levels. Zuckerman has presented a convincing and thought-provoking argument for a catecholamine arousal theory of sensation seeking. The next step is to convince us that the markers or patterns of markers are unique to sensation seeking. This cannot be accomplished without a broader approach to developing a more inclusive personality model. One solution is to use a general systems model. There are others (e.g. Humphreys & Revelle, in press).

Biological correlates of personality: Suppose it's not so simple

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The comparative approach to the study of human traits seems the only way to go. With the many options for rotation available to factor analysts, sources of convergent data are essential. Biology is a prime source of such data. Thus, for personality theorists, biology could be the royal road to respectability.

Sensation seeking is one of the most interesting and potentially biologizable personality traits, and Zuckerman has laid out a clear approach to such a project. One must admire Zuckerman for the studies he has carried out and for the impressive synthesis he has attempted. However, at this stage, criticism is likely to be more productive than applause. Because of this, I have picked out what I see as two weak areas in his presentation. These have to do with: (1) augmenting and reducing and (2) arousal.

1. The case for augmenting and reducing as a single biological variable seems much less strong than Zuckerman implies. For example, if augmenting and reducing of visual ERP (event-related potential) reflected some general sensory processing trait, measures of visual augmenting and reducing should correlate with measures of auditory augmenting and reducing. That does not appear to be the case (Raine, Mitchell & Venables 1983). One might then argue that auditory evoked potentials do not reflect augmenting and reducing, or if they do, that the measures are too noisy to correlate with anything. Again, that is not the case. Auditory augmenting and reducing correlates with homovanillic acid secretion (Bruneau, Roux, Barthelemy, Jouve & Garreau 1983). Thus, one must consider that auditory sensation seeking is a somewhat different trait from visual sensation seeking.

In addition, Buchsbaum (1976) has pointed out how the subject's task (read versus count flashes) and environmental factors (noise versus no noise) can drastically shift the ERP intensity–amplitude slope. Thus, the strategy subjects select probably plays a role in whether they are augmenters or reducers. Thus, augmenting and reducing may be not only modality specific, but determined by voluntary "psychological" as well as more automatic "biological" factors.

2. Although Zuckerman discusses multiple arousal systems and is more aware of neurophysiological complexities than most psychologists, he still tends to oversimplify the notion of arousal. For example, he relates both exploratory and consummatory behavior to only one arousal system. By contrast, this journal recently carried an article by Vanderwolf and Robinson (1981) which delineated a different arousal system for each sort of behavior. Thus, whereas Zuckerman speaks of only aminergic arousal systems, Vanderwolf and Robinson described an aminergic system (probably neither dopaminergic nor noradrenergic) that is related to exploratory, operant behavior and a separate cholinergic arousal system related to automatic, consummatory behavior. These differences in points of view may be important. Potential contradictions may be more useful in the long run than vague supportive data, for conflict can point out where essential complexity may have been overlooked.

The U-shaped functions associated with arousal deserve more attention than they received. Again, Zuckerman does remark on the fact that the same sort of behavior may occur at both high and low levels of catecholaminergic activation. However, he fails to mention how such U-shaped functions can complicate the lives of people who draw inferences from linear correlations. For example, given two U-shaped functions 90 degrees out of phase or even a linear function and a U-shaped function, correlations will be zero if the range is large enough and either negative or positive if the range is restricted, depending on where we have sampled. In studies of traits and cognitive styles, correlations with biological variables of .4 are generally the rule. Zuckerman mentions only one such correlation by number, and that correlation is .42 (albeit with $p < .004$). Such correlations tell you that something is going on but are unlikely to provide more precise information. If you can only account for about 16% of the variance in a measure, it is hard to tell if a manipulation has had any effect.

For sensation seeking, both the psychology and the biology seem more complex than Zuckerman suggests. Although past studies of sensation seeking have provided promising leads, a more molecular approach now seems called for. Separable and limited aspects of sensation seeking need to be selected and studied as biological systems are manipulated over narrow ranges. Tools for finer selection of psychological processes are being honed (e.g. Sanders 1983), and perhaps finer-grained studies of information processing may allow us to define behavioral variables that can be transferred to the animal laboratory and that will be more specific than our current selection. In any case, it would seem a shame to settle for the current oversimplification given such a promising start.

Going over the top with optimal arousal theory

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In this provocative and imaginative paper Zuckerman makes a bold effort to justify the idea that sensation seeking is a fundamental behavioural trait, showing continuity between animals and man and underpinned by distinctive neurophysiological and neurochemical mechanisms. I have to confess, however, that I came away from the paper reeling slightly, still uncertain about the biological status of sensation seeking, and unclear about its exact place in personality theory. There seem to be several problems.

One problem is where sensation seeking fits into Eysenck's (1967) theory or Gray's (1981) modification of it. There is, of course, no obligation that it *should* fit in with either, or that Eysenckian theory *must* be used as a reference point for evaluating any historically more recent biological personality constructs. But since Zuckerman himself chooses to do so it is appropriate to consider his conclusions in that respect. It would be conceptually neat if sensation seeking coincided with the missing dimension (impulsivity?) caused by Gray's leftward 45-degree rotation of Eysenck's E and N factors to produce "anxiety." Zuckerman, however, appears to be arguing that sensation seeking is actually a composite of impulsivity and stable extraversion or sociability (low anxiety in Gray's scheme). To complicate matters, it also seems to be related to "psychot-

icism," a separate dimension in Eysenck's theory but having no status in Gray's. Where, then, does this leave us? Personally, I am left with an overloaded vestibular system after contemplating the consequences of rotating three dimensions, simultaneously with collapsing them into two! Nor does Zuckerman's Figure 6, in which he reduces it all to a *single* dimension, help very much. Whatever one may think of the Eysenckian format, it is intuitively and empirically unlikely that one continuum can contain such varied personality traits and psychopathological states as sociability, reactive and endogenous depression, cyclothymia, anxiety, hostility, panic, and paranoid reactions. Of course, widely differing phenomena *can* be ranged along almost any continuum of choice – here it is "catecholamine systems activity" – but the real question is whether doing so advances our understanding of those phenomena. In this case I believe it does not.

A second problem with Zuckerman's analysis is the ambiguity surrounding the choice of an animal model which, he suggests, might help to establish sensation seeking as a significant biological dimension of personality. Fear and anxiety display a fairly obvious continuity between animals and man, at least up to behaviours as complex (or simple) as those mediated by the septo-hippocampal system. But such is not the case with sensation seeking. Zuckerman offers several possibilities, but they are all, unfortunately, equally persuasive.

A third problem stems from what I believe is Zuckerman's overoptimism about the present explanatory power of neurochemical data. These have an illusory exactitude about them, and I am not sure that one is necessarily likely, to be drawn nearer to the biological basis of sensation seeking – or any other personality trait – merely by choosing the finest-grained measures available. On my reading of the evidence, the neurochemistry of behaviour, and especially of personality, still seems to be in chaos, and I feel that in his search for the biological significance of sensation seeking Zuckerman is starting at the wrong end. It would be better, in my view, to try to come up with a rough and ready conceptual nervous system model of sensation seeking on the basis of primarily *psychophysiological*, rather than neurochemical, measurements. These provide a firmer bridge between brain and behaviour; we do, after all, know more about the neural basis of the GSR (galvanic skin response) as it relates to psychological processes than we do about MAO (monoamine oxidase) or DBH (dopamine-beta-hydroxylase), looked at from the same point of view. Having established a set of psychophysiological descriptors for sensation seeking it might then be easier to interpret the neurochemical correlates of the trait. Zuckerman has, of course, partly adopted this intermediate strategy – his work on augmenting–reducing is a good example – but in his current paper he seems inclined to abandon it in favour of trying to jump in one go the chasm between psychological processes and nervous system chemistry; and, furthermore, to do it simultaneously in animals and man.

Bearing these remarks in mind, what might one expect a human conceptual nervous system model of sensation seeking to look like? In contemplating the question, I am reminded of an attempt I made some years ago (Claridge 1967) to try to encompass, in a single biological model, personality and psychopathological variations associated with both "psychoticism" and "neuroticism." A key concept in the model was the notion of central nervous balance and imbalance, construed as individual variations in the degree to which homeostasis is maintained by reciprocal excitatory and inhibitory mechanisms. I suggested that a relative degree of *imbalance* characterizes the psychotic states and, in the normal personality sphere, defines a dimension rather like Kretschmer's (1925) dimension of "cyclothymia–schizothymia." It occurs to me that there is a strong resemblance between that description of "psychoticism" and Zuckerman's sensation-seeking continuum and that, by the same token, it may be useful to view the latter's biological basis in a similar way; put concretely, that sensation seekers vary not only in arousability but also, simultaneously, in having weakened central nervous inhibitory control, tending overall to veer away from normal CNS homeostasis. It is interesting to note that Haier, Buchsbaum, Murphy, Gottesman, and Coursey (1980), in a paper to which Zuckerman himself refers, recently revived this model when interpreting some of their augmenting-reducing–MAO data. They point out that it is not so much augmenting-reducing or MAO variations alone that significantly differentiates personality types; rather, it is the way the two measures combine to define "balanced" and "unbalanced" styles of central nervous responding. One unbalanced style was the combination of an augmenting mode of responding – indicative perhaps of weakened inhibition – found in association with platelet MAO signs of high CNS arousal. As reported by Haier and his colleagues, that particular psychophysiological configuration looks very much like sensation seeking, a fact that does of course fit Zuckerman's own interpretations, separately, of the MAO and augmenting–reducing correlates of sensation seeking.

It is worth recalling, incidentally, that the notion of excitatory–inhibitory balance as a fundamental nervous system property, related in its own right to individual differences, has a respectable history in the Pavlovian theory from which most current ideas in the field are descended. As a suggestion, it might offer a new perspective on the biological basis of sensation seeking, one that Zuckerman does not seem to have considered but which could perhaps provide a clearer route than his own through the mass of evidence he has accumulated on the trait.

I have confined my remarks here mainly to one particular difficulty with sensation seeking: that of fitting it into other current biological conceptions of human personality and human psychopathology. Zuckerman, I know, shares my concern, and if we differ, it is more a matter of research "style" than any disagreement about the importance of sensation seeking as an observed piece of human behaviour. Lacking his enthusiasm for (and grasp of) neurochemistry and the usefulness of animal modelling, I am less convinced than he that the quickest route to an understanding of sensation seeking is to adopt the twin downward-facing strategies he recommends. Much more important, however, his paper does generate more hypotheses than I can count. If personally I have no taste for testing many of them, one hopes that others will.

The logic of the comparative approach

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If the "comparative approach" proceeded in the way Zuckerman claims, it could not justify any inductive inferences from animal data to the physiological bases of human personality. Since some of those inferences are (it seems to me) justified, Zuckerman's characterization of the approach must be faulty. I would like to focus on Zuckerman's metatheory – his description of the method – since his actual application of the approach rarely seems to make the error that his metatheory sanctions.

The critical issue is this: How do we justify the claim that some animal behavior A provides a model for human behavior H – in this case, sensation seeking? Without some such justification, animal data and animal physiology are not relevant to humans, and yield no evidence for a physiological model of human personality. The justification Zuckerman proposes is far too weak, and I believe he leaves out the critical premise in the comparative approach: namely that it must employ comparative *psychology* as well as comparative biology.

Zuckerman's justification for the use of animal data relies on similarity: similarity of behaviors A and H, and sameness or

similarity of the biological factors correlated with A and H. Why is a shared biological correlate required? The risk of the comparative approach, as Zuckerman points out, is to falsely judge some animal behavior to be similar to human behavior (anthropomorphism) or some human behavior to be similar to that of animals (zoomorphism). The search for shared biological correlates is meant to test such similarities and differentiate merely "metaphorical" comparisons from genuine ones.

My first point is that even if we show animal behavior A and human behavior H to be genuinely similar, such similarity does not justify inferences across species. The reason is that the concept of "similarity" is too weak to carry the load. In philosophical analysis similarity has turned out time and again to be "a pretender, an imposter, a quack" (Goodman 1972, p. 412), and it is a false friend to psychological theory as well.

First, similarity cannot be the basis for judging two organismic events as instances of the same behavior, or for judging that one behavior "models" another. For example, stock-car racing employs behavior more similar to ordinary driving than to skydiving, though stock-car racing and skydiving are both classed as risky sports. A behavior similar to the first would not resemble the second. The wrestling match of the rat may be similar to the "agonistic social interaction" of street brawls but not to the version found in corporate board rooms. Primate grooming behavior is more similar to the commercial transactions of beauty parlors than to the human social behavior it purportedly models, such as affiliative interaction. What these examples show is that similarity (literally construed) is not necessary to class events as the "same" behavior.

Furthermore, similarity is not *sufficient* to group behaviors. The problem is that between any two instances of motor events there is *some* similarity, so similarity cannot be the ground for distinctions. For example, Zuckerman rejects the open-field test as a model for human sensation-seeking behavior. This rejection cannot be based on dissimilarity, since *some* similarity between distinct behaviors can always be found. Both behaviors (for example) show extinction, are sensitive to conditioning, covary with sensitivity to reward, and so on. The upshot is that claims for the similarity of animal and human behavior are not meaningful until we specify which properties those behaviors must share to be counted "similar."

Does adding shared biological correlates do the trick? Zuckerman argues that the demonstration of such biological correlates is sufficient to establish an animal model A for human behavior H. The comparative approach would rely on inferences of the following form:

1. Behavior A of species S is similar to H in humans.
2. A and H are both correlated with a biological factor F present in both species.

Thus: The physiology of A in S provides evidence for the physiology of H in humans.

But is this inductive argument any better than merely showing behavioral similarities? Note first that the biological correlates are not literally shared across species, in that (for example) limbic systems of humans and test species S are not identical. So once again we must judge similarity, this time of biological factors. Second, because the biological correlates that can be measured in humans are peripheral ones, correlates we can show to be shared will be peripheral. As Zuckerman notes, peripheral biological indices bear no stronger relation to the construct (sensation seeking) than does behavior. To demonstrate a correlation with peripheral indices does not show that A and H proceed from like causes, but merely adds a further respect in which A is similar to H. So how does adding (2) strengthen the argument from (1) alone?

Finally, the inductive argument sketched above is simply too weak to support its conclusion. Suppose we judge monkey colony grooming behavior, play, and aggressiveness as similar to human sensation seeking, and correlate both with low platelet MAO (as in Redmond, Murphy & Baulu 1979). Does that correlation provide good grounds for inferring the physiology of human sensation seeking from rhesus physiology? Clearly it does not. The correlation between behaviors and platelet MAO may be mediated by distinct variables in the two species. No common mechanism has been shown to link the trait to the various behaviors and biological factors in the two species. There may be totally distinct mechanisms at work in the two species.

Mere correlation and similarity do not suffice; one must rule out rival explanations of the link between biology and behavior. That is, if we could rule out the possibility that different intervening variables account for the correlations, and show that the same mechanism is at work in the two species, then our inductive argument would be strengthened. This is precisely why the comparative approach needs comparative *psychology*.

Suppose we add a third premise to our induction, as follows:

1. Behavior A in species S is similar to human sensation seeking.
2. A and sensation seeking correlate with the same biological factor F found in S and in humans.
3. Both A and sensation-seeking behavior are produced by the following mechanism, influenced by the following variables, and organized in the following way . . .

Thus: The physiology of A is S provides evidence for the physiology of sensation seeking in humans.

Adding (3) rules out the possibility that the correlation between biological factors and behavior is produced by different mechanisms in the two species. This shows that the common biological factor is not merely coincidence, and so allows inferences to the physiology of sensation seeking. Demonstration of a common cause justifies the claim that the behaviors are similar, for the relevant respect in which A and sensation seeking are "similar" is precisely that they are outputs of the same psychological mechanism. As Gray (1973, p. 412) pointed out, to say (for example) that the wrestling of rats is similar to corporate boardroom infighting is to say something "irreducibly theoretical," to the effect that both behaviors are produced by the same mechanism operated on by the same variables. So to justify the claim that one behavior provides a model for the other, one must show that both species are characterized by the same psychological theory. Fortunately, this is something one *can* do, using behavioral experimentation. Such experimentation can answer for both species the question of which variables control a given behavior and of how those variables are functionally organized.

Without a psychological comparison, the comparative approach cannot justify its inferences about the physiological bases of human personality. In fact, in his application of the approach, Zuckerman makes implicit use of psychological comparisons. For example, when he rejects various putative animal models of sensation seeking, such as emotionality in the open field or rat sociability in novel environments, the reason is simply that the variables determining the animal behavior have a different organization and interact differently from those controlling human sensation seeking. Since this is clearly a good reason to reject those animal models and is not included in Zuckerman's metatheory, I propose that we bring the metatheory in line with practice, and include explicit *psychological* comparisons across species.

Monoamines and human traits: A nice idea, but . . .

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Despite the inclusion of the promising heading "New Theoretical Formulations," Zuckerman's target article presents the old optimal level theory in new clothing. This theory has been

stated and restated in the literature, and some of the historical sources have been cited by the author. In fact, the theory remains as intuitively sound today as when Hebb (1955) postulated it in terms of cue and arousal. Zuckerman states that we are optimally adaptable at the theoretical mid-range of activity of our central catecholaminergic systems. He rates *activity* as a major dimension of catecholaminergic function, but this is clearly another way to say *arousal*, while *adaptability* can easily be defined in terms that Hebb would use to describe "cue" functioning. With regard to another facet of brain catecholamine system activity, *clinical condition* calls to mind the established norepinephrine model of mania and depression. Thus, if we seekers of novel sensation were hoping for some from this article, we'd be compelled to seek on.

There are other problems with the proposed model. Many of these are discussed by the author, though Zuckerman is clearly more optimistic than I about their resolution with advanced technology. I would like to deal with the only aspect of the article with which I feel comfortable, in order to illustrate my personal pessimism.

Redundancy of brain reward mechanisms. The author's treatment of intracranial self-stimulation includes a focus on the involvement of catecholamines, and also a brief allusion to the fact that some sites in the brain do not seem to contain catecholamines, yet support the behavior. I suppose my personal perspective leads me to concentrate on the latter fact. Data from my laboratory over the years show clearly that self-stimulation is obtained from areas in the rat brain that are not famous for their concentrations of catecholamines (e.g. thalamus) (Clavier & Gerfen 1982). Also, whenever the behavior has been obtained from catecholamine-rich areas (e.g. locus coeruleus [Clavier, Fibiger & Phillips 1976], substantia nigra [Clavier & Fibiger 1977], amygdala [Clavier & Wen 1982], prefrontal cortex [Gerfen & Clavier 1981], etc.), the removal of those amines, either electrolytically or neurochemically, has failed to alter the behavior. In the case of the substantia nigra (the only area to be so tested) the depletions of dopamine prior to electrode implantation also had no effect on the acquisition of self-stimulation. So, whatever the involvement of catecholamines in self-stimulation is, it is certainly not critical, as authors like Stein (1974; 1978; 1983) and Crow (1977) have suggested. This being the case, what is the implication for Zuckerman's target article?

First, it can be said that reward in the brain is represented in a redundant fashion by a number of neurochemical systems with an unknown number of interactions. Further, each of the animal correlates and human traits related by Zuckerman to sensation seeking may be similarly redundant in its neural representation, the relation to catecholamines notwithstanding. That neuroscientists have not reached this conclusion to date may be a consequence of the same forces that have led authors to play down evidence that disputes their central theses. Authors feel more comfortable focusing their research on a set of convergent data, which only serves to ensure the continuation of that convergence at a time when the real nature of human brain function seems to be divergent, redundant, and plastic. This, of course, is like putting all one's eggs in the catecholamine basket, the dangers of which are obvious.

In this commentary, I seem to be expressing an almost reflexively cynical and superficial reaction. This in fact belies, my overall reaction to this well-investigated, well-presented effort at eclecticism by the author. The search for comparative correlates of human behavior is a noble one; it serves to bring many of us back to the basics of our involvement in neuroscience. I admire Zuckerman for his recognition of this need, and I thank him for an enjoyable, readable review. My only concern is that he may be constricting his view of the brain when what is needed is an expanding view. I realize that this means we are facing a tougher, more insoluble brain than the simpler one everyone wants to find. But then, who among us would ever describe the brain as simple?

Are sensation-seeking behavior, sleep patterns, and brain plasticity related?

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Zuckerman's review is a bold attempt to analyze sensation-seeking (SS) behavior from man to mice and from biochemistry to sociology. The complexity of the problem reminds us of the words of the late David Krech: "There is no phenomenon, however complex, which when examined carefully will not turn out to be even more complex" (quoted by McGaugh 1976, p. 135). We would like to discuss some additional aspects of SS behavior, and in this manner point to further complexities of the problem.

We agree with Zuckerman that besides being genetically determined, SS behavior is modifiable by experience. The latter point was reliably demonstrated in rodents by exposing animals to different environments. Rats reared in environmental complexity (EC) score higher in a Greek cross test than littermates reared in an impoverished condition (IC) (DeNelsky & Denenberg 1967; Ferchmin, Eterović & Levin 1980). The Greek cross test was originally designed to measure the amount of stimulation seeking, so by this criterion EC rats are higher sensation seekers than their IC littermates. When rats are exposed to EC for one hour daily for one month, their activity in the EC cage increases continually during this time (Eterović & Ferchmin, unpublished results). This observation supports the view that environmental stimulation increases SS behavior. But in addition to stimulating subsequent SS behavior, environmental enrichment produces other behavioral modifications such as increased learning abilities. Furthermore, there are anatomical and biochemical changes in the brain, such as an increase in cortical weight and RNA content, dendritic branching, and larger synapses (Rosenzweig & Bennett 1977). It has been suggested that these changes represent brain adaptation to an increased load of information processing and storage. There are also some indications that this brain adaptability or plasticity is related to the catecholamine systems activity (CSA) in a manner similar to Zuckerman's behavioral adaptability (target article Fig. 6). Let us briefly compare both systems: Low CSA results in an underaroused animal (point A in Fig. 6) whose interaction with the environment is minimal. When underaroused rats are exposed to EC, the increase in cortical weight is minimal. This happens when rats are exposed individually to EC (Rosenzweig & Bennett 1972) or when the environment is not enriched enough (Rosenzweig, Bennett, Hebert & Morimoto 1978).

At the optimal level of CSA (point C in Fig. 6), the animal will interact actively with the inanimate and social environment. This is observed with rats exposed to EC in large groups or individually under the effects of a small dose of methamphetamine. The increase in cortical weight is much larger under these conditions (Bennett, Rosenzweig & Wu, 1973).

Finally, overaroused or stressed animals (points E and F in Fig. 6) will become unsociable and less active, sometimes to the point of "freezing" behavior. We have repeatedly observed that in short-term exposure to EC, cortical weight does not increase when the animals are stressed (Eterović & Ferchmin 1974).

In a different system, studying eye dominance and binocularity, Pettigrew and his collaborators have shown that increasing norepinephrine concentration in the brain by microperfusion causes an increase in brain plasticity (Kasamatsu & Pettigrew 1979).

An additional indication of CSA involvement in brain plasticity comes from studies on rapid eye movement (REM) sleep. This type of sleep, which depends on norepinephrine pathways (Leconte & Hennevin 1981), is increased in EC rats, although there are some discrepancies among authors concerning the exact nature of these changes (Gutwein & Fishbein 1980;

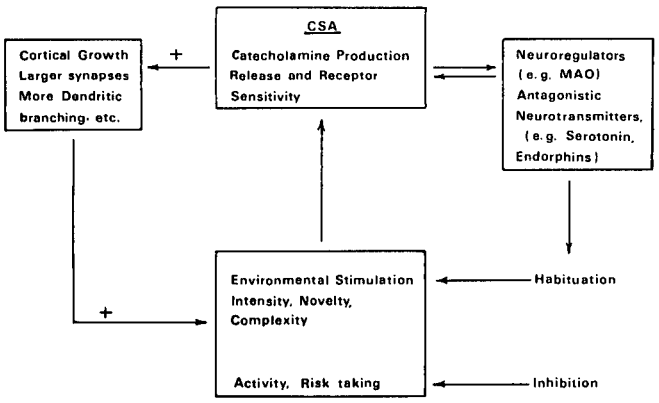

Figure 1 (Eterović and Ferchmin). Elaboration of Zuckerman's Figure 6.

Kiyono, Seo & Shibagaki 1981; Mirmiran, Van den Dungen & Uylings 1982; Pearlman 1983). On the other hand, suppressing REM sleep by clonidine injections decreases the activity of female rats in EC cages (Mirmiran & Uylings 1983). When REM sleep is suppressed pharmacologically in early life, the brain permanently loses its capacity to grow in response to environmental stimulation (Mirmiran, Uylings & Corner 1983; Mirmiran, Scholtens, Van de Poll, Uylings, van der Gugten & Boer 1983). These experiments suggest that activity, cortical growth, and REM are closely interrelated.

There are also some indications that genetically determined patterns of REM sleep are related to genetically determined levels of SS behavior. Mice of C57BL/6 strain respond to a modification of the environment with an increase in REM sleep, whereas BALB/c mice show a decrease under the same circumstances (Sinton & Jouvet 1983). The genetically related strains C57BL/6J and BALB/cJ display, respectively, high and low exploratory activity (target article Fig. 5 and Bovet 1977). Thus it seems that both genetically predetermined and experientially induced high sensation seekers spend more time in REM sleep than low sensation seekers. Conversely, REM sleep seems a prerequisite for high activity. Thus REM sleep might fulfill the requirements for a biological correlate of SS behavior. Has any study been done in humans correlating ranking in SS with REM sleep?

In summary, we propose that the model presented by Zuckerman in Fig. 6 could be expanded in the following way: Define "adaptability" not only as the ability to respond properly to the inanimate and social environment, but also to include the concomitant brain plasticity. The scheme in the lower part of their Fig. 6 would then look like our Figure 1.

Increased CSA induced by environmental stimulation triggers trophic factors which result in a physically modified cortex. It is possible that this modified brain is responsible for increased learning abilities and increased interaction with the environment (increased SS).

The comparative approach in personality study

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Zuckerman's approach to the study of personality is so similar to my own that I have little but praise for his determined and long-continued effort to treat man as a biosocial animal and to emphasise the genetic as well as the environmental contribution, the physiological as well as the psychological determination of behaviour, and in general for the reductionist attempts to discover relationships between behaviour and hormonal, biochemical, and physiological secretions and activities. In particular, I am at one with him in valuing highly the possibility of finding animal analogues for human behaviour and personality traits, a field that has been very much neglected by experimental psychologists but which holds out a great deal of promise.

Consonant with what I have said so far, it will be obvious that my contribution to this discussion will be very limited; agreeing as I do with Zuckerman's approach, and the many detailed findings that his theories have yielded in the past, there is comparatively little I have to criticise or add. Indeed, what I have to contribute is not so much a criticism as an elaboration and possibly a somewhat different interpretation of the facts. My first comment relates to the following statement by Zuckerman:

> Sensation seeking seems to constitute a dimension lying between two of Eysenck's primary dimensions, E and P (perhaps closer to P) rather than representing a component trait of either. The question of which are the primary orthogonal dimensions of personality cannot be answered by factor analyses of tests alone but must involve comparative alignments of test-defined dimensions with biological dimensions of personality.

I have two comments. The first is that sensation seeking does not really constitute a dimension; the correlations between the four components of sensation seeking are so low (sharing on the average only about 10% of the variance) that they do not define a single dimension. I would prefer to regard the items in Zuckerman's sensation-seeking scale as points in a three-dimensional space, defined by the three major dimensions of P, E, and N. Most of these points lie in the plane defined by P and E, and may be roughly grouped in the four sectors identified by Zuckerman. In part, these points lie outside the three-dimensional space defined by P, E, and N, and it might be particularly interesting to direct research at the percentage of the variance for each item, or subscale, lying outside this three-dimensional space (Eysenck 1983).

The second part of the quote from Zuckerman raises a problem that I think is essentially indeterminate. By raising the question of which *are* the primary orthogonal dimensions of personality, Zuckerman seems to imply that in some way such "true" dimensions exist in reality, but I would suggest that this constitutes an impermissible reification of factors. In all taxonomy there are diverse ways of dividing up the field, and each of these represents subjective judgments and values; to think of factors and traits as otherwise is to misinterpret their proper scientific status.

Later in his target article Zuckerman questions the hypothesis that sensation seeking is based on individual differences in arousal and points out that "although high sensation seekers use a variety of drugs, such as amphetamine and cocaine, that raise arousal levels . . . the fact remains that they also use drugs, such as opiates and barbiturates, which lower arousal levels." This is true, but it does not necessarily speak against the theory. Arousal is not only produced by stimulant drugs; it may also be produced by any *change* in either the stimuli that act upon the person, or his mood states and other internal responses. Thus, paradoxically, a depressant drug may have an arousing effect because it represents a change in the mood or level of the person, just as conditions that might be thought to depress the arousal level, such as boredom-inducing conditions, may in fact produce arousal (Gale 1981).

The last point relates to Zuckerman's discussion of the open-field tests. As he points out, behaviour in the open field depends on two dispositional traits: exploration and fear, the former usually measured by an index of ambulation, the latter by some index of defecation. Zuckerman points out that these two measures tend to be negatively correlated, but this is very much dependent on the actual stimulus conditions. In the standard open-field situation the animal is bombarded with loud white noise as well as strong illumination; hence a great deal of fear is produced, and this interferes with exploratory tendencies. At the Barcelona Free University, a determined attempt has been made to measure extraversion through exploration under conditions in which the fear produced by the open field is not

excessive. Eliminating the noise, they have shown that under these conditions there is *no* correlation between exploratory behaviour and defecation and that under these conditions it is possible to verify a large number of predictions from the arousal theory of extraversion–intraversion by selecting and testing animals showing much exploratory behaviour (extraversion) or little (introversion). Much of this work has been summarised by H. J. Eysenck and M. W. Eysenck (1984) and by Garcia-Sevilla (1984); it seems doubtful to me whether these predictions could have been made on the basis of sensation-seeking theory. This is clearly an empirical problem, however, and insofar as Zuckerman's views and mine constitute alternative theories, it should be easy to set up experimental conditions that would present us with crucial evidence to decide between them. Both theories are scientific in the sense that they are falsifiable, and this indeed is one of the greatest virtues I see in Zuckerman's whole approach.

Is there a relationship between sensation seeking and strength of the nervous system?

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Zuckerman's general approach to the problem of analysing the biological basis of personality, as exemplified by his Figure 1, with its nice balance between human and animal research, seems to me to be just right. His own work on sensation seeking is an excellent and important example of this approach. The complexity of the problems he addresses raises many issues of detail which will require continuing empirical and theoretical analysis for many years to come. It is the purpose of this commentary to raise simply one of these issues.

Figure 3 of the target article demonstrates an impressive relationship between sensation seeking (more accurately, disinhibition) and augmenting–reducing: high disinhibitors show a greater increase in the amplitude of visual evoked potentials with increasing stimulus intensity than do low disinhibitors. Inspecting Zuckerman's figure, I was struck by its great resemblance to three figures (60, 61, and 63) that are to be found in Nebylitsyn's (1972) book *Fundamental Properties of the Human Nervous System.* These figures show the relationship between strength of the nervous system (Gray 1964; Nebylitsyn 1972) and the amplitude of the photic driving reaction, measured electroencephalographically, in response to repetitive visual stimulation. Individuals with a strong nervous system show a much more pronounced increase in the amplitude of the driving reaction with increasing stimulus intensity than do individuals with a weak nervous system, especially in the theta and beta bands. Given the relatively small differences between the two methods employed (consisting chiefly in the repetition rate of the applied visual stimulation and the use of either EEG or evoked potential indices of the response), these two sets of results raise an obvious question: Is there a relationship between sensation seeking (or perhaps, more specifically, disinhibition) and strength of the nervous system, with high disinhibitors equivalent to individuals with a strong nervous system? A relationship of this kind seems particularly probable, given previous evidence that (1) extraverts are likely to behave like "strong" individuals on tests of strength of the nervous system (Gray 1967; 1981), and (2) sensation seeking constitutes "a dimension lying between two of Eysenck's primary dimensions, E and P" (target article).

The question raised above is easily open to experimental attack, though a considerable research effort would be required to answer it. If the answer were positive, we would have a valuable link between research on personality in the West and the East.

Sensation seeking and augmenting–reducing: Does a nerve have nerve?

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Few researchers in the field of personality have attempted so bold and comprehensive an approach as that proposed by Zuckerman. To most personality researchers, the synapse is far distant from a trait or state (or even a behavior), and, in principle, any discussion of synaptic events is often reduced to reductionism. A review of recent issues of the *Journal of Personality and Social Psychology*, for example, reveals few papers that address or indirectly mention connections between the brain and personality. This apparent bias is embarrassing because advances in the neurosciences are provocative and directly related to many of the original problems of interest to students of personality and individual differences. In fact, as partially reviewed by Zuckerman, the evidence for genetic, neurotransmitter, and psychophysiological influences on personality is so compelling that one wonders whether the field of personality can contnue to exist as a viable intellectual and empirical enterprise without reference to neuroscience progress.

The comparative approach suggested by Zuckerman and illustrated so convincingly with the literature on sensation seeking offers an array of creative, testable hypotheses. I comment here only on the relationship between augmenting–reducing measures of sensory modulation and sensation seeking.

The general premise Zuckerman advances is that "genes create a type of nervous system that leads us to seek specific kinds of experience." Augmenting–reducing is a construct that was intended to differentiate basic types of nervous systems. Zuckerman reviews the evidence that augmenting (defined by increasing evoked potential amplitudes *at the vertex* to increasing light stimulus intensity) is related to greater sensation seeking, especially more disinhibition. Such a relationship, however, is opposite to that originally predicted by Petrie (1978) who defined augmenting (on the basis of kinesthetic procedures) as a relative sensitivity of the nervous system to sensation. She argued that people with an augmenting type of nervous system should be sensitive to stimulation and thus should avoid strong sensation. Such individuals would be expected to exhibit less sensation-seeking behavior. On the other hand, reducers should be relatively insensitive to stimulation and more sensation seeking would be apparent.

Elsewhere, I have criticized in some detail the available studies suggesting that augmenting is related to high sensation seeking (Haier, Robinson, Braden & Williams 1984). Our conclusion was that these studies were empirically weak or used unusual evoked potential procedures for measuring augmenting–reducing. The most important procedural detail in these studies appears to be the use of light stimuli of extremely high intensity. Such a procedure may confuse the issue dramatically since at such high stimulus intensities, augmenters may become reducers and vice versa (Robinson, Haier, Braden & Krengel, submitted). Thus, at least some of the data discussed by Zuckerman may be consistent with a relationship between reducing and sensation seeking, as predicted by Petrie. Without reference to this procedural detail, Davis, Cowles, and Kohn (1983) have argued that the augmenting–reducing terms are used in reverse by Petrie and by Zuckerman and Buchsbaum. Moreover, Goldman, Kohn, and Hunt (1983) reported a positive correlation between reducing (measured by a paper and pencil test) and sensation seeking.

Our own study of extreme augmenters and extreme reducers identified from an evoked potential screening (using the standard Buchsbaum procedure) of 120 normal volunteers showed relatively strong differences in sensation seeking (Haier et al., in press). Congruent with Petrie's formulation, reducers had high-

er scores (2-tailed *t*-tests) than augmenters on the general sensation-seeking scale and the experience-seeking scale ($p < .001$); the thrill and adventure-seeking scale ($p < .02$), and the boredom-susceptibility scale ($p < .05$). Only the disinhibition scale showed no difference.

We also screened a second sample of normal volunteers (N = 54) on the sensation-seeking scale and compared high and low scorers (on each subscale) on augmenting–reducing. This was done using a relatively new technique of topographic mapping which is based on recording augmenting–reducing at 15 sites simultaneously on the left side of the scalp. A preliminary comparison between the highest ($\bar{x} = 57.5$; $sd = 8.0$) and the lowest ($\bar{x} = 32.6$; $sd = 6.8$) scorers on the general sensation-seeking scale is shown in Figure 1. Although the distribution of augmenting–reducing for high ($N = 15$) and low ($N = 10$) scorers is not statistically different (note the relatively small N) the patterns clearly suggest more augmenting at the *vertex* in the low group; the high sensation-seeking group shows more augmenting in the frontal lobe. Very similar patterns are apparent for each of the sensation-seeking subscales. Figure 2 shows the augmenting–reducing cortex distribution for high (≥ 54; $N = 7$) and low (≤ 32; $N = 6$) scorers on the boredom-susceptibility subscale.

The fact that strong relationships exist between various measures of augmenting–reducing and sensation seeking, despite some disagreement on interpretation of direction, supports Zuckerman's central premise. An important goal for future research is to establish the mechanisms of augmenting–reducing, especially as related to neurotransmitter systems. Platelet MAO activity (not level) in combination with augmenting–reducing, for example, may be related to vulnerability to psychopathology, and to personality types (see Buchsbaum & Haier 1983; Haier, Buchsbaum, Murphy, Gottesman & Coursey 1980).

As we begin to establish relationships between complex synaptic events and complex behavior, in animals and in humans, personality researchers have an extraordinary opportunity to integrate findings from historically separate (if not isolated) fields. The current paradigm in personality may well shift to include new concepts of etiology, in addition to the traditional focus on assessment, structure, and dynamics.

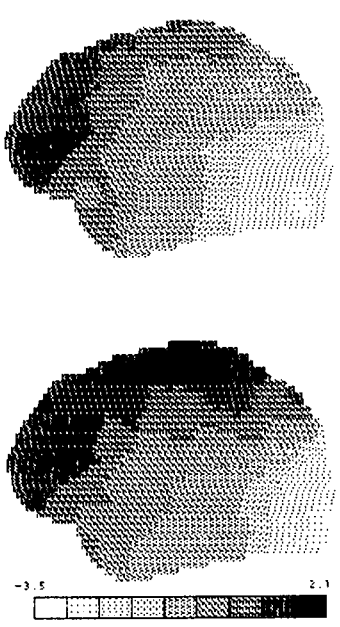

Figure 1 (Haier). Distribution of augmenting–reducing on left cortex (frontal lobes on left) of high scorers on the general sensation-seeking scale (top) and low scorers (bottom). Darker shades represent more augmenting.

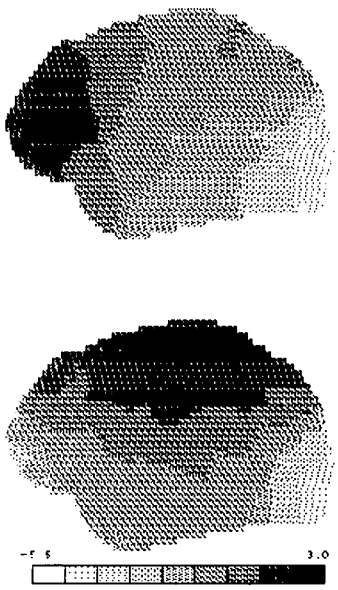

Figure 2 (Haier). Distribution of augmenting–reducing on left cortex (frontal lobes on left) of high scorers on the boredom-susceptibility subscale (top) and of low scorers (bottom). Darker shades represent more augmenting.

Emotion variables as personality traits

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I applaud Zuckerman's extensive work on sensation seeking (SS), first at the experiential level through self-report (SSS), then at the behavioral level through observational measures, and more recently at the biochemical level through assays of neuroregulators and neurotransmitters. The stable subscale alpha coefficients, high test–retest reliabilities, and substantial validity indices for the SSS bespeak Zuckerman's clear conception of the cognitive-experiential domain of sensation seeking and his remarkable knack for item writing and scale development. His successful efforts in examining relationships between SS measures and arousal, evoked potentials, gonadal hormones, and monoamines are testimony to a good balance of sensation seeking and persistence in his own personality.

Zuckerman and his students have amassed sufficient evidence to make a good case for the status of sensation seeking as a trait that has significant biological determinants and important influences on personality and behavior. Assuming this status for SS, it may be useful to search for the components of this trait that account for its biological roots and behavioral significance. At the phenomenological or experiential level, SS apparently contains both cognitive and affective elements. Cognitive processes are obviously involved in the measurement of SS, for the SSS is a verbal self-report measure. Yet, the verbal content of the scale clearly depends on the vocabulary of emotions, both positive and negative. It is my contention that it is the emotional aspects of SS that account for its biological roots. The wording of SSS items suggests that the most important positive emotion in SS is interest-excitement (in risk taking and nonconforming behavior) and the most important negative emotion is fear (or its modulation in the face of risks and norm violations). Evidence for the heritability and traitlike nature of emotion variables is mounting (Buss 1982; Buss & Plomin 1975; Hinde, Easton, Meller & Tamplin 1982). Goldsmith and Campos (1982) have reviewed evidence relating emotions to temperament and biological determinants. They maintain that in infancy all dimensions of temperament are emotional in nature and are stable characteristics of the individual.

Several recent studies in the Delaware Human Emotions Lab have shown that emotional expression styles indexed in early infancy show continuity over time and relationships to neural activity and behavior. Facial indicators of interest in 2-, 4-, 6-, and 8-month-old infants were associated with heart-rate deceleration and predicted attention focusing, as indexed by visual fixation (Langsdorf, Izard, Hembree & Rayias 1983). The ratio of anger to sadness expressions in 13-month-old infants during brief separation from their mothers was significantly related to a temperament (mother-interview) measure of distress to limitations, designed as a measure of day-to-day anger-related behavior (McGinnes & Izard 1983). McGinnis and Izard also found that mothers' self-reported emotional experiences and emotion-expression styles in daily life were significantly related to their infants' emotional expression patterns during brief separation. Hyson and Izard (1983) found substantial continuity of emotional expression patterns during brief separation over the first half of the second year of life. Indices of expressions of interest and anger during brief separation at 13 months were correlated .90 and .61 respectively, with the 18-month measures of interest and anger expressions during brief separation. Emotional expression blends and total negative emotional expression during separation also showed significant correlations over this period, .53 and .90 respectively.

The foregoing findings support the hypothesis that early emotion measures index stable traits that influence individual behavior and social interactions. Earlier research (Ekman, Friesen & Ellsworth 1972; Izard 1971) provided robust evidence of the innateness of emotions, evidence suggesting that emotion variables have important biological determinants. These lines of research do not point simply to emotionality. Rather, they show that each of several discrete fundamental emotions can be operationally defined and that each can account for unique variance in predicting temperament or personality traits. That SS involves more than one discrete emotion has "face validity" in terms of the content of the SSS. Therefore, the rapidly growing body of data on emotion (see Izard, Kagan & Zajonc 1984) suggests that more could be learned about the biological and behavioral characteristics of SS by giving careful attention to its discrete emotion components.

Sensation seeking: A clarification, a caveat, and a conjecture

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Zuckerman, in a revision of his earlier aminergic hypothesis of sensation seeking (e.g. Zuckerman 1979b), now argues that low norepinephrine (and possibly other amines) may predispose to greater sensation seeking, providing thereby an optimal level of sensory-affective stimulation. Since he has already noted the problems inherent in testing a bitonic hypothesis (as is now proposed), I will not comment further on this. I do believe, however, that a clarification, a caveat, and a conjecture are in order.

First, the clarification: Zuckerman's new hypothesis is deductively powerful, and leads to a large number of interesting additional tests and hypotheses. Fairness dictates that it be noted at the outset that many hypotheses and findings are not unique to the theory, that at least certain of the latter have already been published, and that they have not been so noted in the target article. For example, an essentially similar proposal of a bitonic relationship of noradrenergic activity and reinforcement was made in 1982 by Leiblich and his coworkers (Leiblich 1982). Moreover, the locus coeruleus was postulated to be a novelty-reinforcement system three years ago by Zajonc and his coworkers (Zajonc 1980). These findings are not mentioned to detract from the originality of the sensation-seeking studies, but merely to give appropriate intellectual credit where due.

I feel that one caveat is in order as well. This concerns the use of monoamine oxidase (MAO) as an experimental variable. For many of Zuckerman's comparative analyses a relationship of MAO is necessary. For example, MAO appears to be low in high sensation seekers, and vice versa. As noted in the target article, both adrenergic and tryptaminergic compounds are metabolized by the same enzyme. Their functions may be antagonistic, however (e.g. Brodie & Shore 1957). This may make specific attribution of neurochemical control due to changed MAO or of altered control with altered levels of MAO impossible in principle. Changes in MAO may effect parallel changes in normally balanced and mutually antagonistic systems. A converging neurochemical strategy is necessary. The data on NE, if confirmed, would offer just such a means of better and more specific chemical attribution.

Finally, I would like to offer a conjecture, based upon some studies of my own on brain stimulation as a model of sensation seeking in animals. I and my coworkers have found that rats seek both rewarding stimulation and novel preferred tastes (such as saccharin) with some degree of statistical regularity (Katz 1979; 1980; Katz & Watson 1983). If sensation seeking is a valid trait at a comparative level then on the basis of consumption patterns in both rats and hamsters I conjecture that the following relationships will obtain in further cross-species analyses: First, sensation-seeking episodes will be sought randomly across time, and a statistical analysis of the relationships of episodes and interepisode intervals will indicate neither a drive for, nor satiety following episodes of sensation seeking. We have found that times between episodes of feeding and stimulation may be predicted from a Poisson distribution, that times prior to an episode (when drive might cumulate) bear no relationship to episode duration, and that times after an episode when satiety might be expected to dissipate show a correlation with episode duration and intensity reflecting a process just inverse to satiety.

In addition, sensation-seeking episodes should become more probable as they continue; episodes of sensation seeking should be characterized by robust warm-up effects. Finally, episodes of sensation seeking should be more likely immediately following previous episodes, and increasingly less likely with the progression of time. All the above findings may appear counterintuitive, but all have been found repeatedly in our analyses of two prototypic behaviors in Zuckerman's comparative analysis.

Assuming that exploratory behaviors, sexual activity, drug seeking, or related sensation-seeking behaviors in humans obey the above constraints, this would imply a high degree of formal similarity and motivational continuity in the sensation-seeking trait across species. My hunch is that these relationships do exist in a species-continuous fashion, provided that an appropriate degree of methodological continuity underlies the analysis.

ACKNOWLEDGMENT

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The biochemical basis of sensation-seeking behavior

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Zuckerman has presented a valuable review of sensation seeking and its biological correlates. From the literature, there seems to be enough evidence to conclude that sensation seeking is a stable personality trait, to a great extent under genetic control. There are also enough data to conclude that sensation seeking is

Table 1 (von Knorring). *Changes in amplitude-intensity (A−I) slope in the visual evoked potential and in 5-HIAA, HVA, and MHPG in CSF during treatment with zimelidine or placebo*

	Zimelidine treatment	Placebo treatment	
Baseline A-I slope	+4.65±4.30	+0.11±3.47	
Change	−6.31	+0.19	$F = 29.70$; $p < 0.01$
Baseline 5-HIAA	107.1±35.1	113.5±28.9	
Change	−28.6	+1.7	$F = 5.54$; $p < 0.05$
Baseline HVA	201.2±27.4	250.6±31.5	
Change	+3.7	+40.8	$F = 0.25$; n.s.
Baseline MHPG	36.1±3.2	41.6±2.8	
Change	−5.0	+8.0	$F = 1.41$; n.s.

Note: Double blind, four-week trial. Chronic pain patients (A-I slope, $N = 32$, CSF data, $N = 20$); analysis of covariance with initial values as covariates.
Source: Johansson 1982; Johansson et al. 1980; von Knorring & Johansson 1980.

a separate personality trait, correlated with, but not identical to similar traits, such as extraversion, sociability, impulsivity, and monotony avoidance. Thus, the biological correlates of this personality trait are of considerable interest.

In the target article, Zuckerman moves away from the optimal level of arousal (OLA) theory. There seems to be enough experimental evidence to conclude that the OLA theory is at least an oversimplification.

Instead, Zuckerman relates sensation seeking to the monoamine systems in the brain. There is no doubt considerable evidence relating sensation seeking to the monoamines. However, when one tries to relate the sensation-seeking trait to one single monoamine system, difficulties occur, and several separate hypotheses might be suggested.

There are two biological correlates of sensation seeking that seem to be especially well documented. High sensation seekers appear to have an augmenting response in visual averaged evoked potentials (VEP) and low activity of the enzyme monoamine oxidase (MAO) in platelets. Thus, the central biochemical correlates of these indicators seem to be important.

An augmenting response in the visual evoked potential has been related to low concentrations of the serotonin metabolite 5-hydroxyindole-acetic acid (5-HIAA) in CSF (cerebrospinal fluid) and low concentrations of the dopamine metabolite, homovanillic acid (HVA); no relationship has been established, however, with the noradrenaline metabolite, 4-hydroxy-3-methoxyphenylethylene glycol (MHPG) in CSF (von Knorring & Perris 1981). Thus, the augmenting–reducing response could be related to the serotonin or the dopamine system. Zimelidine, however, a rather specific serotonin reuptake inhibitor, has been shown capable of changing the augmenting–reducing response in the direction of more reduction both in chronic pain patients (von Knorring & Johansson 1980) and in healthy volunteers (von Knorring 1982). Parallel to these changes in the augmenter–reducer response, 5-HIAA in CSF is reduced, while no changes are seen in HVA or MHPG (Johansson 1982; Johansson, von Knorring, Sedrall & Terenius 1980), indicating that changes have been provoked only in the serotoninergic system and yet changes do occur in the augmenting–reducing response.

The experimental results thus favor a relationship between the augmenting–reducing response and the serotonin system.

Platelet MAO levels are a stable characteristic of the individual, and the activity seems to be under genetic control. It has been suggested that platelet MAO activity reflects the size of the central serotoninergic system (Oreland 1980). In healthy volunteers, there is a significant positive correlation between platelet MAO activity and the serotonin metabolite, 5-HIAA in CSF (Oreland, Wiberg, Åsberg, Träskman, Sjöstrand, Thorén, Bertilsson & Tybring 1981). In platelets, only MAO-B exists, whereas the MAO-A form of the enzyme is the most important one in serotonin metabolism in the brain. In animal experiments, however, there is some evidence indicating that mouse strains with low MAO-A activity also have low MAO-B activity, low concentrations of 5-hydroxytryptamine (5-HT), and low concentrations of 5-HIAA in their brains, whereas no relationship is found with dopamine (DA) or its metabolite HVA (Oreland & Fowler 1982).

There is also a striking similarity between the clinical groups in which low platelet MAO activity and low concentrations of 5-HIAA have been found (Oreland & Fowler 1982).

Thus, there is evidence indicating that both the augmenting–reducing response and platelet MAO activity are related to the serotoninergic system in the CNS while there are also data contradicting a direct relationship between the augmenting–reducing response, platelet MAO activity, and the catecholamines, dopamine and noradrenaline in CNS. As both the augmenting–reducing response and platelet MAO are related

Table 2 (von Knorring). *Concentrations of MAO, DA, and HVA in mouse brains*

Strain	MAO-A	MAO-B	5-HT	5-HIAA	DA	HVA
CBA	1.24±0.04	0.30±0.01	331±13	344±21	900±57	201±7
BALB	1.18±0.06[a]	0.21±0.05[a]	262±12[a]	313±21[a]	808±37	306±32
C57	1.28±0.02	0.27±0.01	319±10	380±13	508±15[a]	196±6[a]

Note: The mice were all 16–19 days old. [a]Lowest value.
Source: Oreland & Fowler 1982.

to sensation seeking, there is thus some evidence for a relationship between sensation seeking and serotonin turnover in the CNS.

Zuckerman moves from an optimal level of arousal theory to an optimal level of catecholamines theory. He writes: "It is with great reluctance that I am beginning to reentertain the idea of an optimal-level theory," and it is easy to understand why he is reluctant. Carroll, Zuckerman, and Vogel (1982) rejected the optimal level of arousal theory of sensation seeking on the basis of drug experiments, and the criticism would be as valid against an optimal catecholamine level theory. Even if the relationship between serotonin and sensation seeking could be more firmly established, it seems unlikely that sensation seeking could be explained by means of an optimal level of serotonin theory.

We are aware today of at least 34 transmitter systems in CNS, most of them not investigated in relation to sensation seeking, and we know very little about the complicated interactions among these systems. It seems more likely that sensation-seeking behavior is a result of a balance between two or more transmitter systems than it is related to an optimal level of one single transmitter system.

The noradrenergic locus coeruleus – the center of attention?

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Zuckerman's theory ranges widely and invokes all of the monoamines – noradrenaline, dopamine, and serotonin – but without differentiating a separate role for each. This is a pity, since more than enough evidence now exists to show very different effects on behaviour of manipulating each amine independently. To suggest that they are all somehow involved in sensation seeking in an undifferentiated fashion is to rediscover the law of mass action on a neurochemical footing. Just as psychology has moved away from Lashley to sharp localisation of function in the brain, so too should the neurochemical bases of behaviour progress from the early formulations of the sixties about amines in general to the hypotheses of the eighties about dopamine versus noradrenaline versus serotonin (see the extended discussion in Mason 1983a). Thus, Zuckerman's theory is a retrograde step in this respect.

The support the author offers for his theory from the existing research literature is also rather selective and episodic. Thus, long before Gray (1982) "argued forcefully against the Stein-Crow theory concerning the role of NE in reward" others had indeed produced the experimental evidence to discredit this view (in temporal order, Mason & Iversen 1975; Amaral & Foss 1975; Roberts, Price & Fibiger 1976; Sessions, Kant & Koob 1976; Peterson & Laverty 1976; Mason & Iversen 1977a; 1977b; 1977c; Roberts & Fibiger 1977; Price, Murray & Fibiger 1977; Ogren & Fuxe 1977; Koob, Kelley & Mason 1978; Mason & Iversen 1978a; 1978b; I stop at 1978 purely arbitrarily, subsequent workers have continued the trend).

Zuckerman also seems unaware of previous theoretical formulations in the literature that could lead directly to a role of amines in sensation seeking. Thus Mason and Iversen (1977b; 1979) have suggested a role for noradrenaline in selective attention. The evidence, as reviewed by Mason (1980; 1981; 1983a; 1983b) indicates that the noradrenergic system, originating in the locus coeruleus and running via the dorsal bundle to provide the sole noradrenergic innervation to the hippocampus and entire cerebral cortex, serves to filter out irrelevant stimuli impinging on the organism. This is based on interventions in the central noradrenergic systems a good deal more direct and specific than the studies of platelet MAO that Zuckerman cites at length – hence the title of the present contribution.

Mason and Iversen (1975) destroyed the dorsal bundle noradrenaline projections from the locus coeruleus to the cortex in the rat and found that these animals responded for longer than normal in extinction situations (dorsal bundle extinction effect) in a way that suggested increased sampling of environmental stimuli during acquisition learning (reviewed in Mason & Iversen 1979). Roberts et al. (1976) found that such noradrenaline-depleted rats were more distractable than normal by stimuli irrelevant to the performance of a previously learned runway response. Sophisticated tests of attentional sampling such as latent inhibition, nonreversal shift, and blocking confirmed a change in the animal's ability to ignore irrelevant stimuli as a result of loss of forebrain noradrenaline (see Mason 1980; 1981).

Other workers have also reached the conclusion that noradrenaline is involved in stimulus processing from their use of recording techniques. Thus, Aston-Jones and Bloom (1981) found that single cells in the rat locus coeruleus increased their firing when novel visual, auditory, or somatosensory stimuli were presented to the freely moving animal. When the animal was engaged in grooming or consumption of sweet water, and so not attending to these visual stimuli, a much smaller response was seen from locus coeruleus cells. The converse finding was demonstrated by Segal and Edelson (1978): Electrical stimulation of the locus coeruleus just prior to each trial in a runway significantly sped up reversal of a black-white discrimination, where a response had to be emitted to a previously unrewarded visual stimulus.

If, as these direct manipulations indicate, brain noradrenaline systems are involved in the control of stimulus sampling it might be possible to devise perturbations of them that would lead, via homeostatic mechanisms, to the active seeking out of stimuli and sensation. (Conversely, changes in the opposite direction might lead to the avoiding of stimuli; and indeed Mason, Roberts & Fibiger [1978] have shown enhanced neophobia consequent to manipulation of brain noradrenaline systems.)

The strange thing is, then, that despite his reluctance to cite the relevant references and his disinclination to differentiate roles for each catecholamine, Zuckerman's theory may well be basically sound. The Roman emperor Vespasian, when pressed by his critics for having placed a tax on public urinals, is reputed to have replied, "Money has no odour." Perhaps, in the neurosciences, too, good ideas may arise from base beginnings.

Physiological substrates of a psychological dimension

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Some may argue that a search for the biological underpinnings of a dimension of personality that is typically quantified as test scores or factor-analytic results is overly ambitious and premature. Others may suggest that it ensures parsimony and conceptual economy by subsuming a variety of related behaviors under a "construct" (sensation seeking); thus, comprehensiveness of approach and identification of biobehavioral relations that might otherwise go undetected are built in *ab initio*. Zuckerman has presented an extensive review of the biology of the sensation-seeking dimension in a fashion rather persuasive of the approach's potential value. In contrast, earlier references to possible biological substrates of personality dimensions have been rather cursory at best (e.g. Cattell & Scheier's 1961 description of a dimension of stress reaction as "ACTH-related response patterns").

Often, biological accounts of personality variables have been restricted to specific emotion-related behaviors such as "condi-

tioned suppression" and "learned helplessness" (e.g. Anisman 1978). In the area of psychopathology, animal behavioral models, and corresponding analyses of physiological substrates, have usually addressed rather circumscribed escape-avoidance behaviors (relevant to phobias) or stereotyped responding (relevant to compulsions; see Costello 1970; Eysenck 1973). An approach emphasizing discrete behaviors has usually been "safer" than searching for substrates of a dimension – a description of a characteristic held in common by a variety of behaviors. In the former case, after a reliable biobehavioral association is identified, generalization to a parent construct of which the focal behavior is a "correlate" is not at issue. On the other hand, the present approach invariably carries the following risk: The biological correlates of the construct- or dimension-specified behavior (e.g. participation in hazardous sports) may, in turn, carry a trivial or zero relation to the personality dimension to which the behavior itself is imperfectly related. It is a well-known psychometric dictum that things correlated with the same thing (e.g. sensation seeking and specific biological events, each correlated with a given behavior) are not necessarily correlated with each other (the specific biological events and sensation seeking). The applicability of this caveat varies with the patterns and sizes of obtained relations. For instance, few would contend that two variables, each correlated with a third to the magnitude of .80 or greater, are not also significantly related to each other; there would be more contention if the values were .35 or less. But since, in the present review, numerical indices of strengths of association are seldom reported, it is difficult to establish one's degree of belief in the validity of the various sensation-seeking substrates presented throughout the article.

Undoubtedly, the most straightforward solution to such difficulties is to include biological variables in analyses which incorporate the definitive self-report (questionnaire) "markers" of sensation seeking, and to examine the tendency for both sets of variables to load on the same factor(s). Such analyses are mentioned in the target article, and it is noted that their results are generally concordant with the hypothesized biological correspondents of the focal scale or subscale scores. An issue not unlike the previous one concerning "degree of association" is raised, however. It is reported throughout Zuckerman's review that the sensation-seeking dimension is not factorially orthogonal to those of the Eysenck group (e.g. "neuroticism" and "extraversion"); similarly, it is somewhat negatively related to fear, according to factor analytic and other findings from "animal emotionality" studies. Of what magnitude is the factorially orthogonal portion of the sensation-seeking dimension? And what proportion of variance remains after the association with related "dispositional" dimensions such as fear has been statistically removed? Would sensation seeking be prominent in, for example, a second-order factor analysis in which the Eysenck dimensions of "extraversion" and "neuroticism" were included? The question is, To what extent can the present dimension be described simply as a "linear combination" of other, longer-established ones, and to what extent is it a necessary complement to the latter dimensions for a comprehensive description of salient personality continua? Further, can one expect to find biological substrates unique to a specific dimension such as sensation seeking, when it is aligned with other prominent dispositional traits? Perhaps only a penetrating evaluation of the numerical coefficients of intervariable relations mentioned above will provide the answer.

The preceding reservations aside, the biology of personality as put forth by Zuckerman and by Eysenck and Gray carries considerable heuristic stimulation to personologists working strictly from behavioral, self-report, and psychophysiological levels of analysis. But this value stems from a somewhat broader perspective involving the relative merits of neurochemical and electrophysiological accounts of personality variables as a "domain of explanation"; the opposing domain consists of somewhat more diffuse cognitive-behavioral accounts. For example, in assessing responses to stressing stimulation among more "anxiety prone" individuals, it has been found that neurosensory formulations surrounding this trait as put forth by Eysenck (1967; 1970a) and Gray (1970; 1971) appear to be more accurate than those involving "cognitive appraisal of stress" (see Dobson & Neufeld 1981; LeFave & Neufeld 1980; Neufeld & Herzog 1983). A similar assessment may favor the biological-mechanism approach to sensation seeking as espoused by Zuckerman. Of course, as the general orientation toward a particular level of explanation (biological) is vindicated in this way, pursuit of the responsible mechanisms operating at that level is strongly endorsed.

Spanning the transspecies gulf

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The general approach Zuckerman is advocating for understanding the bases of human personality is timely and ambitious. Considering how little success such approaches have had in the past (because of our inadequate understanding of the brain), Zuckerman's analysis will no doubt address only a small part of the problem. Because such approaches will become more influential in the future, we would like to raise the question of how scientific cross-fertilization could be improved not only so that neuroscience can continue to provide a substructure for understanding subtle psychological constructs, but also so that the higher levels of analysis can begin to generate superstructures to guide the analysis at more basic levels. The time is ripe for reciprocity, for it is a common hope that we will eventually learn to discuss the basic processes all mammals share in a single, mutually clear, and useful language. The many facts from neuroscience will continue to assume the form of a disorganized heap of fine building materials (which remains evident in Zuckerman's extensive coverage of the neuroscience-psychobiology literature), unless they can be used to construct a credible and sturdy functional structure. Although such "back fertilization" was not highlighted by Zuckerman, and may not be his concern, it is something we would ask him to consider. His molar concept of "sensation seeking" should apply compellingly across all mammalian species if it is to be an attractive concept for psychobiologists.

Although advances in understanding the bases of human behavior will surely depend critically on advances in neuroscience, conversely, the promise of many findings in neuroscience will be realized only through accurate neurobehavioral taxonomizing. There are many instinctual, environmentally refined brain processes that humans share with other mammals, including some abhorred by classical behaviorism, and it seems evident that only the joint analysis of the human mind and the animal brain can guide a behavioral taxonomy. Accurate classification of intrinsic control systems should highlight the natural "cleavage lines" of neurobehavioral control circuits. Although it is self-evident that all organisms seek to stimulate their senses, sensation seeking, as conceptualized in Zuckerman's behavioral work, seems more a conditional than an unconditional neurobehavioral dimension – similar to other acquired traits such as dominance–submission, assertiveness–passivity, altruism, chastity, and piety. Although all such processes must, in the final accounting, have powerful biological underpinnings, sensation seeking is, almost of necessity, a heterogeneous learned expression of several concurrently interacting neurobehavioral systems – especially those that mediate emotions. Unless such intermediate levels of analysis are adequately constructed, the reductionistic chasm may prove too wide to cross. From such a perspective, it seems unlikely that Zuckerman's unidimensional

brain analysis of a multidimensional psychological trait will be a resounding success, although it certainly seems on a correct track.

Perhaps the pertinence of a concept such as sensation seeking is largely restricted to the behavior of humans living in technologically advanced societies (and their pets and laboratory animals), where material well-being is taken for granted. In many human societies, basic bodily needs are satisfied more by established institutions – the "safety nets" of human cultural evolution – than by individuals' foraging behavior. Such lifelong societal nurturance can generate ever-increasing leisure time, boredom, and hence sensation-seeking behaviors that are not directed at essential survival goals. Although both sensation-seeking and life-sustaining "foraging" could be elaborated by the same circuits, it seems to us misplaced emphasis to conceptualize the shared underlying mechanisms as sensation-seeking processes of the brain.

The catecholaminergic circuits which Zuckerman argues control sensation seeking, have already been conceptualized as important nonspecific neural systems for mediating sensory–motor processes essential for efficient goal-directed behaviors in animals. These and closely related systems help generate exploration–investigation–foraging, the appetitive eagerness and attention that animals direct at all incentives. Such circuits are surely involved in comparable human behaviors, but there is no cogent evidence that they should be considered as other than nonspecific, permissive ingredients in much broader functional and neurochemical schemes. Descending circuits, perhaps cholinergic from basal forebrain areas projecting to the ventral tegmentum, may be as essential for the generation of coherent investigative–foraging activities as the dopamine cells on which they synapse. Further, opioids (which, contrary to Zuckerman's assertion, can activate dopmaine systems; Bozarth & Wise 1981; Broekkamp, Phillips & Cools 1979; Joyce & Iversen 1979), GABA, neurotensin, serotonin, substance P, and other amino-acid and peptide transmitters all converge on dopamine cells of the meso-diencephalic junction which presently appear so important for mammalian appetitive behaviors; and all may be as important for the generation of sensation seeking as catecholaminergic ones.

More interesting, perhaps, is that human cultural evolution, having freed so many of us from pursuing immediate survival needs, has now established an environment in which brain evolution of some humanoid lines is yielding totally new adaptive brain circuits whose function is to alleviate boredom. If that is the case (assuming that frontal lobe tissue and the basic play circuits of the brain have not already brought it to pass), then we might anticipate that catecholamine systems will continue to remain involved, as they are now, with practically every psychological trait constructed by the brain. However, considering the widespread dispersion of these systems, many behavioral changes that have been attributed to catecholamines will actually be due to effects on overlapping systems. For instance, contrary to Zuckerman's assertion, the induction of hyperphagia by damage in the vicinity of the ventral norepinephrine system (Ahlskog & Hoebel 1973) does not appear to be simply due to catecholamine depletion (Lorden, Oltmans & Margules 1976).

Granted that catecholamines will have to participate in any trait as broad as sensation seeking, the "optimal level" or "adaptation level" approach provides an attractive focal point for Zuckerman's theorizing, as it did for Harry Helson's (1964). However, the tricky issue of how the "set point" for the system is determined remains as enigmatic as ever. More troublesome is the ability of Zuckerman's version of adaptation-level theory to work prediction in all possible directions depending on the degree of presumed activity in the underlying systems. Although this may be the way the system is constructed, to prevent the theory from explaining everything and hence nothing, more effort should be devoted to specifying how we extricate ourselves from such troubles.

Biochemical substrates for a human "sensation-seeking" trait

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Zuckerman's attempt to integrate a large body of mostly correlative biological data with a sensation-seeking trait in humans is heroic and provocative. The data from human studies which show stable factors for thrill and adventure seeking, experience seeking, disinhibition, and boredom susceptibility, all classed together as "sensation seeking," are most convincing. The attempt to relate these factors to neurochemically defined brain systems is less successful, for reasons Zuckerman has outlined. The following description of problems should not be taken as criticism of the effort, but as a suggestion that the difficulties may be too great and the reliable data as yet too meager to support an integrative theory that attempts to encompass depression, anxiety, boredom, euphoria, sociability, and sensation seeking in the same schema.

The most direct data are based on correlations between CSF (cerebrospinal fluid) monoamine concentrations or enzymes in CSF, platelets, or plasma, and factors that measure sensation seeking in humans. The highest sensation seekers have the lowest concentrations of CSF norepinephrine, for example. These same studies also suggest a relationship with the intervening variable of "anxiety," which is consistent with other correlative psychometric studies. Unfortunately, none of the biological data presented can be interpreted unequivocally as showing either increases or decreases in functional activity of specific neurochemical systems in the central nervous system. Low or high monoamine enzyme activities might be part of homeostatic mechanisms either to increase or to reduce function. Low concentrations of CSF metabolites might result from feedback inhibition in a system with sensitized postsynaptic receptors at a high level of functional activity, or high concentrations might result from increased turnover to overcome subsensitivity or loss of neurons. Zuckerman, in the good company of most biological psychiatrists through the mid-1970s, draws at least tentative conclusions regarding the direction of these effects in his integrative efforts. For example, in the theoretical formulations (Figure 6), he locates major depression on his scale with a catecholamine deficit, according to the old catecholamine hypothesis (Bunney & Davis 1965; Schildkraut 1965); but newer more direct data from the effects of antidepressant drugs (Huang 1979; Huang, Maas & Hu 1980; Vetulani & Sulser 1975) and the measurements of monoamine metabolites from CSF instead of urine (Koslow, Maas, Bowden, Davis, Hannin & Javaid 1983) are suggesting that depression fits better at the upper range of noradrenergic activity (along with anxiety, with which it often coexists). Other examples of possibly incorrect inferences regarding the direction of differences in catecholamine function are related to differences between BALB and C57B mouse strains and the functional effects of 6-hydroxydopamine lesions (where effects have been shown to result in functional hyperactivity as well as hypoactivity; Acheson & Zigmond 1982; Kostrzewa & Jacobowitz 1974; Sporn, Harden, Wolfe & Molinoff 1976).

Zuckerman's recognition of the importance of both naturalistic and experimentally controlled animal studies is to be applauded. Without such studies, it is difficult to surmount the limitations of the correlative approach in humans. Yet the extensive review of relevant animal literature underscores a major limitation of the approach: There is no "model" of sensation seeking in animals (or in my opinion, of human semantic constructs of the emotions in general). The experimental paradigms that may be relevant suggest simultaneously the numerous intervening variables that complicate the interpretation of the human data, such as anxiety, arousal, motor activity, so-

ciability, dominance, appetite, and reward. Since there are considerable data to suggest human trait differences in each of these dimensions, and since state-dependent changes in these areas might also influence sensation seeking directly or indirectly, the linkage to a sensation-seeking trait becomes very complex and confounded. This confusion is often resolved by premature interpretations from animal studies which obscure important differences or lead us in the wrong direction (my critics believe that I am guilty of this, too). A few specific examples (which Zuckerman repeats from the original investigators) should suffice: "fear reactions" are the interpretation of behavioral responses to unfamiliar or novel foods; failing to rear or move is interpreted as being "helpless." Each of these labels obscures the equal probability that other variables in the experimental situation contribute to the effect described (as Zuckerman suggests that social effects in the open-field test may contribute as much to the result as anxiety). An even more serious problem occurs with the grouping of distinguishable neurochemical systems: Single neurotransmitters have a variety of sometimes different functions in the brain depending upon the location, the concentration, and the combination with other neurotransmitters, hormones, or drugs. Norepinephrine, for example, has alpha-1, alpha-2, beta-1, and beta-2 adrenergic receptors which mediate completely opposite excitatory or inhibitory effects depending upon location, concentration, and other simultaneous influences (Woodward, Moises, Waterhouse, Hoffman & Freedman 1979). Dopamine also has several distinctive and functionally independent systems which, although interconnected with NE and other systems, nonetheless operate independently. Combining dorsal noradrenergic bundle lesions of specific noradrenergic projection systems with effects of the entire locus coeruleus system, or combining all dopamine systems and all norepinephrine systems into *catecholamine systems activity* (CSA) obscures important differences.

Finally, aspects of my own studies, with a number of collaborators, are supportive of Zuckerman's overall thesis at least to the extent that anxiety may be involved as an inhibitor to sensation seeking. As might be predicted, I believe that new data are more convincing than those cited by Zuckerman; and some valid criticisms have led to additional studies. Although electrical field stimulation of the locus coeruleus in monkeys is certainly not (as Zuckerman notes) a physiological activation, the firing rates produced are not inconsistent with the spontaneous rates of firing measured in awake animals (Grant, Huang & Redmond 1980). Furthermore, the behavioral effects produced by this stimulation are identical to the behavioral effects of a "natural" fear stimulus (such as a facial threat gesture from a human) and of a conditioned stimulus (light previously paired with electrical shock; Redmond & Huang 1979). Identical behaviors are produced by two drugs which activate the spontaneous firing of the locus coeruleus and probably the entire central noradrenergic system as well. Both of these drugs produce increased anxiety in human subjects (Charney, Heninger & Redmond 1983; Holmberg & Gershon 1961; Soffer 1954). Unrestrained monkeys, free to move about a large cage, show many of the same behaviors that are seen in chair-restrained monkeys during locus coeruleus electrical stimulation, but in addition show walking, circular pacing, running, and jumping up to a perch (Redmond, Huang & Grant 1983). With regard to consummatory behavior, there is some evidence that eating in primates is affected by NE systems in a direction that would be easier to integrate into Zuckerman's overall theory than the rodent data: Locus coeruleus lesions in monkeys cause large increases in food and water intake, and electrical stimulation at levels that are too low to produce any other detectable behavioral or motor effects will inhibit eating in food-deprived monkeys (Leverenz, Redmond & Huang 1978; Redmond, Huang, Snyder, Maas & Baulu 1977). These effects are consistent with the effects on human appetite of a number of drugs that cause weight gain or appetite suppression (tricyclics and amphetamines). Many new data suggest, however, that the old appetite literature from rodents is not inconsistent with these findings. Even with regard to the behavioral effects of lesions and the pharmacology of the locus coeruleus, not all studies are as negative concerning the involvement in anxiety as the data mentioned by Zuckerman would suggest (Redmond & Huang 1979). The conditioned startle reflex (Davis & Astrachan 1978; Davis, Cedarbaum, Aghajanian & Gendelman 1977; Davis, Redmond & Baraban 1979) in rodents is entirely consistent with much of Zuckerman's interpretation and with the effects of anxiolytic drugs in humans as well as the predicted effects of conditioned fear in animals.

Zuckerman's stimulating review should focus further attention on a well-established and reliable psychometric phenomenon. Many of his interpretations, and those of the original authors, might be as adequately explained in other ways; and substantial pharmacological or biochemical data are sometimes inconsistent. The answer as to which interpretations of human emotions and behaviors are correct must come from hypothesis testing in humans. I look forward to the results from the tests Zuckerman and his collaborators seem to be involved in now.

ACKNOWLEDGMENTS

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The concept of sensation seeking and the structure of personality

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I am concerned about the nature of the concept of sensation seeking, and where it fits, if at all, as a component in the structure of personality.

Although Zuckerman refers to sensation seeking as one of the basic dimensions of personality, his analysis in the target article suggests that it is not a trait, but a behavioral complex or a type which is to be accounted for in terms of such dimensions. The clearest expression of this state of affairs occurs in the section "Relationships to primary dimensions (Eysenck's) of personality," where Zuckerman says that sensation seeking is a function of Eysenck's extraversion and psychoticism dimensions. Furthermore, Zuckerman points out that sensation seeking breaks down into the four factor components of thrill and adventure seeking, experience seeking, disinhibition, and boredom susceptibility. If the concept of sensation seeking can be accounted for by Eysenck's extraversion and psychoticism factors on the one hand, and is reducible to four lower-order factors on the other, how are we to regard the concept?

Upon first reading one might come to the conclusion that sensation seeking should be viewed as one of the basic personality dimensions, such as Eysenck's dimensions. Detailed analysis, however, suggests that it is a complex of some sort, probably a behavioral complex that identifies high and low sensation-seeking types, which are to be accounted for by such personality dimensions.

As part of a general theory of individual differences in personality (Royce & Powell 1983), I have developed a subtheory of the affective system which involves a synthesis of the research of Cattell (1965), Eysenck (1970b), and Guilford (1967), among others. The result is a hierarchical structure (Figure 1) of factors at four levels of increasing generality. For example, Eysenck's

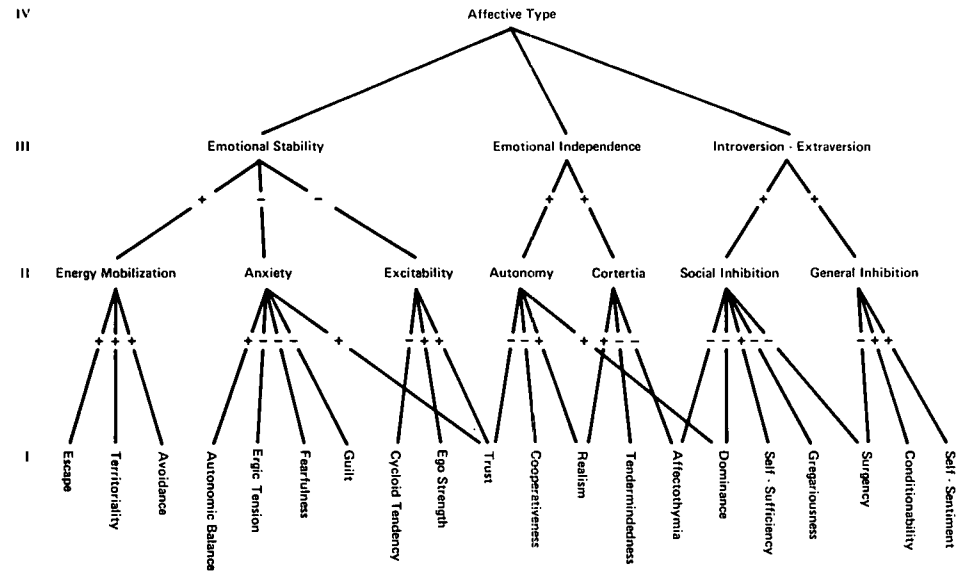

Figure 1 (Royce). The hierarchical structure of the affective system (modified from Royce & McDermott 1977).

three factors appear at the third level as emotional stability (Eysenck's neuroticism), emotional independence (Eysenck's psychoticism), and Eysenck's introversion–extraversion.

Although Zuckerman's elaboration of the biological bases of sensation seeking is impressive, relatively little has been done to determine the psychological significance of this concept. In the spirit of promoting further inquiry along these lines, I, therefore, pose two questions. First, what kind of a concept is sensation seeking? Specifically, is it a basic personality dimension or is it a behavioral complex or type which is to be accounted for by such dimensions? Second, if it is claimed that sensation seeking and its four factor components are basic personality dimensions, where do they fit in the structure of personality?

From my perspective sensation seeking appears to be a behavioral complex which is characterized (for high sensation seekers) by high emotional independence and extraversion at the third order and by high surgency, dominance, and gregariousness at the first order (see target article section "Sociability and dominance" regarding the first-order factors). As pure guesswork (because of the lack of evidence), I also hypothesize that high sensation seekers would be low on the affectothymia, conditionability, self-sufficiency, and self-sentiment dimensions. The profile for low sensation seekers would be exactly the opposite: low emotional independence and high introversion at the third order, low surgency, dominance, and gregariousness at the first order, and probably high on affectothymia, conditionability, self-sufficiency, and self-sentiment.

Sensation seeking: Exploration of empty spaces or novel stimuli?

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Zuckerman correctly points out that "the open-field test may not be the best model for sensation seeking in nonhuman species." Many of the problems inherent in the use of this procedure have been delineated by Walsh and Cummins (1976), and further clarified by Simmel and Bagwell (1983). The most important of these problems, and the one most pertinent to Zuckerman's discussion, is the conceptual (and phenotypic) confusion resulting from operationally defining just what an animal does in the open field: Ambulation becomes activity, activity becomes exploratory activity, and exploratory activity = exploratory behavior. The last of these, exploratory behavior, would seem to have a great deal to do with what sensation seeking is all about, and studies using genetically controlled animals in open-field tests would therefore seem to provide highly relevant input into understanding the genetic sources of individual differences in sensation-seeking behavior. But what is actually being measured in the open field? What if other procedures, more closely related to sensation seeking, were to provide genetic data contradictory to those obtained from the open field? In the remainder of this commentary I provide evidence suggesting that this is indeed the case.

Is the responsiveness of an animal to novel stimulation necessarily correlated with ambulation in an open field? The usual testing procedures require the subject to ambulate toward or away from novel stimuli (which are, in any case, rarely present in the usual open-field situation). In my laboratory, we have developed a procedure that permits us to measure stimulus-seeking (exploratory) behavior separately from activity by permitting the subject to move about in a plain compartment or within a compartment of equal size decorated with novel and complex visual stimuli, or to move freely between the plain and novel compartments. This procedure (described more fully in Simmel & Eleftheriou 1977 and Simmel & Bagwell 1983) provides four separate measures: latency to the initial entry into the novel compartment; proportion of time spent in the novel compartment; crossings between the plain and novel compartments; and total bodily activity, derived from an activity meter upon which the apparatus is placed. A factor analysis of these measures from 132 mice representing 11 genotypes revealed the presence of two factors. Latency and novel-side time loaded only on the first factor (termed "stimulus reactivity"); activity loaded solely on the second factor (gross activity); crossings between the two sides loaded about equally on both factors (Simmel & Eleftheriou 1977). This basic factor structure has been replicated with dogs (Wright 1980), gerbils (Rosenfeld, Lasko & Simmel 1978), and rats (Haber & Friedman 1979). The conclusions seem to be straightforward: Responsiveness to novel stimulation is not necessarily correlated with activity. Therefore, performance by animals in an open field devoid of novel stimuli is not an adequate measure of exploratory behavior.

Are the genetic differences found for stimulus reactivity

compatible with those obtained from open-field tests? Zuckerman cites data showing that on open-field tests, BALB/c mice exhibited very low "exploratory" activity, whereas C57BL/6 (B6) mice had very high scores. Our results were contrary (Simmel & Eleftheriou 1977): B6s had very low stimulus reactivity scores, and the BALBs had very high ones (e.g. for novel-side time, BALB = 322 sec.; B6 = 192 sec.). The two strains did not differ on activity. Further analysis revealed that the low exploration scores of the B6 mice were due to a neophobic reaction to novel stimuli in this strain. This is supported by findings from an earlier study (Simmel & Walker 1972) using the closely related C57BL/10 strain, in which we found that the B10s would more readily approach a novel object if a companion was present.

The data I have presented here are not incompatible with Zuckerman's proposed trait, sensation seeking. On the contrary, our results from studies with genetically controlled animals in situations in which they respond either positively or negatively to novel stimulation suggest that his approach may well prove to be a most valuable one. The contradictions between some of the data cited by Zuckerman and some of the data I have presented do point out the dangers inherent in accepting conclusions drawn from studies employing inappropriate operational definitions. If the genetic differences in the behavioral phenotype turn out to be 180° different from what was stated, can the underlying biological phenotypes be on the correct course?

Sensation seeking and the orienting reflex

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My first objection concerns the loose definition of the term "sensation seeking" which includes both the nonspecific response to novelty and the tendency to seek specific attractants. The contradictions arising from a comparison of sensation seeking and the orienting reflex are due to the fact that these concepts fail to coincide.

The orienting reflex as an element of investigative behavior is characterized not only by motor, vascular, and electroencephalographic manifestations but also by a specific exploratory drive, directing the organism toward novel stimuli, complex objects, and unfamiliar surroundings. This motivational aspect of the orienting reflex coincides with sensation seeking for novelty. Sensation seeking for attractants differs, however. The separation of the novelty drive from sensation seeking in general opens a new perspective for research on individual differences, one that is specifically related to exploratory behavior.

My second objection concerns the absence of single unit analyses of sensation seeking in animal models. An attempt to build a bridge directly from behavior to neurotransmitters without examining the neuronal level of integration is of doubtful value because one transmitter may affect various postsynaptic receptors differently. Individual differences should be attributed to neuronal mechanisms with characteristic pre- and postsynaptic features.

Sensation seeking, orientation, and defense: Empirical and theoretical reservations

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Overall, I endorse Zuckerman's attempt to exploit physiological, psychophysiological, and pharmacological research with animals and humans in developing an understanding of the sensation-seeking trait (SS). My comments on this enterprise are intended to draw attention to some of the difficulties in applying concepts from Soviet psychophysiology, the orienting (OR) and defense (DR) responses, in accounting for differences between high and low sensation seekers in their need for novel stimulation.

First, it must be acknowledged that the data base for evaluating individual differences in the electrodermal OR is quite limited. Only one of the studies cited (Ridgeway & Hare 1981) has been published at this time, and the experimenters failed to demonstrate the enhanced initial OR for high SS subjects reported by Neary and Zuckerman (1976). In terms of OR theory (Sokolov 1963), the OR is thought to facilitate sensory reception, attention, and conditioning. If high and low SS groups differ in OR, one would expect such a difference to be expressed in the enhanced sensitivity of high SS in psychophysical tasks and in their superior performance in vigilance and conditioning experiments. There appears to be a paucity of such demonstrations. Moreover, on the basis of the positive relation between SS and the extraversion (E) and psychoticism (P) dimensions of Eysenck's classification one would expect the difference to be in the direction opposite to those derived from the Neary and Zuckerman (1976) report. It is subjects low in E who display enhanced sensitivity, vigilance, and conditioning (cf. Stelmack & Plouffe 1983); subjects low in P also display enhanced conditioning to stimuli of moderate intensity (Beyts, Frcka & Martin 1982).

It is important to note that there is an intriguing contradiction between the positive relation of SS with electrodermal OR, observed by Neary and Zuckerman (1976) and the skin conductance effects reported for E (Smith 1983; Stelmack 1981) and psychopathy (Hare 1975). Although SS is positively related to E and psychopathy, both of these dimensions are *inversely* related to electrodermal responsiveness. In a recent examination of these paradoxical effects (Stelmack, Plouffe & Falkenberg 1983), significant negative correlations between SS and initial SCR to pictures were observed ($N = 98$) whereas significant positive correlations between SS and initial SCR (skin conduction response) to words were found ($N = 46$). Analysis of the data indicated that the positive correlation of SS with SCR was influenced by differences in skin conductance level (SCL), with low SS displaying a lower SCL in the word condition than in the picture condition. Partial correlation analysis, controlling for differences in SCL, removed the significant effects in the word condition. Since SCL was measured prior to the first stimulation, the contradictory results cannot be attributed to differences in the specified stimulus conditions but must be due to either sampling differences, or, more likely, an unobtrusive experimental effect which lowered SCL for the low SS group. In any event, before the electrodermal OR can be usefully employed to account for differences between high and low SS in response to novelty, the SS effects must be reconciled with the paradoxical effects observed for the related dimensions of E and psychopathy and integrated with predictions from OR theory. Our data suggest that the circumstances of the experiment may be significant in influencing the direction of differences in electrodermal response to stimulation of high and low sensation seekers.

Second, with respect to differences in the need for novel stimulation, it is not clear from the discussion of the OR whether the effects are meant to refer to differences in response to novel stimulus change in general or whether they simply reflect a differential sensitivity to stimulation. In stimulus change paradigms where a change stimulus is introduced following a habituation series, the OR has been shown to be equally sensitive to an increase or decrease in stimulus intensity of the same magnitude. The OR can also be elicited if the change event is the omission of a stimulus from a regular series of stimuli (Sokolov 1963). These demonstrations have established that the OR is a

response to novelty or change per se and is not solely a function of stimulus intensity. In the absence of such demonstrations showing differences between high and low sensation seekers, it may be more precise to refer the electrodermal effects to differential sensitivity or reponsiveness to stimulation rather than to apply the more general term OR to the effects or to refer the effects to differences in response to novelty.

There are also some difficulties in interpreting the cardiac data in terms of DRs. One feature thought to distinguish a DR from an OR is that for the DR, heart rate acceleration is resistant to habituation (Graham 1979). Since the cardiac effects, showing accelerative responses for low SS and decelerative responses for high SS, were apparent only on the first one or two trials, it is doubtful that this criterion of a DR was met. What is clear, however, is that with increasing stimulus intensity there is an increasing incidence of accelerative responding (Turpin 1979). In this respect, the high SS respond as if the stimulus were less intense. The "augmenting" of the visual evoked potential by high SS is a similar effect. Overall, it seems to me that the psychophysiological data may be best viewed as evidence of the lower sensitivity to physical stimulation for high SS. This viewpoint is congruent with that which emerges from a similar analysis of the SS-related dimensions of E (Stelmack 1981) and psychoticism (Stelmack et al. 1983) in Eysenck's system of classification. The glaring contradiction is the enhanced electrodermal response of high SS to moderate intensity stimulation reported by Neary and Zuckerman (1976). Clearly a series of investigative studies are required to determine the conditions under which consistent differences in psychophysiological responses between high and low sensation seekers emerge and to determine the functional significance of such effects.

Zuckerman's sensation-seeking theory: A view from Eastern Europe

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Marvin Zuckerman is one of the very few differential psychologists to develop a theory of personality dimension skillfully combining the correlational and the experimental approach, studies of human beings and of animals, and behavioral and biochemical measures. This multidirectional approach has given rise to a causal theory of individual differences in sensation seeking.

There is growing interest in this personality dimension. Its popularity is not accidental, but is, in part, a reflection of the fact that the second half of our century appears to be creating more and more situations involving extreme deprivation or overstimulation (Strelau 1983). Under such conditions, sensation seeking, a personality dimension associated with adaptability to the stimulation level of the environment, holds considerable promise. The second reason for its growing importance is the nature of sensation-seeking theory itself. Zuckerman's coherent, multidisciplinary approach, presented in a flexible framework, stimulates many psychologists, psychophysiologists, behavior geneticists, and investigators studying biochemical correlates of behavior to test the many hypotheses his theory offers.

It is impossible to discuss in a short commentary all the problems this theory deals with; therefore, I shall limit myself to some remarks from the point of view of my own research on temperament.

The sensation-seeking trait, as measured by the SSS, is not a unitary phenomenon. The data collected by Zuckerman (1979b; 1980) and other authors (e.g. Stewart & MacGriffith 1975) show that it is composed of four factors: thrill and adventure seeking, experience seeking, disinhibition, and boredom susceptibility. Zuckerman's theory, although biologically oriented, does not offer any hypothesis to explain this differentiation of sensation seeking on the level of physiological or biochemical mechanisms. There is some evidence that suggests that the biological correlates may differ for different scales. This follows, among other things, from the fact that the heritability estimates of sensation seeking differ, depending on which scale is taken into account (Zuckerman 1979; target article). Analyzing the content of items of the four scales included in the SSS, one cannot avoid thinking that the ways in which individuals seek or avoid sensation are explicable by social-environmental hypotheses (see, e.g., Farley 1973).

It is not accidental that Zuckerman (1979b) uses the notion of *sensation* instead of *stimulation*. There are several reasons for this: For example, "it is the sensory effects of external stimulation that are most important" (Zuckerman 1979b, p. 10) not the stimulation itself; unusual sensations are the product of extraordinary activities and situations, and even when these are the same, they may evoke different sensations in different individuals. Most important for the understanding of the significance of the environmental factor in Zuckerman's theory, however, is his interpretation of the definition of sensation seeking. By the latter he means "the need for varied, novel, and complex sensations and experiences and the willingness to take physical and social risks for the sake of such experience" (1979b, p. 10). "The phrase 'varied, novel, and complex sensations and experiences' describes the qualities of stimulation that are valued by a sensation seeker" (p. 11). Risk, which is also a component of Zuckerman's definition, is in turn defined as "the appraised likelihood of a negative outcome" (p. 11). The processes of *evaluation* and *appraisal*, which are important in Zuckerman's theory, imply that we are dealing with a *cognitive* theory of sensation seeking, which may be contrary to Zuckerman's own intention. His concept reminds us of the cognitive, interactional theory of stress developed by Lazarus (1966), McGrath (1970), and others. If this is valid, the understanding of the interaction between biological and social factors in determining sensation seeking can hardly be neglected. This is not to say that Zuckerman does not feel the need for environmental studies in his personality dimension; but his statement in the target article that "it is difficult to advance on two fronts simultaneously" does not solve the problem.

Comparing the sensation-seeking dimension with other personality traits, Zuckerman (1979b; 1980; target article) mentions the concept of strength of the nervous system (more precisely, the strength of excitation) developed by the neo-Pavlovian typologists (Nebylitsyn 1972; Teplov 1961). The traits of the sensation seeker are regarded as corresponding to those of the strong type of nervous system (NS), those of the sensation avoider correspond to the weak NS. Zuckerman bases this comparison partly on the studies in which the augmenting–reducing phenomenon has been shown to be related to individual differences in sensation seeking. Buchsbaum (1976; 1978), to whom Zuckerman refers, calls individuals in whom there is a decrease in evoked-potential (EP) amplitude with an increase of stimulus intensity "reducers." Individuals who respond to an increase in stimulus intensity with an increase in EP amplitude are "augmenters." Buchsbaum notes that reduction can be compared to Pavlovian transmarginal inhibition. Zuckerman (1979) also takes this position. According to the neo-Pavlovian theory, the weak NS is more prone to transmarginal inhibition, which is expressed in behavior by a decrease of reaction. The analogy between NS strength and augmentation (sensation seeking)–reduction (sensation avoidance) has certain weaknesses, however. Nebylitsyn (1972), who devised the "slope of motor reaction time" method (on which Buchsbaum's EEG method for measuring augmentation–reduction is based), using visual and auditory stimuli of different intensities, did not observe transmarginal inhibition in individuals with weak NSs. The lack of transmarginal inhibition in individuals with weak

NSs when the "slope of motor reaction time" was used to measure the strength of the NS has been confirmed in many other studies (see Strelau 1983).

It is possible that this divergence is due to the fact that strength of the nervous system has been diagnosed on the basis of motor responses, whereas in Buchsbaum's and Zuckerman's experiments sensory EP amplitudes were measured. Bazylevich (1974) used the amplitude of somatosensory EP to measure strength of NS, and her subjects with high somatosensory evoked potential (SEP) amplitudes (augmenters, hence sensation seekers) have weak nervous systems, whereas those with low SEP amplitudes (reducers or sensation avoiders) are said to have strong NSs. This is consistent with Nebylitsyn's (1972) data, and, again, inconsistent with Zuckerman's statement about the relation between sensation seeking and the strength of NS.

Furthermore, according to Zuckerman (1979b; target article), one of the most typical behaviors in high sensation seekers is a strongly developed orienting reflex. But here again, if there is to be any comparison between strength of NS and sensation seeking based on the orienting reflex, the relation is just the opposite to the one proposed by Zuckerman. As Nebylitsyn writes, individuals "with weak nervous systems should exhibit more expressive ORs" (1972, p. 69).

In general, one must state that there are very few empirical data on the relation between the two dimensions under consideration. Paisey and Mangan (1980), who factor analyzed the items of Eysenck's Personality Questionnaire (EPQ), Zuckerman's SSS, and Strelau's Strelau Temperament Inventory (STI) found sensation seeking to be an independent factor, unrelated to the NS traits (strength of excitation, strength of inhibition, and mobility of nervous processes) or to extraversion. Some data collected in our laboratory (Strelau 1983), however, do lend support to Zuckerman's hypothesis about the relation between sensation seeking and nervous system traits. Measuring strength of excitation in 171 subjects (men 16–20 years old) by means of the STI and sensation seeking with the SSS (Form IV), we were able to show that there is a clear-cut but not very large positive correlation between strength of excitation and the general scale ($r = 0.25$; $p < 0.01$), thrill and adventure seeking ($r = 0.36$; $p < 0.001$), and boredom susceptibility ($r = 0.25$; $p < 0.01$).

Probably the only conclusion to be drawn is that stronger empirical evidence is needed to illuminate the relationship between sensation seeking and the neo-Pavlovian strength-of-nervous-system constructs.

Finally, in studying Zuckerman's theory of sensation seeking as presented in his excellent monograph (1979b) and papers (1980; target article), I get the impression that we are contending with a dynamic concept that fluctuates under the pressure of facts. This allows us to expect that Marvin Zuckerman has not yet said his last words on the subject of sensation seeking.

Sensation seeking: Where is the meat in the stew?

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Ever since the decline and fall of drive theory, psychologists have been busily trying to rescue what bits and pieces they can. Alternatives to primary physiological needs as the basic energizers or guides of behavior are posited to work in ways very similar to the original model. The old ingredient that most frequently finds its way into a variety of new pots has been the energizing or "D" aspect of drive, now relabeled as activation or arousal. The discovery that motivated behavior is not dependent upon disturbances of physiological homeostasis also spawned a list of hypothesized innate tendencies or needs, of which those dealing with information processing remain perhaps the most prominent. These include exploratory drive, need for novelty, need for information, and Zuckerman's sensation-seeking (SS) motive.

In the last 20 years or so, SS has expanded so that it is now an explanatory or moderating construct for a large number of specific behaviors. This expansion has reached the point at which a very wide range of psychological and physiological factors is being subsumed. In this stew, the flavor of each new ingredient is both imparted to and diluted by all of the others. Hypotheses about the operation of SS in one particular situation must both incorporate and be adjusted to its other aspects. The target article is a fine example of this, with great numbers of relationships, some empirically supported and some hypothesized, being cooked up together. Unfortunately, I am not as sure as Zuckerman is that the ingredients are really compatible. In any case, it is difficult to tell exactly which lumps nourish scientific progress and which are spicy but perhaps not digestible.

One of the problems is the measurement of SS. The literature contains many studies that used different subscales, found relationships between the dependent variable and some (but not all) SS scores, or found a relation for male subjects but not for females. Consumers who are somewhat skeptical may not take this with as much equanimity as Zuckerman does. The substantive meaning of scale scores also needs to be explored. SS is related to a specific and quantifiable component of the external world. One would like to see answers to questions such as how much more sensation high scorers need, what characteristics of "sensation" might be differentially reinforcing, whether satiation with sensation is related to the SS trait, and the like. I am also fascinated by unanswered questions about the interaction of trait and state SS. What environmental circumstances, emotional states, cognitive processes, physiological conditions, and the like affect the degree to which SS is expressed in behavior? How are these factors modified by, and how do they in turn modify, the underlying personality factor?

The biological aspect of the paper raises similar issues. Here again, Zuckerman deals with a large number of specific phenomena, covering a range of brain systems, neurotransmitters, and related biological constructs from evoked potentials through drug reactions and general activity levels. The literature review leaves the reader with a vague idea that something has been demonstrated, but without a sure grasp of just what that something is. Fine distinctions – between responding impulsively in spite of anxiety rather than because of anxiety reduction, between sensitization to stimuli as opposed to sensitization only to stimuli associated with either positive or aversive reinforcement, and so on – add complications that are not consonant with the wide theoretical sweep being attempted. The degree to which MAO is associated with general arousal rather than with SS, and in fact the relationship between general arousal and SS, needs to be explained in much more detail and more convincingly than is done here. Zuckerman's claim that "activity level is a component of sensation seeking" does not seem adequately supported; the relationship might be just the reverse, or both may be components or correlates of some other factor, such as general energy level or responsiveness to environmental cues. The analogies between human and infrahuman subjects are likewise not obvious. Behavior that can just as easily be interpreted in terms of a fairly large number of other explanatory constructs (aggression, general activity, responsiveness to the environment, exploration, sociability, and so on) are at least by inference equated with SS in human beings. Although Zuckerman recognizes that these are merely "likely models for sensation-seeking behavior in humans," just how likely they are is debatable. Can the hypothesized biological substrate explain motivational changes in response to environmental conditions? If it cannot, then do we need to look for another underlying factor? Should parsimony lead us to look for alternative explanations of the trait as well?

This brings us to the general question of causal relationships. "Social influences" appear to be tacked on as an afterthought, and are comprised mostly of developmental factors. Some of the hypotheses are questionable (e.g. why don't only children have higher SS scores than firstborns in larger families, and why do older but not younger siblings produce sufficient stimulation?). The contemporary social and informational environments are not considered. The implication that trait SS is primarily inherited attempts to explain a very complex and as yet mysterious set of psychological–behavioral phenomena on the basis of an at least equally complex and mysterious set of biological ones. Complexity and mystery make for a savory stew, and SS qualifies; but the chef should avoid the temptation to throw *everything* into the pot before learning more about *each* ingredient.

What are sensation seekers seeking?

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Zuckerman has made a persuasive case for an explanation of sensation seeking in terms of certain biological processes. He has not, however, told us very clearly what the nature of the trait is that he is trying to explain. This commentary is intended to elucidate that question, and, more particularly, to examine the dimensionality of sensation seeking. On the basis of that examination, I propose that we should differentiate between at least two different aspects of sensation seeking: a search for intensive stimulation, and a search for stimulus information.

Let us start with Zuckerman's definition of sensation seeking, presented all too incidentally near the beginning of the section "Animal models," as "a trait defined by the need for varied, novel and complex sensations and experiences and the willingness to take physical and social risks for the sake of such experience" (quoted by Zuckerman from Zuckerman 1979b, p. 10). To what extent does this definition correspond in fact to what is measured by the scales of sensation seeking that Zuckerman has developed to study this trait?

If we refer to the scales (Zuckerman 1971) that Zuckerman has used in his work on this problem over the past decade, we find that the answer to this question is by no means straightforward, for it appears that there are actually four separate components to sensation seeking, as Zuckerman points out in the target article as well. Significantly, the items that entail physical risk taking (e.g. "I would like to try parachute jumping"; "I like to dive off the high board") load on the first factor, "thrill and adventure seeking" (TAS), whereas those that refer to a desire for varied, novel, and complex stimuli (e.g. "I get bored seeing the same old faces"; "I like to try foods I have never tasted before") load on the fourth factor, "boredom susceptibility" (BS).

Upon closer examination, the thrill seeking and risk taking involved in the behaviors referred to in the items defining the TAS dimension entail not so much a desire for "novel, complex, or varied" stimuli, as a heightening of the *intensity* of stimulation to a maximum – corresponding perhaps to what Berlyne (1960) has called the "arousal jag."

The second and third factors are somewhat less readily interpreted, but it does seem that one, "experience seeking" (ES), relates to a search for novelty, much as does the BS factor ("I like to listen to new and unusual kinds of music"; "I like to dress in unusual styles"). In contrast, the other, "disinhibition" (Dis) entails a search for intensity of stimulation ("I often like to get high"; "I like to date members of the opposite sex who are physically exciting").

Granted that, as Zuckerman asserts both in the target article and in the earlier paper (Zuckerman 1971), the four factors are not uncorrelated, and that a general factor can be extracted, there are good conceptual as well as empirical grounds for differentiating the sensation-seeking trait into at least two separable components: one entailing the seeking out of intense stimulation, the other a search for information-laden stimulus environments – or, conversely, an avoidance of monotonous, and thus boring, situations. Such a differentiation would be substantiated through the finding of differential patterns of correlations between each component and other variables. In the target article, Zuckerman actually presents a certain amount of evidence of this type. Such physiological variables as heart rate, cortical evoked potentials, and gonadal hormones are found to correlate primarily with the *disinhibition* scale – one of the two that, it is being argued, reflect the intense-stimulation aspect of sensation seeking. Indeed, Zuckerman specifically identifies the "augmenting" process that appears to be indexed by cortical evoked potentials with response to *high levels* of stimulation. MAO levels, on the other hand, appear to correlate (negatively) with experience seeking and monotony avoidance.

These differential patterns of correlation should caution us against treating sensation seeking as a unitary variable, as Zuckerman, throughout most of his analysis, is prone to do. In the process, a number of issues become obscured. One of these is the relationship between sensation seeking and tolerance for sensory deprivation. Zuckerman cites an earlier study from his laboratory pointing to such a relationship, but upon closer examination it becomes apparent that the relationship is primarily with tolerance for the confinement and immobility aspects of sensory deprivation (SD), rather than with sensory deprivation per se. The latter presumably relates above all to the need for varied or information-laden stimulation (see Jones 1966), suggesting that a separate analysis of relationships between SD tolerance and the different components of sensation seeking would be eminently worthwhile.

How would this reformulation of sensation seeking in terms of two separable components relate to Zuckerman's "Animal models?" As Zuckerman himself indicates, behavior in the open field, which is taken as a rough analogue, at the animal level, of human sensation seeking, is in fact a function of need for exploration and fear inhibition. These two seem closely akin to the two processes that I have tried to differentiate, and so again one may look for differential relations between behavior in the open field and other variables. Indeed, in keeping with this argument, proneness to explore unfamiliar environments seems to relate to early stress experience, mediated by the autonomic nervous system (see Thompson & Grusec 1970), whereas preference for environmental complexity is a function of the animal's early perceptual experience (Sackett 1965; 1972).

In sum, it does not seem farfetched to suggest that physiological mechanisms of the kind that Zuckerman has pointed to, relating to the maintenance of optimal levels of catecholamine systems activity, would play a different role in ensuring exposure to intense levels of stimulation than in mediating a search for stimulus variation. The same would apply, a fortiori, to the postulated genetic basis for these aspects of behavior. There is thus ample reason to attempt to refine the behavioral trait that is the subject of Zuckerman's penetrating analysis along the lines I have proposed.

Author's Response

Home from a perilous journey

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A comparative and biological approach to personality involves many disciplines ranging from behavior genetics

to multivariate trait research. Writing a target article like this one, to elicit commentaries from experts in all of the scientific areas discussed, is like undertaking a somewhat perilous journey through many foreign countries. I cannot claim expertise in many of these areas, and my knowledge of their history, customs, and language is acquired by scholarship rather than by direct experience, except in collaborative studies. Despite this lack of credentials my welcome has been generally warm, or at least tolerant and respectful, and specific criticisms have been constructive. General judgments have ranged from "noble" (**Clavier**) to "base" (**Mason**). Although one cannot deny some degree of affective response to these kinds of general judgments, they were not the goal of doing this article. The valuable parts of the experience have been the suggesting by commentators of new areas and references, the pointing up of weak areas in the model and theory, and the offering of new hypotheses derived from the model. Nothing better illustrates the heuristic value of theory than its capacity to generate testable hypotheses. To the extent that this has happened with commentators, and will happen again with readers of the entire BBS treatment, I consider it a success. If the results of the subsequent research support the model or do not invalidate it, so much the better.

The theoretical approach

The brain is too complex for the model. It is suggested by **Callaway, Clavier, Eterović & Ferchmin,** and **Redmond** that the model is oversimplified in relation to the complexity of the biological processes involved. **Suedfeld** accuses me of making broad generalizations that ignore the discrepancies in the data and, on the other hand, of making "fine distinctions . . . that are not consonant with the wide theoretical sweep being attempted"! **Callaway** and **Clark** argue that more experimental work is needed to delineate the information-processing and molecular biological processes, and **Sokolov** says that single-unit neural studies are particularly required. **Barrett** says that only a systems model can incorporate the complex data from different disciplines. I am particularly struck by **Panksepp & Siviy**'s comparison of neuroscience to "a disorganized heap of fine building materials." The analogy is even more applicable to experimental psychology, which has shied away from general theories of behavior for the last 25 years. Information-processing studies hold considerable promise for the study of intellectual abilities, but my guess is that studies of generalized sensitivities to positive or negative reinforcement, as proposed by Gray (1982), hold more promise for the process study of personality. [See also *BBS* multiple book review of Gray (1982) in *BBS* 5(3) 1982.]

I believe I have indicated the complexity of the data and difficulties in moving from one level of analysis to another in a number of places in the target article. As expected, many commentators pointed out the missing areas, particularly in the environmental shaping of the trait (**Baldwin, Strelau, Suedfeld**). The model is admittedly simple and undoubtedly too simple to accommodate the complexity of the data. What I have done is to build the model on the clearer findings and attempted to reconcile some opposing theories in an inclusive framework. I have tried to steer a route between the Scylla of excessive preoccupation with the specific details of conflicting data and the Charybdis of totally unwarranted generalizations with little regard for facts. Parsimony dictates that we begin with simple models before rejecting or amending them in favor of more complex ones.

The complexity of a system like the noradrenergic one (**Redmond**) does not exclude the possibility that the excitatory and inhibitory effects of the neurotransmitter at different sites function just as the coordinated excitation and inhibition of skeletal muscle groups in purposeful movement. If one neurotransmitter served opposing behavioral functions with no organizational pattern we could not find any relationship between functional levels of the bioamine and behavior or traits at the higher levels. Although the field is far from settling on specific functions for specific neurotransmitters, as indicated in this review, the situation is hardly total chaos and randomness either. Unless a vast amount of negative data is being secretly disposed of rather than published, the null hypothesis has not yet carried the field. On the other hand, the functions of many of the systems may be very broad ones such as "arousal," "selective attention," or "behavioral inhibition," rather than more specific ones like "reward," "punishment," or "fear."

Certainly, more than one neurotransmitter must be involved in each of the behavioral traits described (**Clavier**), and the model suggests that both dopamine and norepinephrine are so involved, with serotonin and endorphins often playing antagonistic roles in reactions to environmental stimulation (see Figure 6).

The implications of Figure 1 are that the correlative biological behavioral data and the model derived from these data will suggest more precise experiments to elucidate the processes producing the correlations. Most of these experiments will have to be conducted using nonhuman species for the reasons I indicated in the target article.

As **Barratt** suggests, systems models can be useful, particularly in indicating the interaction of processes at different levels and in specifying feedback relationships. A simple systems model is provided in Figure 6 (bottom), where the relationships among bioamines, neuroregulators, environmental stimulation, behavioral activity, and psychological processes (such as habituation and inhibition) are portrayed. The danger in systems models is that they may never go beyond the box and arrow stage. They are not explanations in themselves and only suggest a strategy of experimentation. The model in Figure 6 does suggest that novel, intense, and complex stimulation can stimulate catecholamine systems activity (CSA), and that certain neuroregulators are involved in the processes of habituation of orienting to stimuli and inhibition of activity in response to stimuli. The model suggests that we will find curvilinear relationships between CSA and behavior and emotions if we sample the range of CSA.

The logic of comparative study. The criteria for establishing equivalence of animal and human traits are questioned by Clark. I agree that mere similarity of behavioral expression does not establish equivalence, but I never said it did. The theory itself should dictate the kind of similarity we are looking for. Stock-car racing and skydiv-

ing are dissimilar activities on the surface, but the definition of sensation seeking as the need for unusual sensations and the willingness to take risks for them points up the basis for their equivalence in relation to sensation seeking. "Ordinary driving" is not related to sensation seeking, but there is a linear relationship between habitual driving speeds and sensation seeking (Zuckerman & Neeb 1980).

Similarity of behavior within a species is not sufficient to establish equivalence of underlying motivation or biological determinants, and the same applies in comparisons between species. We must determine what is reinforcing a given activity and what function it serves for the individual. The finding of a common biological correlate, like MAO, for what appears to be similar kinds of behavior in two species is not sufficient, for we still need to know whether the same function is mediated by the biologically related behavior in both species. Of course, the receiving of grooming in monkeys is a meaningless analogue for human sensation seeking (even though both are related to MAO) unless we inquire into the function of grooming in a colony. Usually it seems to be a good indication of dominance, and it serves affiliative and contact needs.

Deduction from correlation. Before establishing the equivalence of processes underlying similar behaviors through experimentation it is possible to establish the likelihood of common causation through empirical correlation. As **Neufeld** points out, two variables correlated with a third variable are not necessarily correlated with each other unless the two correlations are much higher than those usually found in behavioral biological variable correlations. For this reason, it is important to "triangulate" such correlations. For instance, sensation seeking is related to low platelet MAO levels and to certain phenomena such as mountain climbing, drug abuse, legal infractions, and sociability. We cannot simply assume that all of these phenomena are therefore related to low MAO, but research has established that they are so related. It is less important to triangulate two behavioral phenomena both of which are related to the sensation-seeking (SS) trait because the phenomena may represent alternative forms of expression of the underlying trait. It is unlikely that mountain climbers typically use drugs or break laws even if low MAO levels and SS trait are common to these activities.

Another valuable kind of triangulation involves SS and two related biological variables. This is important because it provides some insight into the structure of the biological model. As an example we have the negative correlation between serum DBH (dopamine-beta-hydroxylase) and augmenting of the cortical evoked potential (EP) (von Knorring & Perris 1981). The next step is obvious. Does inhibition of DBH (or NE) lead to augmenting of the EP? Correlation does not demonstrate causation, but within the context of a model it may point to the crucial experiments that will show the antecedent–consequent relationship.

Neufeld suggests that factor analysis is useful in understanding the interrelationships of trait and biological variables. I agree, but with a caution: Such analyses can be misleading; two variables with a zero correlation may load on a common factor because of their moderate relationships with a third variable. The fact that they load on a common factor might mislead some to assume a relationship where there is none. The common practice of not presenting the original correlation matrices along with the factor matrices fosters this sort of illusion. A factor matrix can hide many signs of omission.

Callaway points out that simple bivariate correlations only account for about 16% of the variance in relationships between traits and biological variables, but given the likelihood of multivariate biological determination and the limited reliabilities of these variables one could not expect higher relationships looking at only two variables at a time. A dependence on imprecise statistical decision rules is one of the facts of life in experimental *or* correlational studies of living organisms where variability is the rule (Zuckerman 1979c).

Animal models. I had expressed reservation about the open-field model for sensation seeking, because activity in the usual open-field test is a function of two traits, emotionality and explorativeness, and only the latter is relevant to sensation seeking. **Eysenck** has pointed out that the use of dimmer lighting conditions, as done by the Barcelona group (Garcia-Seville 1984), may be sufficient to eliminate emotionality as an influencing trait. However, a second reason is that this model does not incorporate the social aspects of human sensation seeking. Earlier cited work by McClearn (1959) showed consistency of behavior in strains of mice in an open-field area, hole in the wall, and barrier situation. However, **Simmel**'s studies would indicate that responsiveness for what he calls "novel" stimuli is not correlated with activity, and the BALB strain, which is generally found to be low in activity in the open field, is actually high in response to "novelty" in the shuttle box. Simmel's stimulus reactivity factor is more of a response to complexity, however, since one of the two measures of the factor – portion of time spent in the "novel" compartment – would indicate a perceptual preference for the complexly painted compartment *in spite of* inevitable habituation to it. Crossings between the two sides, which would seem to be a better indication of need for novelty or change, loaded on both the stimulus reactivity *and* activity factors.

Simmel's results on the rankings of strains for activity in different situations is consistent with other data showing reversals of strain differences or of differentially lesioned groups (Ellison 1977) when seminatural cage or colony situations are compared with the open-field one. It would be interesting to see the effects of neurotransmitter lesioning on the responses in Simmel's two-compartment situation. In Gray's (1982) review of the literature on the effects of lesioning of the dorsal noradrenergic bundle on tests of exploration and activity (see table 11.4 in that book) he notes that rearing in the open field, spontaneous alternation in the T maze, and activity in the hole-board test are all decreased by the lesions, but there is little evidence of effects on activity in the open field or activity in cages or boxes. A better understanding of the differences between these experimental tests is needed before we can fix on any or several of them as models for sensation seeking. For this reason I am inclined to assign greater importance to the results using reliable social behavior observed in animal colonies (e.g. Ellison 1977; Redmond, Murphy & Baulu 1979).

Sokolov criticizes the lack of distinction between responses to novelty and the "tendency to seek specific attractants." I am not sure what the latter phrase refers to, but if it means specific positive reinforcers like sex and food, I would maintain that novelty and variation are an important determinant of activity directed toward these "attractants" as well as toward simpler kinds of stimuli. Humans, like rats and cats, consume more foods when presented with a variety of them (Rolls, Rowe, Rolls, Kingston, Megson & Gunary 1981). Monkeys (and probably humans as well) show increased sexual behavior when stimulated by novel sexual partners (Michael & Zumpe 1978).

Claridge would have us concentrate on psychophysiological models of the nervous system, derived from human research, rather than neurochemical ones which are heavily dependent on comparative studies. The problem is that surface electrodes can tell us little about what is happening in the limbic system where much of the action relating to emotion and motivation seems to be occurring. The basic construct of psychophysiology, "arousal," seems to have fallen on hard times (Zuckerman & Como 1983) because of the evidence that at least several arousal systems interact in complex ways. One of these seems to be the dorsal ascending noradrenergic bundle which seems to function as a cortical arousal system. Until we can find an ethical way to measure the activity of this system using surface electrodes or scans we must rely on neurochemical measurements. I believe that the chemistry of the brain is more central to an understanding of psychological processes like "anxiety," "arousal," or "reward" than peripheral measures like palmar skin conductance or heart rate. EEG measures, like the EP, are more useful in understanding brain processes, but measures like these are relatively nonspecific in neural system origins. However, the work of Stelmack, Plouffe, and Falkenberg (1983) on early components of the EP and peripheral sensory-motor reflex arcs, studied by Stelmack and Plouffe (1983), are relatively specific in their neuronal location. The amazing thing about their findings is the consistency of the relationships between neural arousability and extraversion at all levels of the nervous system. I do not believe it as essential to use single-unit studies as **Sokolov** does. Although such studies will be useful in animals they are obviously not feasible in humans.

Although psychophysiological methods have been useful in understanding information processing, they have been less successful (with the exception of facial myography) in distinguishing emotional reactions. I agree with Zajonc (1980) that "preferences need no inferences," or that emotional and noncognitive reactions precede the cognitive ones in most human interactions. I also agree with **Izard**'s view that personality differences evolve from early differences in "emotional expression" styles. A good example of this is the difference in emotional and behavioral reactivity in newborns with high and low MAO levels (Sostek, Sostek, Murphy, Martin & Born 1981). A longitudinal study of these infants would be illuminating. I have neglected the developmental aspect of sensation seeking, and Izard has neglected the biochemical determinants of emotions.

Traits and factors. The trait construct of sensation seeking is called into question by **Baldwin** because there is as yet little evidence about environmental influences, which may be very important despite high heritability estimates for the trait. I do not understand this argument. A trait may be primarily determined genetically or environmentally or by some complex interaction of heredity and environment. The existence of a sensation-seeking trait depends upon the demonstration (1) of consistent self-reports of experience, attitudes, and desires across a diversity of situations (in a recently developed version of the SSS, Zuckerman 1984, I have separated reports of actual experience from those of intention in separate scales, and both types show high internal reliability) and (2) that the scales composed from the consistencies among self-reports do correlate with actual behavior or preferences, or do relate to other external criteria predicted from the theory. The first demonstration is reliability and the second is validity. If we limit the discussion to sensation-seeking *behaviors* without the underlying construct of a "trait," we have no theory, and we fail to search for the common aspects of the varied behaviors, whether cognitive or biological, that provide an understanding of the motive underlying the trait.

Barratt says that I have not devoted attention to the interaction of sensation seeking with other traits, such as anxiety, in the determination of behavior. In fact, I have developed a model suggesting how the traits of sensation seeking and anxiety interact and, through their influence on affective states, influence the seeking or avoidance of risky situations (Zuckerman 1979a; 1979b). Both sensation seeking and anxiety have been shown to have *independent* influences on the magnitude of the electrodermal orienting response (OR) (Neary & Zuckerman 1976) and reactions to phobic situations (Mellstrom, Cicala & Zuckerman 1976).

Redmond also points out the likelihood of influence by more than one trait in both human and animal models. Since the trait approach is relatively new in animal behavior, there has been little broad-range sampling of behavior on a variety of measures using the same animals in different experimental situations. One such study (Garcia-Sevilla 1984), measuring 15 variables from nine experimental tests, found five definable factors: emotional reactivity, movement, stimulus seeking, exploration, and activity. Some variables had high and relatively specific loadings on only one factor whereas others loaded about equally on several factors. This type of multivariate analysis seems to me to be the only way to answer the question of which behavioral traits are assessed by responses in specific experimental paradigms. For instance, "rearing" responses in the open field or similar tests are interpreted by **Gray** and **Redmond** as "fear" reactions whereas I interpret them as orienting responses related positively to reactions to novelty and, by inference and OR data, to a sensation-seeking trait. Data from a study by Gomà reported in Tobeña, Garcia, and Garau (1982) showed a positive correlation ($r = .55$, $p < .01$) between rearing and open-field ambulation, supporting my interpretation of "rearing" as "sensation seeking" rather than "fear," since fear would inhibit rather than facilitate ambulation in the open field.

Claridge, Eysenck, Neufeld, and **Royce** have all questioned how sensation seeking fits into the Eysenckian three-factor system and whether sensation seeking is a

fundamental dimension of personality or an amalgam of other factors. Eysenck questions whether the subfactors comprising the total score of form V of the SS scale are really components of a general SS trait or separately related to broader dimensions such as E (extraversion), N (neuroticism), and P (psychoticism). **Wohlwill** has suggested that two dependent factors may be measured by the subscales, one related to a need for intense stimuli and the other to the need for novelty.

Eysenck's reference to the subscales sharing only about 10% of the variance refers to form V where subscales were shortened and an effort was made to select more specifically loading items. In form IV the correlations between subscales ranged from .3 to .6, with .4 being about median (Zuckerman 1979b, Table 4.7, p. 113). This means that 16% of the *reliable* variable is accounted for by the typical intercorrelation between scales, which is about as high as one might expect for component scales with some specific and some group factor variance. The fact remains that the internal reliability of the 40 items comprising the total score on form V is .85. If the score were based on items from unrelated factors one could not find this kind of consistency in response to all items. Unpublished factor analyses that I have done on the four subscales together with the Eysenck's P, E, N, and impulsivity scales have yielded a separate sensation-seeking–risk-taking factor with secondary loadings from the E scale in males and the P scale in females (see Table 1). These data are from the twin study cited by Eysenck (1983). If one examines the correlations, it is apparent that none of the SS subscales correlates more than .30 with any of the Eysenck dimensions, and only the TAS (thrill and adventure seeking) scale shows any degree of correlation with E. There is no instance of a subscale of the SSS correlating more highly with E, N, or P than with at least one of the other SS subscales. A broader range of studies cited in Zuckerman (1979b, pp. 142–48) show that in general SS correlates about .3 with E and P and zero with N. This means that, taken together, E and P account for about 18% of the variance of sensation seeking. This is far from supporting **Royce**'s attribution to me that "sensation seeking is a function of Eysenck's extraversion and psychoticism dimensions."

At a recent symposium in London, **Gray** (1983a) suggested that we use **Eysenck**'s factor-defined dimensions as the standards to identify all new dimensions. The problem with this proposal is that the E dimension has changed, becoming almost entirely a sociability factor, and there is still considerable debate about conceptual aspects, the reliability, and the validity of the P dimension. There is no reason why personality structure must be defined in a hierarchical fashion, as in **Royce**'s model, rather than as a circumplex in which all definable axes within a two-dimensional framework are equal in status. In this sense, we may say that sensation seeking lies between the dimensions of E and P, or that E lies

Table 1. *Factor analyses of SS subscales, Eysenck personality questionnaire, and impulsivity scales*

			Varimax rotation			Oblique rotation		
			Factor loadings			Factor loadings		
Sex		Scales[c]	I	II	III	I	II	III
Males[a]	SSS	Dis	*.61*	.37	.09	*.67*	−.25	−.10
		TAS	*.58*	−.09	.12	*.63*	.21	−.03
		ES	*.40*	.04	.35	*.30*	.08	.31
		BS	*.36*	.30	.23	*.31*	−.20	.14
	EPQ	P	.23	*.50*	.26	.16	*−.43*	.21
		E	.39	.00	.05	*.44*	.08	−.06
		N	−.16	*.64*	−.11	−.13	−.69	−.13
		L	−.30	−.30	−.03	−.35	.24	.08
	Imp	Imp	.12	*.56*	.25	.03	*−.51*	.24
		Risk	*.61*	.32	.27	*.60*	−.17	.12
		Non-Plan	.15	.15	*.87*	−.22	−.01	*1.00*
Females[b]	SSS	Dis	*.71*	.19	−.10	*.83*	−.18	−.14
		TAS	*.58*	.08	.22	*.68*	.15	−.15
		ES	*.61*	.12	.03	*.73*	−.03	−.16
		BS	*.49*	.21	−.03	.52	−.07	.02
	EPQ	P	*.37*	.39	−.13	.27	−.14	*.30*
		E	.18	.27	*.35*	−.03	*.36*	*.34*
		N	.07	.13	*−.61*	−.10	−.63	.01
		L	−.39	−.21	.15	−.38	.18	−.06
	Imp	Imp	.14	*.82*	−.10	−.29	−.03	*1.00*
		Risk	*.61*	.23	.18	*.64*	.13	.03
		Non-Plan	.31	*.45*	.20	−.12	.22	*.48*

[a] $n = 222$. [b] $n = 625$. [c] SSS = Sensation Seeking Scale; TAS = Thrill and Adventure Seeking; ES = Experience Seeking; BS = Boredom Susceptibility; EPQ = Eysenck personality Questionnaire; P = Psychoticism; E = Extraversion; N = Neuroticism; L = Lie; Imp = Impulsivity; Risk = Risk taking; Non-Plan = Non-planning.

between the dimensions of SS and sociability, rather than that sociability, impulsivity, and sensation seeking are component traits of E.

From a biological viewpoint the most "basic" dimensions will be those best representing the genotypes and the intervening biological dimensions (Gray 1983b; Zuckerman, Ballenger & Post, 1984). **Eysenck** claims that by raising the question of which are the primary orthogonal, "basic" dimensions of personality, **Gray** and I are construing personality dimensions as realities rather than mathematical abstractions. What we are really suggesting is that the organizing of personality at the behavioral trait level is probably a function of the organization of relevant factors at the biological level. The alignment of factors at both levels will come from factor analyses of both kinds of data, not from factor analyses of psychological tests alone. As described in this article and elsewhere (Zuckerman et al., 1984), such studies seem to show that Gray is right in suggesting that the dimension that runs from stable extraversion to neurotic introversion is of greater biological significance than the one that runs from stable extraversion to stable introversion.

A comparison of the biological correlates of E and SS (Zuckerman 1983d) shows that common biological correlates of both, like testosterone and MAO, could account for their similarities. A recent manuscript by Schalling, Åsberg, and Edman (1984) shows that P and SS (monotony avoidance) share similar correlations with CSF metabolites of serotonin (5-HIAA) and dopamine (HVA). These kinds of findings suggest that we should avoid theoretical imperialism and attempt to discover the differences *and* similarities among the psychological dimensions in their biological substructures.

One problem with **Royce**'s factor model is that it seems to combine behavioral constructs that are appropriate to rodents (i.e. "territoriality") with more humanlike traits (i.e., "tender-mindedness," "guilt," "trust") at the same level of analysis. It is certainly conceivable that a construct like "territoriality" may represent something like "dominance," "aggressiveness," or "autonomy" at the human level. Unless fairly precise operational definitions are provided for trait terms and their relationships to other phenomena are clearly understood, this kind of "metatheory" is little more than word play. Such semantic classification is not likely to contribute to our understanding of personality organization. Elaborate factor structure outlines without a sufficient base in research definitions are of little heuristic value.

Baldwin and **Suedfeld** say that I have concentrated on trait explanations and ignored the motivational influences of situation and cognition. I must plead guilty to this charge, although my analysis of risk-taking behavior (Zuckerman 1979a) does suggest the directions such research could take and presents some data on anticipated responses to hypothetical situations varying in risk.

Optimal level of arousal versus optimal level of CSA. The finding that sensation seekers use both stimulant and depressive drugs does not, **Eysenck** argues, weigh against the optimal level of arousal theory, since arousal may be produced by a change in mood states and internal responses. I think that Eysenck is speaking of something other than cortical "arousal"; otherwise his statement that lowering arousal is a change that may produce higher "arousal" would be illogical. It is true that the subjective effects of lowering arousal may be reported as "getting high," and depressant drugs may increase behavioral arousal through disinhibition of limbic centers. However, these are all dependent effects; the theory says that the motivation of behavioral activity is to reach an optimal level of arousal and that extraverts are chronically below this level. Why either extraverts or sensation seekers use depressant drugs is not answered by optimal level of arousal theory unless arousal is redefined in nonphysiological terms.

Von Knorring says that the Carrol, Zuckerman, and Vogel (1982) study, which failed to show a differential effect of d-amphetamine and diazepam on high and low sensation seekers, could also be used against an optimal level of CSA theory. To some extent this is true since d-amphetamine stimulates activity in both catecholaminergic systems. However, this would be based on the assumption that the dosage used for all subjects would bring high sensation seekers closer to optimal CSA activity and "push" low sensation seekers well over the optimal level into the range of anxiety (E or F in Figure 6). In actual fact the changes in both groups may have been within the B to D range (Figure 6) which would produce equal effects on mood and performance in high and low sensation seekers, even assuming the highs started at a lower level. An adequate test would require higher doses of the drug. Of course, the optimal arousal theory might be defended from the results of the experiment on the same basis. The experiment bears repeating with a higher range of drug dosage and concomitant EEG recordings to measure changes in cortical arousal.

Clavier claims that an optimal level of CSA is just another version of the optimal level of arousal theory since CSA is the same as cortical arousal and adaptability is simply "cue functioning" in the Hebb model (1955). The Hebb model of "arousal" depends on the activity of the reticulocortical activating system, whereas the CSA model has a different biological substrate in the dorsal ascending noradrenergic bundle and the dopaminergic system. Since the former has been shown to have an arousal function it could be argued that the models are functionally similar even if the biological substrates are different. However, cortical arousal and behavioral activity are *not* the causal or independent variables in the current model but are epiphenomena of CSA. The point of activities that increase CSA up to an optimum level is rarely the increased arousal. Amphetamine and cocaine may of course be used to increase "cue function" through increased arousal, but the more general motive for their use seems to be the positive mood effects suggesting the stimulation of an intrinsic reward mechanism in some CS centers.

Yes, any optimal level theory is "tricky," as **Panksepp & Siviy** point out. However, the basic requirements for crucial experiments are clear. We need: (1) an independent definition of groups assumed to differ on optimal level "set points"; (2) two or more drug dosages, treatments, or stimulus intensities over a range broad enough to encompass the optimal level curves of both groups; (3) a measure of the biological substrate, CSA in the case of the current model and electrocortical or reticular activity in the case of the Hebb and Eysenck models; (4) appropriate

measures of both mood and performance before and after treatments.

Regulatory or balance models. It is suggested by **Claridge** that a regulatory or balance model of the type devised by him (Claridge 1967) or by Haier, Buchsbaum, Murphy, Gottesman, and Coursey (1980) might be more suitable for sensation seeking than the optimal level ones. The Pavlovian concept of a dimension of equilibrium or balance between excitatory and inhibitory processes was superseded by the strength of the nervous system concept (equivalent to the strength of the excitatory process) in Nebylitsen's (1972) neo-Pavlovian theory, and by the optimal level of stimulation and arousal in Eysenck's (1967) revised theory of extraversion. One reason for the shift in concepts was that most direct measures can only assess the net difference between excitatory and inhibitory processes, which cannot be defined independently. Identification of special excitatory and inhibitory areas of the brain and direct neuronal study of these areas might yield independent measures, but such methods have only recently been used by psychologists. A theory like Claridge's provides an interesting way to conceptualize psychopathology, but it does not tell us much about what underlies normal variations in personality. The Haier et al. (1980) model uses the balance between a psychophysiological dimension (augmenting–reducing of the EP) and a biochemical one (levels of MAO). It seems confusing to mix levels in the model. I would suggest that some of the biochemicals, such as serotonin and dopamine-beta-hydroxylase, which have been implicated in augmenting–reducing (see **von Knorring**'s commentary) should constitute one dimension of the model and the excitatory catecholamine systems the other. This kind of regulatory model is suggested at the bottom part of Figure 6. I also suggest in the target article that low levels of MAO may allow the monoamines to fluctuate widely in stress conditions and that this may be the explanation for the risk of psychopathology in low MAO groups.

Strength of the nervous system. The disinhibition type of sensation seeking is related to the strength of the nervous system dimension of personality, because of the EP augmenting–reducing findings; **Gray** agrees. However, **Strelau,** a prominent neo-Pavlovian theorist whose recent book (Strelau 1984) provides the latest account of the relationship between Pavlovian and western concepts of personality, is not so sure of the connection between sensation seeking and strength of the nervous system.

Part of the problem is the shifting definitions of the strength of the nervous system concept, as described by both Gray (1964) and Teplov (1964), and its relation to behavioral and psychophysiological phenomena. According to classical theory, the weakness or strength of the nervous system referred to the degree to which cortical neurons were "exhausted" by intense or repetitious stimulation. The weak nervous system is said to be more sensitive to stimulation at low intensities but shows inhibition of cortical reactivity at high intensities; the reverse is true for the strong nervous system. Various operational definitions have been provided, including questionnaires, psychophysiological and psychophysical measures, and behavioral indices like conditioning and reaction-time measures. Not unexpectedly, many of these measures are not highly related to each other.

Strelau reports that sensation seeking is positively related to his questionnaire measure of "strength of excitation" (synonymous with strength of the nervous system), but Goldman, Kohn, and Hunt (1983) found a positive correlation between sensation seeking and their questionnaire measure of reducing, the reducer–augmenter scale (RAS). The latter finding cannot be taken too seriously since the RAS was developed around Petrie's (1967) theory, and the items were written with the assumption that reducers were extraverted sensation seekers. According to Goldman et al. (1983), the RAS measure has never been related to cortical EP augmenting–reducing, and in the one study done, no correlation was found between Petrie's kinesthetic figural aftereffect (KFA) method and the RAS. The KFA is a psychophysical method described in the target article. **Haier** points out that the theory developed by Petrie would lead to the prediction that sensation seekers would be reducers rather than augmenters. Apart from the fact that the KFA is a method with no reliability (Morgan & Hilgarde 1972) and the RAS has no validity in terms of correlation with the KFA or the psychophysiological EP measure, the terms "augmenting" and "reducing" may have opposite meanings in the KFA and EP methods. This is precisely what Davis, Cowles, and Kohn (1983) suggest: "AER [auditory evoked response] reducing is associated with KFA augmenting and vice versa" (p. 494). They suggest that the KFA measures the sensitivity end of the strength dimension, whereas the EP measures the susceptibility to transmarginal inhibition at high stimulus intensities. They cite evidence (Goldman et al. 1983; Kish, Frankel, Masters & Berry 1976) that sensation seeking correlates positively with absolute auditory thresholds; that is, low sensation seekers had lower thresholds and therefore were more sensitive. The RAS itself did not correlate with auditory thresholds in the Goldman et al. study. To summarize briefly: The problem is partly semantic and partly the failure of convergence of putative operational measures of augmenting–reducing and strength of the nervous system.

In view of the evidence linking the Buchsbaum EP method to behavioral, biological, and psychopathological phenomena (Zuckerman, Buchsbaum & Murphy 1980) I would argue that this method provides the most direct, reliable, and valid measure of strength of the nervous system. Reaction time and psychophysical measures like pain tolerance, sensory thresholds, and the KFA are vulnerable to behavioral and cognitive sets that limit their reliability and validity as measures of cortical activity. It must be emphasized that Pavlovian theory concerns cortical functioning and the EP measure is the most direct way to assess this. As **Gray** suggests, the EEG photic driving measure constitutes another method that might be explored. Another potentially useful method might be to study sensitivity in terms of the minimal intensity of stimulation able to elicit an EP.

The findings and their implications

EP augmenting and sensation seeking. The claim that EP augmenting–reducing is a reliable measure of a cortical

reactivity trait is questioned by **Callaway** on two grounds: (1) it is modality specific, with no relation found between visual and auditory augmenting–reducing; (2) it is affected by task and environmental factors and therefore depends on the "strategy subjects select."

Callaway cites the study by Raine, Mitchell, and Venables (1983) showing little reducing in the auditory mode and no correlation between the visual and auditory measures. Como (1984; preliminary data reported in Zuckerman & Como 1983) assessed augmenting–reducing in visual and auditory modalities using first a long interstimulus interval (ISI) of 17 seconds and then a short ISI of 2 seconds (the same ISI Raine et al. used). Like Raine et al., we found little reducing in response to auditory stimuli at the 2-second ISI, but reducing was apparent in many subjects with the 17-second ISI. The individual slope measures of augmenting–reducing for visual and auditory stimulation correlated significantly using the long ISI, but not using the short ISI. Our data do not contradict the findings of Raines et al. since they used the short, 2-second ISI. Perhaps a longer recovery period is necessary for auditory evoked potentials to elicit the individual variations in augmenting–reducing.

Buchsbaum (1976) has reported three situational factors that can affect the EP measure, although the effects may have little to do with the subject's "selection of strategies," given the fact that the analyzed EP component only follows the stimulus by about 100 milliseconds. The first effect is an across-sessions lowering of "arousal" for EPs at all intensities of stimulation. Although Buchsbaum claims that all intensities are equally affected and therefore the slope should not be affected, Birchall and Claridge (1979) have reported that individual variations in slope across sessions are related to changes in skin conductance level (arousal) across occasions. Despite the evidence for some individual variation in slopes, the correlation between first and second occasions was .77 and the coefficient of concordance for all four occasions was .66. These reliability figures are comparable to those obtained by Buchsbaum over longer intervals of time.

The standard EP procedure does not encourage "attention" to the stimuli but simply a passive attitude. Buchsbaum (1976) reports that an "attention" enhancing set increases EP amplitude for low-intensity stimuli relatively more than for high-intensity stimuli and therefore changes the slopes in the direction of reducing. Sensory overload, in the form of loud background noise, operates mainly to reduce EP amplitudes at high intensities thereby producing more reducing slopes. These are all interesting effects of environmental variables on the slope measure, but what do they have to do with the standard procedure for obtaining the measure which does not encourage close attention to the stimuli and is done in quiet, soundproof chambers? Conceivably, some subjects might pay closer attention to the light flashes than others for obscure reasons, but how long would they maintain this attention over 800 to 1,000 stimulus presentations? Except for changes in intensity there is little stimulus novelty in an EP experiment. Even with variations in arousal, which may affect slope measures, there is still a level of reliability in the EP technique that is comparable to most tests of personality traits.

Buchsbaum's (1971) original hypothesis of a positive relationship between sensation seeking and augmenting has been supported in six studies cited in the target article. Consistent with the direction of these findings, augmenting is correlated with the related trait of impulsivity (Barratt & Patton 1983) and is found to be characteristic of high sensation-seeking groups like sociopaths, delinquents, drug users, and persons disposed toward hypomanic states. It was therefore rather disconcerting to read **Haier**'s report of finding a negative relationships between sensation seeking and EP augmenting.

On closer examination it would appear that the Haier, Robinson, Braden, and William's (1984) study reflects a simple failure of replication, not a finding in a reverse direction. Haier et al. screened a large number of subjects using Buchsbaum's EP procedure and then recalled the highest and lowest 10% on the distribution of augmenting–reducing to give them psychological tests, including the SSS. They ended by comparing the SSS scores of 11 extreme augmenters and 10 extreme reducers. Although the selection on the basis of the EP variable rather than the SS one could yield different findings, von Knorring (1981) also did his comparison this way and found significant differences in the previously reported direction. What is more bothersome is the fact that the augmenter group consisted of 10 males and one female, whereas the reducer group had five subjects of each sex. Most of the findings relating EP augmenting to disinhibition have used males. Females typically show greater augmenting but score lower than males on disinhibition. Although Haier used SSS standard scores in the comparisons, the sex imbalance is still undesirable and could affect the findings in other ways.

When Haier (personal communication) correlated augmenting with sensation seeking over the entire range of scores (28 males and 85 females) within each sex separately, he found only one significant correlation out of eight; the TAS subscale correlated $-.37$ with augmenting in the male group. It is true that all correlations were negative, but with the exception of the one that was significant all were less than $-.30$. The correlations with disinhibition were very close to zero in both sexes. Thus, what appears to be a reversal of findings in the inappropriate comparisons turns out to be a simple failure to replicate the disaugmenting relationship. The presentation of the topographic mapping data from a second study is quite premature, considering that the differences are not significant.

Is there an explanation for the failure to replicate other than the usual variations of means in small samples? **Haier** has pointed to one possible factor: range of stimulus intensity. Following Buchsbaum's procedure, Haier used only the four lower intensities of the stimulator. In the experiments that found a relationship between augmenting and sensation seeking the higher intensity was used. Lukas (1982) actually used a Maxwellian-view optical system which ensures that the light beam passes directly into the pupil. Others have used diffusion screens that attenuated the light, but broadened the area of stimulation. I would argue that it is essential to use the highest intensities of stimulation (consistent with ethical considerations) if one hopes to find an augmenting–reducing effect that is related to personality. In most studies the differences between augmenter and reducer groups is primarily found at the highest intensity of stimulation. If we had not used the highest intensity of

light in the Zuckerman, Murtaugh, and Siegel (1974) study, high and low disinhibitors would not have been differentiated. If "augmenters . . . become reducers" at high intensities *in the range used in experiments*, then they were "reducers" and not "augmenters" to begin with.

Orienting and sensation seeking. As noted in the target article, we have been discouraged about the lack of replication for the electrodermal OR findings of Neary and Zuckerman (1976) and have turned to heart-rate change as a measure of OR and DR (defensive reflex) or SR (startle reflex). **Stelmack** has persisted in studying the electrodermal OR in relation to sensation seeking and Eysenck's three dimensions of personality (Stelmack, Plouffe & Falkenberg 1983). Stelmack et al. subtitled their article "Probing a Paradox." Their "paradox" is the fact that the electrodermal OR has been positively related to sensation seeking and negatively related to extraversion in previous studies. To begin with, this is not necessarily a paradox given the low order of correlation between extraversion and sensation seeking. As **Neufeld** points out, two variables related to each other at a low level of correlation are not necessarily both related to a third variable (even in the same direction). The results of Stelmack and his colleagues do little to resolve the "paradox." The initial findings were that high sensation seekers (and high P scorers) responded with greater initial SCRs to pictures of words and weaker SCRs to pictures of things than low sensation seekers. The results with words were in the direction found in Neary and Zuckerman (1976), but the opposite effect for pictures creates a new paradox. Neary and Zuckerman only found results for males and not for females. The correlations between initial SCR and sensation-seeking scales were unusually high for males in the Stelmack et al. study, ranging from .58 to .63. The correlations in the picture condition were significant but lower (−.23 to −.39). None of the correlations was significant for females. Despite the strong findings for males in the word condition, the authors removed the effect by partialing out SCL (skin conductance levels) from the SCR–SSS relationship. I believe this is a dubious correction procedure, rarely applied to OR data in other studies. Sensation seeking was not related to conductance in other studies, and the relationship between conductance and SCR is much stronger in the word condition than in the picture condition in this study. Correcting for what may be a random and meaningless fluctuation of conductance has distorted the actual results. In my opinion, the paradox was not a paradox to begin with, but may have represented a real difference in the biological bases of sensation seeking and extraversion. Stelmack's results involving an interaction with type of stimuli simply create a new paradox calling for new explanations.

I did not say that the heart-rate acceleration of low disinhibitors represented a pure DR as implied by **Stelmack.** What I said was that these rapidly habituating differences represent "DRs or SRs, or some mixture of these responses." I am not sure that these distinctions from Pavlov and **Sokolov** fully exhaust the possibilities for classification of physiological responses to stimulation. The heart-rate acceleration to the exposure of a novel stimulus of moderate intensity suggests an interaction between the novelty and intensity aspects of the stimulation. I believe that this could be a physiological expression of neophobia in low sensation seekers.

Stelmack's suggestion that low sensation seekers are more sensitive to stimulation does not really explain the OR and EP augmenting–reducing data. There is, of course, no reliable way to distinguish the subjective intensity from that indicated by the responses. However, sensitivity differences should have their major effects at the low end of the intensity continuum, and both the heart rate and EP results occur toward or at the high end of the auditory and visual intensities used in experiments.

Intensity and/or novelty. It is correctly pointed out by **Wohlwill** that the phenomenal definition of the SS trait does not include intensity of stimulation but only novelty and complexity. The definition was formulated largely on the basis of the item content of the SSS before the full import of the research on augmenting–reducing of the EP and the heart-rate OR, DR, and SR was considered. Even recent research on phenomenal correlates suggests that the role of intensity may be greater than was supposed. A study by Litle (1984) shows that sensation seekers show a liking for all types of rock music where intensity seems a more salient factor than novelty or complexity. This is consistent with the greater sensitivity of low sensation seekers to auditory stimuli (Goldman et al. 1983) and the greater tolerance for sound intensity (Zuckerman 1979b, pp. 220–21) in the high sensation seekers. The research on the heart-rate OR–DR suggests an interaction of novelty and intensity in producing differences between high and low sensation seekers, because the differences only appear on the first or second presentation of a moderately intense tone. However, Wohlwill's suggestion that the TAS and Dis scales reflect the need for intensity of stimulation whereas the ES (experience seeking) and BS (boredom susceptibility) are based on the need for novelty does not fit the pattern of interscale correlations. TAS and Dis are less correlated than is Dis with ES and BS. A better case might be made for Dis in stimulus-intensity regulation since it is the scale related to augmenting–reducing.

Is sensation seeking a drive? It is suggested by **Katz** that the data from rats show episodic sensation seeking rather than drive or satiety effects that would characterize a need for novelty comparable to other basic appetitive needs. The work of Jones (1969) on the sensation-seeking behavior of humans in sensory-deprivation experiments does demonstrate drive (duration of stimulus deprivation) and satiety effects and shows that these effects are primarily related to the novelty (unpredictability in stimulus sequences) and secondarily to the complexity of stimuli. Katz's and **Sokolov**'s analyses pertain more specifically to appetitive behaviors, such as sex, that involve novelty, intensity, habituation, and the innate drive and satiety effects connected with the specific appetite. Katz suggests that such appetites for sensation "feed" on themselves, becoming more frequent, rather than less, following previous episodes. Such a positive feedback effect, when it occurs, may be due simply to reinforcement and disinhibition, but I question whether such behavior is typical. After a sport-parachute jump, does the parachutist typically want to go right up and jump again?

Katz's hypothesis does suggest a longitudinal study of the episodic timing of sensation seeking in human lives.

MAO and sensation seeking. Inquiring about the basis for the relation between MAO and sensation seeking, **Barratt** and **Suedfeld** point out how little is known about the role of this enzyme or the monoamine neurotransmitters it regulates in related behavioral processes. Of course such questions would never have been raised if the raw empirical correlations were not reported. At present we have only theoretical speculations on the intervening biological and psychological processes in humans. There is the model proposed by Haier et al. (1980) that suggests a role for MAO in regulation of stimulus input. In this model augmenting–reducing represents the dimension of arousability and inhibition, while MAO provides protection against overload at the high end, presumably because of its capacity to regulate levels of the neurotransmitters involved in arousal and inhibition. The stability of platelet MAO (Murphy, Wright, Buchsbaum, Nichols, Costa & Wyatt 1976), even across drastic clinical state changes, and the fact that it is low in well relatives of patients as well as in patients, indicates that it could not be related to "general arousal," as suggested by Suedfeld. Furthermore, MAO-inhibiting drugs do not result in a sudden increase in arousal like the catecholamine stimulants, amphetamine and cocaine.

A frequently raised question concerns the relationship between MAO, the brain monoamines it regulates, and their metabolites in peripheral sources. **Von Knorring** suggests that MAO is *positively* related to serotonin levels in both the brain and its metabolite 5-HIAA in the CSF. However, in the study by Ballenger, Post, Jimerson, Lake, Murphy, Zuckerman, and Cronin (1983), a low but significant *negative* correlation was found between platelet MAO and CSF 5-HIAA. Whereas the Type A MAO, which oxidizes 5-HT in human and rat brains, decreases with age, 5-HT increases with age at least in rat brains (Oreland & Fowler 1982). The positive correlation reported by von Knorring is also puzzling in view of the fact that MAO-inhibitors block brain 5-HT catabolism, thereby increasing brain 5-HT concentration. This would imply a negative rather than a positive correlation between MAO and serotonin. The fact is that these biochemical correlations are not sufficient evidence to generalize about the process interactions between enzymes and neurotransmitters. My hypothesis, which is similar to the model proposed by Haier et al. (1980), is that low levels of MAO in the brain allow monoamine neurotransmitter levels to fluctuate over a broader range in response to stress. The consequence is a vulnerability to the type of psychopathology in the affective disorders that is characterized by a failure of mood regulation. The actual correlation between MAO levels and the neurotransmitters may be either positive or negative, depending on the state of the subjects.

Catecholamine systems and sensation seeking. There is insufficient specificity, according to **Mason** and **Redmond,** and too much specificity, according to **Clavier, Panksepp & Siviy,** and **von Knorring,** involved in assigning a primary role to the catecholamine systems in the trait of sensation seeking. Certainly, as Mason points out, there is evidence of independent functions of noradrenergic and dopaminergic systems, some of which I have cited. However, for many of the behaviors relevant to sensation seeking, such as general activity, sociability, sexual behavior, and intracranial self-stimulation, one sees complementary roles for dopamine and NE. Despite the popular psychiatric focus on NE and 5HT in depressive disorders and a dopamine hypothesis for schizophrenia, dopamine has also been implicated in depression (Jimerson & Post 1984) and NE in schizophrenia (Lake, Sternberg, van Kamen, Ballenger, Ziegler, Post, Kopin & Bunney 1980; Stein 1978). Given the fact that the first correlative findings for sensation seeking in humans were limited to NE and DBH (Ballenger et al. 1983) there has been a temptation to limit the theory to the noradrenergic system rather than involving dopamine and serotonin. However, the comparative studies on other species suggested that this narrowing of the model might be premature, given the uncertainties of the relations between activity of these systems in the brain and the peripheral indicators of their activity in humans. The recently obtained correlative data from Sweden (Schalling et al. 1984) suggest that this caution was justified since these researchers have found some modest but significant relationships between CSF, serotonin and dopamine metabolites, and their sensation-seeking measure (the monotony-avoidance scale) in patients, with similar but nonsignificant tendencies seen in normal individuals. The serotonin metabolite findings should reinforce von Knorring's idea. Given the present state of the literature on humans and other species it would be premature to be more specific about the separate roles of dopamine and NE in the behavioral manifestations for a broad sensation-seeking trait. The term catecholamine systems activity (CSA) was used to cover a great complexity of specific receptors with different effects in these systems (as noted by Redmond) for similar reasons.

In contrast to **Mason** and **Redmond, Clavier, Panksepp & Siviy,** and **von Knorring** feel that it is premature to assign a central role to the monoamine systems in a complex trait like sensation seeking. These commentators believe that all of the brain's transmitter systems are involved in all major activities in complex, unknown interactions. Mason believes that to view brain functions as "divergent, redundant, and plastic" (Clavier's words) is a "retrograde step." I have tried to take a middle ground between these two views of the function of neurotransmitter systems; the separate system may have central roles in certain broad behavioral functions with other systems acting as regulators or modifiers in these activities. Einstein believed that God did not "throw dice in the universe." Although I am not sure of the sensation seeking status of God, I do not believe that she constructed specific neurotransmitter pathways without some guidance from an adaptive behavioral blueprint. It may be that some of these systems serve generalized behavioral arousal or inhibition functions affecting a variety of activities (Panksepp 1982), but this still leaves open the question of the biological substrate of sensitivity to signals of positive and negative reinforcement (Gray 1982). As a strictly practical matter, one cannot solve complex equations with all unknowns.

The role of the noradrenergic system. In a review of the neurobiology of some dimensions of personality (Zucker-

man et al., in press) we pointed out that the theories of the function of the noradrenergic system originating in the locus coeruleus were in more disagreement than those for other neurotransmitter systems. The controversies are discussed in the commentaries of **Clavier, Mason,** and **Redmond.**

Mason faults me for not citing the large body of literature opposed to the Stein-Crow hypothesis that the NE system mediates the intrinsic reward functions. I also did not cite the equally large body of literature supporting the hypothesis. To have cited all of the relevant studies in all of the well-researched areas discussed in the target article would have resulted in more of the text being inside the reference parentheses than outside of them.

Clavier points out that self-stimulation has been obtained from areas of the brain without high concentrations of catecholamines, suggesting that the hypothesis limiting reward to catecholamine systems is in error. The fact remains that the areas from which the highest rates of self-stimulation are obtained, such as the medial forebrain bundle and lateral hypothalamus, are rich in catecholamine neurotransmitters. The crucial studies in relatively specific neurotransmitter sites like the locus coeruleus and substantia nigra have yielded a more equivocal mixture of findings, so Clavier may be right in his assumption that reward is not restricted to one or two systems; I have suggested that only certain optimal levels of activity in catecholamine systems are intrinsically rewarding. The failure of Clavier to find effects of removal of these amines by lesions of the CS systems is surprising in view of the success of Stein (1974; 1978), for whom lesioning the NE system removed such effects and restoring the neurotransmitter reinstituted self-stimulation.

Mason complains that I ignored his selective attention hypothesis of noradrenergic function. Admittedly, I should have paid more attention to this well-documented view, but in actual fact it is a further extension of his hypothesis that NE mediates response to novel stimuli and lesions of the system increase "neophobia" in rats (Mason, Roberts & Fibiger 1978) which I did mention. I also devoted considerable attention to the Aston-Jones and Bloom (1981) study that shows how the NE system mediates attention and response to novel stimuli. Such stimuli may be rewarding at optimal levels and negatively reinforcing at higher levels. The unflattering analogy with which Mason concludes his commentary notwithstanding, my views of the functioning of the noradrenergic system are not very unlike his own. Mason's new theory of noradrenergic function suggests research comparing high and low human sensation seekers on tasks requiring selective attention. This illustrates how good ideas for research can arise from base criticisms or "urine" analyses.

According to my new model, anxiety may be found, secondary to depression, at the low end of the CSA continuum or in a primary function at the high end. Therefore, from the middle range to the high end of CSA the model is in agreement with the fear-related arousal function of NE as suggested by **Redmond.** However, Redmond questions the placing of depression at the low end of the CSA scale, citing the more recent hypothesis that antidepressants work by decreasing sensitivity of brain noradrenergic-beta receptors rather than by enhancing their action and increasing release and blocking reuptake in presynaptic neurons. The inference from this theory is that depression is produced by too much sensitivity of the NE system and a tendency to overrespond rather than by a depletion of NE. A recent review in *BBS* of this controversy (Stone 1983) finds major inconsistencies with research findings in both the older NE depletion and the recent NE overactivity theories. Stone suggests a third hypothesis in which depression begins when the output of noradrenergic cells is too low to meet demands resulting from internal or external stress. The low output of NE-Beta effector cells prevents peripheral adaptation to stress. Stone's hypothesis is similar to the old catecholamine deficit theory in the original causes of depression although it differs in an emphasis on the receptor effects of NE depletion. Whatever one concludes after reading his impressive literature review and the critical commentaries, it is clear that the catecholamine deficit hypothesis, although found wanting, has not yet been displaced by its opposite. As Stone points out, the field of interest has shifted from presynaptic neurons to postsynaptic receptors. It is true that my model is based on the older approaches. There are simply not enough data using the newer approaches, particularly behavioral animal data and clinical or behavioral human data. Data from normal humans show negative correlations between trait depression and anxiety measures and the NE metabolite MHPG in plasma (Ballenger, Post, Jimerson, Lake & Zuckerman 1984).

Redmond points out the ambiguity of the interpretations of differences in CSF metabolites in view of the negative feedback between receptors and presynaptic neuronal activity. Could low levels of NE in the CSF be the result of an *overactive* brain system producing sensitized postsynaptic receptors? This is certainly a possibility, but I believe the most parsimonious assumption is one of a direct positive relationship between brain activity and indices derived from peripheral sources at any given time. Many of these issues could be addressed in autopsy studies correlating neurotransmitter levels in different parts of the nervous system.

Redmond's new data from stimulating the locus coeruleus of unrestrained monkeys suggest that some of the results from chair-restrained monkeys were not due to restraint alone. However, the activity shown by unrestrained monkeys is not the behavioral inhibition one would expect if the stimulation were triggering a fear system. As Redmond himself says in his commentary, we must be wary of interpreting behavior in terms that fit our theories. Even if the activity were symptomatic of fear, the fear might be produced by a sudden, unexplained increment in arousal rather than the triggering of a fear system. Surely he would not maintain that all activity in the locus coeruleus is an indication of fear arousal in view of the variety of nonnoxious stimuli that are capable of eliciting much activity (Aston-Jones & Bloom 1981).

Brain plasticity and sleep phenomena. The commentary by **Eterović & Ferchmin,** applying the model in Figure 6 to the findings on environmental enrichment and REM sleep (Rosenzweig & Bennett 1977), is most provocative and suggests entire new lines of research in animals and humans. Their implication of cortical growth in a positive feedback system with environmental stimulation and CSA suggests a role for environmental stimulation in the

shaping and maintaining of the sensation-seeking trait. Since both social stimulation and complexity of inanimate stimulation promote cortical growth, the importance of these factors in the trait is emphasized. The role of the monoamines, particularly the serotonergic and adrenergic systems in sleep, was pointed out some time ago (Kales 1969).

Although no study on the relationship between REM sleep and sensation seeking has been done, a report by Coursey, Buchsbaum, and Frankel (1975) indicated that high sensation seekers and augmenters (EP) are more "sleep efficient" and lows and reducers (EP) are more insomniac. The sleep efficiency of the highs could be related to a greater proportion of REM sleep. Other environmental conditions that promote brain growth are related to sensation seeking. High sensation seekers prefer more complex to less complex visual stimuli and respond more positively to confinement in an environment with social and changing, complex visual and auditory stimulation (Zuckerman, Persky, Link & Basu, 1968). In a population of high-sensation-seeking drug users, higher sensation seekers show a greater preference for amphetamine and psychedelic drugs (Carrol et al. 1982). If a sensation-seeking style of life promotes cortical growth, there should be some correlation between the trait and intelligence. Low but positive and significant correlations are found between sensation seeking and intelligence (Zuckerman 1979b, pp. 233–35). Although none of these results on humans provides a direct test of the brain plasticity hypothesis they are suggestive. Brain studies of animals and sleep studies of humans addressed to the hypothesis are called for.

Genetics and evolution

My use of genetic-evolutionary theory to explain the origins of the trait of sensation seeking is called into question by **Baldwin, Panksepp & Siviy,** and **Suedfeld.** Panksepp & Siviy classify sensation seeking as an acquired trait along with traits like dominance–submission, altruism, chastity, and piety. I do not know about the last two, which may be more state than trait, but biometric-genetical studies have established the genetic component in the first three (see Rushton, Fulker, Neale, Blizard & Eysenck, in press, for a recent study of altruism). It seems axiomatic to say that sensation seeking is a function of "several concurrently interacting neurobehavioral systems." Is there any complex behavior for which this statement is not true? Panksepp & Siviy also assume that the biological basis for the sensation-seeking trait is just now evolving in the human nervous system and only in technologically advanced societies where people have the leisure to be bored. I believe that their concept of sensation seeking is too narrow and limited to one of the four factors: boredom susceptibility. Even here, it has been noted that other primates, like gorillas, seem to suffer from boredom. However, in terms of the seeking of stimulation unrelated to primary needs, the case for such a general drive has been experimentally demonstrated in animals from the rat on up (Fowler 1965). All human societies have activities like carnivals and contests that have little to do with foraging. For many centuries war itself was probably motivated for the individual volunteer more by the need for excitement and a change of routine than by an innate aggressive drive (Fromm 1973). **Baldwin** believes that I put too much stress on heredity, and **Suedfeld** claims that I am simply substituting a "complex and mysterious set of biological" factors for an equally mysterious set of psychological phenomena. I did not mean to imply that environmental influences were unimportant, although it is curious how most biometric studies of personality show little effect of between-family variance and only some effect of within-family variance (Eysenck 1983). Perhaps the family is wrongly conceived of as the major influence in personality development. The heredity–environment interaction is a complex one, and the reaction to environmental influences is not solely produced by these same influences.

Heredity itself is not so mysterious since the discovery of the DNA molecule. What is mysterious are the intervening biological mechanisms through which the genotype influences behavior. Mysterious as the problem may be, it is a potentially solvable one. In the target article I have tried to guess which biological mechanisms are genetically coded and influence sensation-seeking behavior through the physiology of the brain. I have used animal models and a comparative approach to seek behavioral parallels and common biological correlates of human and animal behavior traits.

Twenty-five years ago we asked what seemed to be a simple question: Is a sensation-seeking trait the source of the wide individual differences seen in response to sensory deprivation? Somehow we have ended by speculating about the variations of brain chemistry as the basic source of dimensions of personality. Conceivably, we have gotten everything wrong about the biological basis of sensation seeking. What we have is a handful of replicated findings in humans linking psychophysiological and biochemical variables to the trait measure which in turn is correlated with a great variety of life phenomena. We have searched the comparative literature in behavior genetics and psychopharmacology and the clinical literature of biological psychiatry to make some sense of the findings. The resultant model is open to testing and demolition if that is necessary. My most optimistic hope is that only some finishing and alterations will be required. In actual fact one of the floors is still fairly empty: the one between physiological functioning and life experience, or the area of reinforcement sensitivity and social interaction. We will have to do something about that.

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