Cognitive Therapy and Research, Vol. 21, No. 2, 1997, pp. 135-156

Neuroticism and the Attentional Mediation of Dysregulatory Psychopathology

John F. Wallace and Joseph P. Newman[1]
University of Wisconsin—Madison

Neuroticism emerges consistently as a fundamental trait in factor-analytic studies of personality and temperament, and its association with diverse forms of psychopathology has been demonstrated repeatedly. Hence, elucidating the nature of neuroticism would be expected to advance understanding of the psychobiological processes contributing to psychopathology. In contrast to formulations that regard neuroticism as a predisposition to negative affect primarily, we emphasize neuroticism's effects on cognition. Specifically, we propose that (a) neuroticism is associated with facilitation of the automatic orienting of attention, (b) this attentional process may adversely affect one's ability to engage in the controlled evaluation and correction of problematic cognitive and behavioral response tendencies, and (c) negative affect and psychopathology associated with neuroticism result from the disruption of these evaluative and corrective processes.

KEY WORDS: attention; neuroticism; psychopathology; self-regulation.

Factor-analytic studies approach unanimity in identifying a small number (typically from two to five) of broad and pervasive dimensions of human personality (e.g., Eysenck, 1967; Eysenck & Eysenck, 1985; McCrae & Costa, 1987; Tellegen, 1985; Zuckerman, Kuhlman, Joireman, Teta, & Kraft, 1993). H. J. Eysenck has called one of these personality dimensions neuroticism (N). To use his terminology, high-N individuals manifest the personality trait of neuroticism, whereas low-N individuals are designated stable.

[1]Address all correspondence to Joseph P. Newman, Department of Psychology, 1202 West Johnson Street, Madison, Wisconsin 53706.

0147-5916/97/0400-0135$12.50/0 © 1997 Plenum Publishing Corporation

This personality dimension is of particular interest because there is a clear association between the trait of neuroticism and the occurrence of diverse forms of psychopathology, such as anxiety and mood disorders (Clark, Watson, & Mineka, 1994; Eysenck, 1967), alcoholism (Sher & Trull, 1994), and personality disorders (Widiger & Costa, 1994). Indeed, even in nonclinical populations, individuals high in trait neuroticism report more anxiety and depression (Eaves, Eysenck, & Martin, 1989) and more symptoms associated with personality disorders (O'Boyle & Holzer, 1992) than do their stable counterparts.

Various explanations have been offered for the demonstrated association between neuroticism and psychopathology. H. J. Eysenck's original conceptualization equated neuroticism with emotionality or general reactivity: The neurotic individual "is emotional, reacting too strongly to all sorts of stimuli" (Eysenck & Eysenck, 1975, p. 5). These "strong emotional reactions interfere with his proper adjustment, making him react in irrational, sometimes rigid ways" (Eysenck & Eysenck, 1975, p. 5).

In contrast to Eysenck's original conceptualization of neuroticism as general "emotional reactivity," the trait of neuroticism has been described by other researchers as a predisposition or susceptibility to experience negative affect states (e.g., Costa & McCrae, 1980; Larsen & Ketelaar, 1989; Tellegen, 1985; Watson & Clark, 1984). That is, in any given situation, neurotic individuals are more likely to experience feelings such as distress, fear, and hostility than are stable persons.

One of the most influential statements of this position is that of Tellegen (1985). He proposed that mood states can be characterized with reference to two higher-order dimensions—positive affect and negative affect—and cited data from numerous studies of self-report mood ratings in support of this position. Tellegen also asserted that the personality dimension N is most closely associated with the mood state dimension of negative affect, whereas Extraversion (E)—a second higher-order dimension described by H. J. Eysenck (e.g., Eysenck, 1967)—is associated with positive affect.

Finally, Tellegen (1985) suggested that the negative affect dimension corresponds to a dimension of sensitivity to punishment cues described by Gray (e.g., Gray, 1981; Gray, Owen, Davis, & Tsaltas, 1983). Positive affect, on the other hand, corresponds to a sensitivity-to-reward dimension. These postulates imply that N may be construed as reflecting individual differences in susceptibility to negative affect and in sensitivity to punishment cues. Consistent with this proposal, Larsen and Ketelaar (1989) found that the self-reported mood ratings of neurotic subjects differed from those of stable subjects following punishment cues, whereas group differences were less pronounced in reaction to success feedback.

However, results using behavioral rather than self-report measures have on occasion been somewhat at odds with Tellegen's hypothesis that N reflects negative affectivity and sensitivity to punishment cues. For example, Derryberry and Reed (1994) used a reaction time task to investigate the influences of personality on the covert shifting of attention. In their paradigm, target stimuli were preceded by cuing stimuli, which were of either positive, negative, or neutral hedonic valence. Derryberry and Reed found that extraverts were impaired in their ability to shift attention away from positively cued locations in order to detect targets, whereas introverts were impaired in their ability to shift their attention away from negatively cued locations. Moreover, these biases tended to be more pronounced in neurotic subjects than in stable subjects.

That is, rather than finding E associated with differences in reactions to positive cues, these researchers observed that extraverts and introverts differed in their reactions to negative, as well as positive, cues. Furthermore, rather than finding N to be associated only with differences in reactions to negative cues, neuroticism was associated with an augmentation of biases involving both senstivity to positive cues and sensitivity to negative cues. Derryberry and Reed (1994) concluded that "the data do not support proposals that E reflects only positive motivation and N only negative motivation" (p. 1137).

In another relevant experiment, Wallace and Newman (1990) investigated the effects of incentive contexts on response speed. The subjects' task was simply to trace a circle template as slowly as possible. One condition entailed the possibility of monetary gain, which was not contingent on task performance. A second condition involved the possibility of losing money. Contrary to what might be expected based on Tellegen's (1985) hypothesis, introverts and extraverts did not significantly differ in their behavioral reaction (tracing speed) to the reward cues present in the situation. Neither did stable and neurotic subjects differ in their behavioral reactions to punishment cues. Rather, as was the case in the data collected by Derryberry and Reed (1994), neurotic introverts were most affected by the punishment cues, and neurotic extraverts were most affected by the reward cues.

We acknowledge that these results do not necessarily refute the associations of N with negative affect and E with positive affect proposed by Tellegen (1985). However, they do raise the question of whether his formulation is as applicable to other behavioral responses as it is to self-report or other measures of one's subjective experience of mood or affect. That is, these studies using behavioral measures indicate that simply characterizing N as a dimension of negative affectivity may not constitute a sufficiently detailed explanation of the psychological processes underlying N.

An alternative to according primacy to affect in characterizations of neuroticism follows from the trend during the past two decades to focus increasingly on information processing and cognition to explain negative affect and psychopathology. Cognitive theorists (e.g., Beck, 1976; Beck, Rush, Shaw, & Emery, 1979; Ellis, 1962) assert that affect states—including negative emotions such as anxiety and depression—are strongly influenced by a person's cognitions (e.g., the interpretations that one forms of situations and events).

This conceptualization of the relationship between cognition and affect suggests an alternative line of inquiry into the nature of neuroticism: It may be that N reflects, at least in part, individual differences in processes that directly affect cognition, and that alterations in these cognitive processes in turn influence the individual's affect state. That is, neuroticism may constitute a predisposition to negative affect only secondarily, as a consequence of its effects on information processing.

Self-regulatory processes comprise one class of cognitive activity that we believe is relevant to understanding maladaptive behavior, excessive negative affect, and indeed much of psychopathology (Newman & Wallace, 1993a; Newman et al., 1993; Patterson & Newman, 1993). Other researchers also have suggested that symptoms associated with psychiatric disorders are caused by failures to regulate one's behavior, affect, and/or cognitive processes (e.g., eating disorders, Kirschenbaum, 1987; impulsivity, Shapiro, 1965; alcoholism, Tarter, 1978; substance abuse, Tiffany, 1990). For example, an aggressive child may tend to interpret (often erroneously) ambiguous interactions as indicative of hostile intent on the part of others (e.g., Dodge & Crick, 1990; Dodge & Newman, 1981). If this erroneous interpretation is not adjusted (i.e., regulated) to reflect more closely the actual situation, the child may feel angry and engage in an act of unprovoked aggression.

We refer collectively to the failures of these processes to perform adequately their adaptive functions as *dysregulation.* So, to use this terminology, many forms of psychopathology may be regarded as manifestations of dysregulation—the disruption of self-regulatory processes. Herein, we suggest that neuroticism contributes to negative affect and psychopathology by impairing cognitive processes involved in self regulation. In other words, neuroticism may be conceptualized as a predisposition to dysregulation.

Because our arguments regarding the nature of neuroticism rest so heavily on the concept of dysregulation, this manuscript is divided into two discrete sections. In the first, we describe a ubiquitous psychological process—the automatic orienting of attention—that at times may have significant dysregulatory consequences. In order to explicate clearly the ways in which the orienting of attention may disrupt self-regulatory processes, we also discuss in more detail our views of self-regulation and dysregulation.

Having laid the necessary groundwork, we then return to the topic of neuroticism, and suggest that this trait reflects in part the facilitation of the automatic orienting of attention. As a result, neuroticism promotes dysregulation, and we conclude that the association of negative affect and numerous forms of psychopathology with neuroticism is a direct consequence of this susceptibility to dysregulation.

ATTENTION AND DYSREGULATION

Automatic Information Processing and the Automatic Attention Response

Our understanding of how a seemingly innocuous process such as the reflexive orienting of attention impairs self regulation is based largely upon (a) Schneider and Shiffrin's (1977; Shiffrin & Schneider, 1977) conceptualizations of controlled and automatic information processing, and (b) their description of the automatic attention response. Although some researchers have questioned the validity of this distinction, noting the difficulty in describing particular processing tasks as *purely* automatic or controlled (Bargh, 1989), differentiating between tasks that are *predominantly* automatic or controlled has been found to be useful in a number of research domains [e.g., Devine (1989), Gilbert (1989), below].

According to Schneider and Shiffrin, controlled processing "is characterized as a slow, generally serial, effortful, capacity-limited, subject-regulated processing mode that must be used to deal with novel or inconsistent information" (Schneider, Dumais, & Shiffrin, 1984, p. 2). On the other hand, "automatic processing is a fast, parallel, fairly effortless process that is not limited by short-term memory (STM) capacity, is not under direct subject control, and is responsible for the performance of well-developed skilled behaviors" (Schneider et al., 1984, p. 1). That is, these processes are "activated automatically without the necessity of active control or attention by the subject" (Schneider & Shiffrin, 1977, p. 2). The automatic attention response is one such automatic process.

Schneider and Shiffrin (1977; Shiffrin & Schneider, 1977) studied this process in a series of experiments that utilized a visual search paradigm, in which subjects attempted to detect one or more target characters among one or more nontargets. They concluded that the automatic attention response

> modifies ongoing controlled processing by attracting attention to a specified locus. . . . When subjects in search tasks are consistently trained to recognize certain inputs as targets, these inputs acquire the ability to initiate *automatic-attention responses.* These attention responses then direct attention (i.e., will direct controlled

> processing) automatically to the target, regardless of concurrent inputs or memory load. (Schneider & Shiffrin, 1977, p. 2)

Automatic attention responses, then, may "interrupt ongoing control processing and forcefully reallocate attention and resources" (Schneider et al., 1984, p. 22).

Controlled Information Processing and the Regulation of Automatic Processes

Again, the other processing mode described by Schneider and Shiffrin is controlled information processing. In addition to the properties enumerated above, controlled processes [which Shiffrin (1988) called *attentive processes*] are "limited-capacity processes requiring attention" (Shiffrin & Schneider, 1977, p. 160). Hence, "because active attention by the subject is required, only one such sequence at a time may be controlled without interference, unless two sequences each require such a slow sequence of activations that they can be serially interwoven" (Schneider & Shiffrin, 1977, pp. 2-3). A simple example of a controlled process is adding a column of numbers; if attention is drawn elsewhere, the addition process terminates.

Several researchers have stressed the importance of controlled information processing for regulating automatic cognitive processes. For example, in the realm of social cognition Devine (1989) has demonstrated that controlled processing may be used to inhibit and modify the effects of automatic stereotype activation. Specifically, nonprejudiced individuals use controlled processes to correct automatically generated stereotype-based responses, so these responses are not manifested behaviorally.

A similar suggestion is embodied in Gilbert and colleagues' (e.g., Gilbert, Krull, & Pelham, 1988; Gilbert, Pelham, & Krull, 1988) model of the social inference process. In this model, people may consider both dispositional and situational factors when making inferences about the causes of others' social behavior. Dispositional influences on social behavior are character traits that cause one to perform an observed action; situational influences are external constraints on a person's social behavior.

According to Gilbert (1989), "perceivers *first* draw dispositional inferences about others and *then* correct these inferences with information about the situational forces that may have coerced the other's actions" (p. 193). The formation of dispositional inferences is termed the *characterization* process. Those dispositional inferences are subsequently adjusted by the *correction* process to take into account situational factors. The characterization process is portrayed as "a relatively automatic process that requires little effort or attention, whereas correction is a deliberate form of conscious reasoning that happens slowly and effortfully" (Gilbert, 1989, pp. 193-194). That is, the products of "relatively effortless or automatic" (p. 195) characterization processes are corrected via "relatively effortful or controlled" (p. 195) correction processes.

Gilbert (1989) concluded that the correction stage involves "a limited-capacity mechanism that does not itself perform tedious and routine analyses (e.g., the sorts of analyses that turn behavioral observations into dispositional inferences), but merely *checks* and *corrects* [italics added] the outputs of other 'upstream' or 'off-line' systems" (p. 206). This mechanism functions "like the inspector in a garment factory: It does not make a product, but it checks to make sure a product is perfect. If an imperfection is found, it institutes a remedy" (p. 206).

The formulations of Devine and Gilbert (see also Bargh, 1984; Posner & Snyder, 1975) suggest that self-regulation often involves the controlled evaluation and correction of the outcomes, results, or products of automatic processes. Hence, in many cases controlled processing is the *sine qua non* of self-regulation (Kanfer & Gaelick, 1986; Shapiro, 1965).

A clinical example of a controlled self-regulatory sequence might involve, first, the evaluation of an excessively negative interpretation of some situation or event [which may be considered the product of an automatic inference process (Hollon & Garber, 1988, 1990)]. If the thought is determined to be inaccurate, the evaluative activity is followed by the correction of this thought (i.e., the generation of a more benign alternative interpretation). However, if the negative interpretation is not perceived to be inaccurate or is not corrected, it adversely influences affect and/or behavior.

The Dysregulatory Effects of the Automatic Orienting of Attention

This view (that controlled information processing is integral to self-regulation) is fundamental to our understanding of the dysregulatory effects of the automatic orienting of attention. Recall first that controlled processes are "limited-capacity processes requiring attention" (Shiffrin & Schneider, 1977, p. 160). It follows that whenever sufficient attentional resources are *not* available, controlled processing will be impaired. Accordingly, if attention is automatically attracted by a stimulus, "such an automatic process *must interfere* with other ongoing attentive [controlled] processing" (Shiffrin, 1988, p. 761). That is, the automatic orienting of attention may "interrupt ongoing control processing" (Schneider et al., 1984, p. 22).

This formulation implies that the orienting of attention to a stimulus may decrease the attentional resources available to support controlled self-regulatory processes. Consequently, self-regulatory processes may be curtailed. In other words, the automatic orienting of attention may foster dysregulation by disrupting controlled information processing. Therefore, automatic orienting of attention that promotes dysregulation may be considered a *common feature* (Ingram, 1990) of psychopathology.

One result of the impairment of controlled self-regulation is an increase in the influence of automatic processes. That is, because the evaluation and correction of the products of automatic processes require controlled processing, as controlled processes are disrupted the likelihood increases that these products will be manifested. In sum, when attention is automatically directed elsewhere, controlled self-regulatory processes may be compromised, and the maladaptive products of automatic processes (e.g., erroneous attributions or inferences, and the resulting inappropriate affective and behavioral responses) are increasingly manifested.

Illustrative Examples

The orienting of attention is often conceived as being elicited primarily by stimuli in one's external environment. Nonetheless, it is clear that covert products of cognitive processes, such as thoughts or mental images, also may function as stimuli that attract attention. For example, Ingram (1990; see also Carver & Scheier, 1986; Pyszczynski & Greenberg, 1987) has described ways in which self-focused attention—attending to internally generated stimuli or cognitive products that relate to the self (e.g., self-deprecating attributions)—is causally related to psychopathological states. Specifically, it is the "excessive, sustained, and rigid attention to information emanating from internal sources" (Ingram, 1990, p. 169) that promotes diverse forms of psychopathology. This rigid attentional focus may be disruptive of the task performance. In addition, Ingram suggested that the specific content of the cognitive products that are the objects of the internal attentional focus (e.g., self-deprecation, worry, grandiosity) determines the type of psychopathology that is manifested (e.g., depression, anxiety, mania).

Likewise, in analyzing processes involved in clinically significant anxiety, M. W. Eysenck (e.g., Eysenck, 1979, 1981; Eysenck & Eysenck, 1985) has emphasized the debilitating effects of internal stimuli, such as negative expectations and self-evaluations. According to M. W. Eyseinck, attending to negative cognitions such as these impairs one's ability to perform cognitive and motor tasks by increasing the amount of effort that is required to achieve a given level of task performance.

These perspectives appear to be quite compatible with our present formulation, particularly when self-regulation is conceptualized as a cognitive task. For instance, a person who views social situations as threatening, expecting failure and humiliation, may consequently experience considerable tension and anxiety in such situations. Internal stimuli such as anxiogenic thoughts (e.g., expectations of negative evaluations by others) strongly attract attention because they are highly significant to the individual. As a result, the attention available to support controlled self-regulation is reduced. Therefore, to the extent that anxiogenic thoughts continue to be generated, the person is susceptible to disruption of controlled self-regulatory processes.

Depression may be viewed in a similar manner to that just described, with automatically generated depressogenic thoughts [e.g., negative thoughts about oneself, one's immediate life situation, and one's future (Beck, 1976)] not only causing feelings of depression, but also functioning as stimuli that attract attention. Again, the result is the ongoing disruption of the ability to engage in the controlled evaluation and correction of the problematic thoughts, with the consequent continuation of the person's depressed mood.

This formulation also suggests why anxiogenic or depressogenic thoughts (and thus the resulting feelings of anxiety or depression) often are so difficult to modify. First, the cognitive processes that generate such thoughts are largely automatic (e.g., Hollon & Garber, 1988, 1990), so those thoughts occur without effort or intention. Second, as long as anxiogenic or depressogenic thoughts are being automatically generated they attract attention on an ongoing basis, curtailing the ability to engage in the controlled evaluation and correction of those very thoughts. Hence, the inappropriate affective reactions caused by those thoughts may continue undiminished.

Note that this analysis implies that orienting *automatically* and without intention to thoughts that require correction may actually compromise the ability to engage in the requisite self-regulatory processes. Although this assertion may appear somewhat paradoxical, it is important to bear in mind that when engaged in a controlled process, it is often necessary not only to attend to the relevant stimuli (i.e., the input data), but also to allocate attention to the control/regulation of the data processing itself. For example, simply attending to a group of numbers (e.g., a street address) does not necessarily produce the addition of those numbers; one also must concentrate on carrying out the addition process. Consequently, to the extent

that one is unable to allocate attention flexibly between data and process, the controlled process will be disrupted (see also Ingram, 1990).

The type of analysis just presented also may be applied to disorders in which the symptoms are primarily behavioral. As an example, we refer to Cloninger's (1987) typology of alcoholism, which identifies two classes of problem drinkers. His Type 1 alcoholics manifest a relatively late onset of problem drinking, and are able to remain abstinent for considerable periods. However, Type 1s are "unable to terminate drinking binges once they had started" (Cloninger, 1987, p. 411): They exhibit loss of control drinking. In addition, individuals manifesting the Type 1 pattern tend to be "hypervigilant and apprehensive" (Cloninger, 1987, p. 411), experience "cognitive anxiety or frequent anticipatory worrying" (Cloninger, 1987, p. 413), and feel guilty about their drinking behavior.

In contrast, Type 2 alcoholics engage in frequent alcohol-seeking behaviors, and the onset of their problem drinking is relatively early. Moreover, they manifest characteristics associated with impulsivity and tend to engage in novelty seeking. Indeed, "alcohol-seeking behavior may be considered a special kind of exploratory *appetitive* [italics added] behavior" (Cloninger, 1987, p. 414).

When analyzing these behavior patterns, we note first that drinking for Type 1 individuals may be conceptualized as a learned strategy for coping with aversive thoughts and feelings. That is, over time drinking becomes an habitual or automatic response to stress. Accordingly, drinking bouts are likely to be initiated by anxiety-provoking thoughts and the ensuing negative affect state.

Because those anxiogenic thoughts divert attention that might be used to support controlled self-regulatory activities, there is a decrease in the likelihood that the habitual drinking response will be adequately evaluated and altered. For example, an impaired ability to engage in controlled processing renders the generation of alternative coping strategies rather difficult (see Steele & Josephs, 1988).

Furthermore, not only does drinking constitute a (maladaptive) coping response to stress, but, for Type 1 alcoholics, stress also is a *result* of engaging in—or even contemplating—this behavior. That is, because drinking is contrary to the person's standards of behavior, and hence is viewed as unacceptable, thoughts of drinking engender feelings of guilt.

Of course, these thoughts and feelings occupy attention, and further decrease the efficacy of self-regulatory activities. Moreover, because these stimuli are aversive, the person's habitual or automatic response to stress (i.e., having a drink) is continually activated. In consequence, the drinking behavior may continue unabated: Loss of control or binge drinking results (see Marlatt & Gordon, 1985; Tiffany, 1990, for related suggestions).

Whereas the crux of the Type 1 drinking behavior involves reactions to aversive stimuli, for Type 2 alcoholics drinking occurs primarily in appetitive motivational contexts. But, again, the stimuli that elicit alcohol-seeking behavior (e.g., thoughts of anticipation of alcohol's pleasurable effects) perforce attract attention, and so may decrease the ability to evaluate and alter the drinking response.

These examples are by no means intended to be exhaustive; other forms of psychopathology (e.g., obsessive-compulsive disorder; see below) and maladaptive behavior (e.g., violence; Newman & Wallace, 1993b) are amenable to analysis in terms of dysregulatory processes, such as the automatic orienting of attention. Rather, we offer these examples simply to demonstrate how the automatic orienting of attention might contribute to psychological disorders.

Now, having laid the necessary groundwork with the preceding discussion of attention and dysregulation, we turn to the consideration of the nature of neuroticism, and discuss how this trait is conducive to negative affect and psychopathology.

NEUROTICISM AND DYSREGULATION

Our research agenda has emphasized individual differences as important moderators of dysregulatory processes (for reviews see Newman, 1987; Newman & Wallace, 1993a; Patterson & Newman, 1993; Wallace, Bachorowski, & Newman, 1991). One result of this focus is a model of personality—which we describe next—that is essentially a synthesis of Gray's (1975, 1987; Gray & Smith, 1969) neuropsychological model and Eysenck's (1967) personality theory of E and N (see also Wallace et al., 1991; Wallace & Newman, 1990). As discussed below, describing N in terms of Gray's model allows us to relate neuroticism directly to potentially dysregulatory attentional and motor processes. Specifically, this model implies that the trait of neuroticism constitutes a predisposition to experience a facilitation of response speed and of the automatic orienting of attention.

Gray's Model

In recent years, investigators have applied Gray's model to such diverse topics as psychopathy (Fowles, 1980, 1988), anxiety (Gray, 1982a, 1982b), alcoholism (Pihl, Peterson, & Finn, 1990), affective disorders (Fowles, 1984), conduct disorder (Walker et al., 1991), and the expression of emotion (Buck, 1984). Briefly, Gray has posited a model of the central

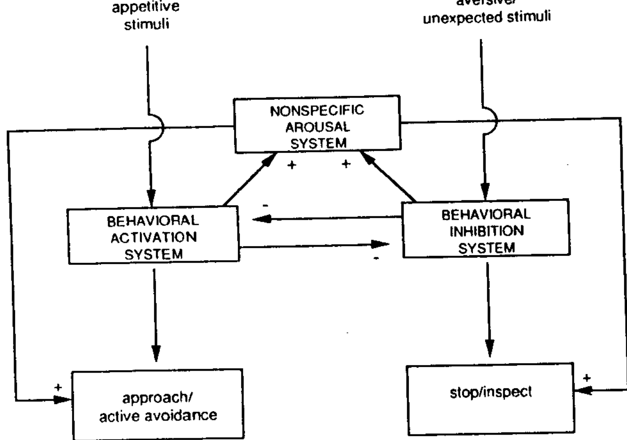

Fig. 1. Gray's neuropsychological model. Adapted from Gray (1987).

nervous system, two components of which are the behavioral activation system (BAS) and the behavioral inhibition system (BIS).[2]

As depicted in Fig. 1, the BAS is sensitive to conditioned appetitive stimuli (e.g., stimuli associated with reward): Its activity level increases when appetitive stimuli are detected. The function of the BAS is to initiate goal-directed behavior. As the level of BAS activity increases, the likelihood that approach behavior will occur also increases.

Conversely, Gray has described the BIS as sensitive to conditioned aversive stimuli (e.g., stimuli associated with punishment), and to unexpected or novel stimuli. As its activity level increases, the BIS acts to inhibit ongoing behavior and initiates the orienting of attention to the discrepant or potentially threatening stimulus.

The third component of Gray's model—the nonspecific arousal system (NAS)—receives inputs from the BAS and the BIS (see Fig. 1); an

[2]Although Gray's formulation of his neuropsychological model has continued to evolve (e.g., Gray, 1987, 1991), we have found the three-system version described herein (see also Fowles, 1980) to be the most useful for investigating the influences of individual differences on dysregulatory processes. We certainly do not claim that this version is necessarily the most appropriate for the purposes of all interested researchers (e.g., for elucidating the interconnections among neural subsystems involved in anxiety reactions).

increase in the activity of either the BAS or the BIS produces a commensurate increase in NAS activity. That is, the level of NAS activity is a function of the combined activity levels of the BAS and BIS.

As shown in Fig. 1, NAS activity intensifies effects of both BAS and BIS activity. Again, BAS activity promotes the initiation of goal-directed motor behavior, whereas BIS activity makes the orienting of attention more probable. Accordingly, as NAS activity increases, motor responses are executed more vigorously, and the orienting of attention is facilitated.

Of relevance to the topic of this manuscript, these NAS-mediated processes at times have dysregulatory consequences. First, to the extent that a response is occurring at a rapid pace, it becomes less likely that an individual will be able to evaluate effectively whether the ongoing response sequence is having the desired effects, and to alter it in an appropriate manner if it is not (Newman & Wallace, 1993a; Wallace et al., 1991). Second, as discussed above, when attention is strongly attracted by a stimulus, it is unavailable to support controlled self-regulation. Therefore, as NAS activity increases, dysregulation becomes more likely.

Extraversion and Neuroticism

Again, the personality framework described herein maps Gray's BAS, BIS, and NAS constructs onto H. J. Eysenck's personality dimensions of extraversion (E) and neuroticism (N). In this model (see also Gray, 1981; Gray et al., 1983), E reflects the relative strengths of the BAS and the BIS. High-E individuals (extraverts)—who are outgoing, sociable, active, and optimistic—are considered to be BAS-dominant; that is, in these individuals the BAS is stronger than the BIS. Extraverts, then, tend to be more responsive to stimuli to which the BAS is sensitive (e.g., opportunities for reward), and are inclined to engage in BAS-mediated goal-directed behavior.

On the other hand, low-E individuals (introverts)—who are quiet, unsociable, passive, and careful—are BIS-dominant: The BIS is stronger than the BAS. Hence, these individuals are more responsive to stimuli such as potential threats and unexpected events, and are predisposed to exhibit behavioral inhibition in the presence of such stimuli (but see Wallace et al., 1991; Wallace & Newman, 1990).

Another way of stating these points is that, in the presence of stimuli that serve as inputs to the BAS, an extravert will tend to experience higher levels of BAS activity than will an introvert. In consequence, an extravert's NAS activity also tends to be greater in appetitive contexts. Conversely, because introverts tend to be more sensitive than extraverts to BIS inputs,

their resulting levels of BIS and NAS activity are higher when aversive or unexpected stimuli are present.

Others have considered the implications of individual differences associated with the BAS and the BIS (e.g., Fowles, 1980, 1988; Gray, 1981; Gray et al., 1983). We, however, have emphasized the NAS as the locus of an individual difference variable. Specifically, our model identifies N with the *reactivity* of the NAS. The high-N (neurotic) individual—who "is emotional, reacting too strongly to all sorts of stimuli" (Eysenck & Eysenck, 1975, p. 5)—is characterized by high NAS reactivity. The low-N (stable) individual, on the other hand, is less reactive and more even tempered. Consequently, the trait of neuroticism is conducive to heightened NAS activity: If all other factors are equal, neurotic individuals experience higher levels of NAS activity than do their stable counterparts.

This model thus implies that the level of NAS activity is determined not only by BAS and BIS activity levels, but also by NAS reactivity (which is reflected in N). Indeed, the level of NAS activity might be conceived as a multiplicative function of (a) the sum of the activity levels of the BAS and BIS, and (b) the reactivity of the individual's NAS. For example, in an appetitive stimulus context, the combination of extraversion (which causes appetitive stimuli to engender relatively high levels of BAS activity) and neuroticism (which reflects high NAS reactivity) induces the highest levels of NAS activity.

A second implication is that the trait of neuroticism constitutes a predisposition to experience NAS-mediated dysregulation. That is, because neuroticism is conducive to heightened NAS activity, neurotic individuals tend to execute behavioral responses more vigorously and orient more readily than do their stable counterparts. In consequence, they are more susceptible to the dysregulatory effects of these processes, which were specified above.

Experimental Studies of Individual Differences in Response Speed and the Orienting of Attention

The personality framework just described has received considerable empirical support. First, in numerous experiments (Bachorowski & Newman, 1990; Nichols & Newman, 1986; Wallace & Newman, 1990; for a review see Wallace et al., 1991), subjects who presumably experienced the highest levels of NAS activity evidenced the fastest response speeds. Specifically, neurotic extraverts were observed to respond fastest under conditions that would be expected to increase BAS activity (e.g., the opportunity to win money), whereas neurotic introverts evidenced the fastest response

speeds when BIS activity was engendered (e.g., by the threat of losing money, or response uncertainty). These results are consistent with the hypothesis that the effects of specific stimulus sensitivities derived from E (which cause the individual to be most responsive to either appetitive or aversive stimuli) are magnified by neuroticism (high NAS reactivity) to influence response speed.

In addition, we recently tested our hypothesis regarding neuroticism and the automatic orienting of attention. In our initial study (Wallace & Newman, 1996), subjects performed a visual search task, in which they searched for designated target letters in a series of five rapidly presented three-character displays. On some trials, all characters in the displays were letters. However, on others a character in one of the displays was a nonletter symbol.

Even though subjects were instructed to search only for target letters, we expected that they would automatically orient to the nonletter distractor stimuli, and hence would be less able to search for targets. This was, in fact, what we observed in our female subjects: Overall, they were less able to perform the visual search task when a distractor also was present in the display. Moreover, in females this effect was significantly more pronounced for neurotic subjects than for their stable counterparts. That is, the visual search task performance of neurotic subjects was significantly more impaired by the distractor stimuli, indicating that their attention was more strongly attracted by the distractors.[3] These results, then, provide some support for the proposal that the automatic orienting of attention is facilitated in neurotic individuals.

Data collected by other researchers also are consistent with this hypothesis. For example, studies of the latency of the P300 component of event-related brain potentials are relevant because P300 latency appears to provide an index of the tendency of a stimulus to attract attention. Support for this position has been provided by Kramer, Schneider, Fisk, and Donchin (1986), who examined the relationships between the development of the automatic attention response and the components of event-related brain potentials. Briefly, these investigators observed that as the strength of the automatic attention response increased, P300 latency decreased. That is, the stronger the tendency of a stimulus to attract attention automatically, the shorter the P300 latency.

Given this conceptualization, we would expect that neurotic subjects would evidence relatively short P300 latencies, because we presume that

[3]The predicted neuroticism effect was not evident in male subjects. However, unlike female subjects, males were not significantly affected by the distractor manipulation, making it impossible to examine whether N would magnify the extent to which distractors disrupted search performance.

the orienting of attention is facilitated in neurotic individuals. Indeed, P300 latencies in male (Pritchard, 1989) and female (Stelmack, Houlihan, & McGarry-Roberts, 1993) neurotic subjects have been observed to be shorter than those of their stable counterparts.

In sum, then, there is initial support from both behavioral and psychophysiological data for the hypothesis that the automatic orienting of attention is facilitated in neurotic subjects.

Neuroticism as a Biologically Based Individual Difference

H. J. Eysenck (1967; Eysenck & Eysenck, 1985) has emphasized that E and N are based on individual differences in the functioning of the central nervous system. He regards E as involving differences in the tonic activity level of the ascending reticular activating system (ARAS). These differences, in turn, affect cortical arousal, with introverts having higher tonic levels of cortical arousal than do extraverts.

The neurological substrate proposed for N is composed of the septum, hippocampus, cingulum, amygdala, and hypothalamus (Eysenck & Eysenck, 1985). These limbic system structures are more reactive in neurotic individuals than in stable individuals, resulting in emotional lability.

We agree that E and N are, at least in part, biologically based, but differ with respect to the hypothesized loci of these individual differences. In fact, our preferred conceptualization is just the reverse of that proposed by H. J. Eysenck. First, we concur with Gray (1981, Gray et al., 1983) that E may be conceived as based on functional differences in components of the limbic system, such as the septum and hippocampus.

With respect to the anatomical substrate of N, recall first our position that N reflects individual differences in the reactivity of the NAS. Gray (1987) has suggested that the NAS construct (and thus, in our view, N) may be identified with the ARAS, and in particular one of its constituent structures, the noradrenergic locus coeruleus (LC). Indeed, the correspondence between the *postulated* effects of NAS activity and the *observed* effects of ARAS and LC activity is rather striking.

First, consistent with the postulate that heightened NAS activity produces an increase in response vigor, electrical stimulation of the ARAS has been demonstrated to increase the speed of motor behavior (Gray, 1987).

Second, Posner and his colleagues have identified a set of central nervous system structures—including the posterior parietal lobe, midbrain areas including the superior colliculus, and the thalamic pulvinar nucleus—that subserves the orienting of attention to visual stimuli (Posner, 1988; Posner & Peterson, 1990). This *posterior attention system* system receives

extensive noradrenergic innervation from the LC (Posner & Peterson, 1990), indicating that the LC plays a substantial role in mediating the orienting of attention.

Furthermore, the LC system is an essential element of the generation of the P300 component of event-related brain potentials (Berridge, Arnsten, & Foote, 1993), and P300 latency is associated with the strength with which stimuli automatically attract attention (Kramer et al., 1986). Taken together, then, these observations provide strong support for the proposition that the orienting of attention involves activity of the noradrenergic LC system.

In sum, due to the correspondence between the properties of the NAS and the LC, it appears to be plausible to conceive of the LC as a biological substrate of N (because N is identified with the NAS). Specifically, we propose that N may be conceived physiologically as reflecting individual differences in the reactivity of the noradrenergic LC system. That is, for neurotic individuals, the reactivity of the LC—and thus that of associated structures (e.g., the posterior attention system)—tends to be greater than for stable individuals.

DISCUSSION

Let us summarize the points that we have presented. First, self-regulation often entails controlled processing, and in particular the controlled evaluation and correction of maladaptive products of automatic processes.

Second, controlled processing requires attention, and attentional resources are limited. Accordingly, when attention is directed elsewhere, it becomes less likely that sufficient attentional resources will be available to support controlled self-regulatory processes.

Third, to the extent that controlled self-regulation is compromised, the influence of automatic processes is enhanced. In other words, maladaptive cognitive, affective, and behavioral products of automatic processes (e.g., erroneous attributions or inferences, and the ensuing inappropriate affective and behavioral responses) are particularly liable to be manifested.

Fourth, individual differences influence the likelihood that self-regulatory activities will be compromised. Specifically, if other factors are equal, the automatic orienting of attention is facilitated in neurotic individuals, and they are thus less likely to have sufficient attentional resources available to support controlled self-regulatory processes. Therefore, neurotic individuals are particularly vulnerable to the impairment of controlled self-regulation.

Neuroticism as a Predisposition to Psychopathology

As noted above, neuroticism has been presumed to constitute a predisposition to experience diverse forms of psychopathology (Eysenck, 1967; Eysenck & Eysenck, 1985). We have suggested that this is because neuroticism is conducive to heightened NAS activity, and hence to the occurrence of potentially dysregulatory processes, such as increased response speed and the facilitation of the automatic orienting of attention.

Dysregulation, in turn, disrupts the ability to engage in the controlled evaluation and correction of the products of automatic processes, so the maladaptive cognitive, affective, and behavioral products of those processes tend to be manifested. These products may be considered primary determinants of the particular form of psychopathology that the individual experiences (Ingram, 1990).

The particular manifestations of automatic processes to which a given individual is prone may be considered to reflect response biases. For instance, a person may tend to generate self-deprecating interpretations of situations and events in a relatively automatic manner [e.g., a depressogenic attributional style (Abramson, Metalsky, & Alloy, 1989)]. Such a cognitive response tendency or bias would constitute a predisposition to depression. Alternatively, one might tend to have a behavioral response bias to drink excessively in response to stress. Although one response is covert and cognitive and the other is overt and behavioral, both reflect tendencies to respond automatically in characteristic ways. To the extent that controlled self-regulation is compromised, the responses associated with such biases (i.e., the products of those automatic processes) tend to be manifested.

Therefore, we argue that because neuroticism is conducive to the dysregulation of any and all maladaptive automatic response biases, and because the possible maladaptive cognitive, affective, or behavioral manifestations of such disparate biases also are quite diverse, the personality trait of neuroticism constitutes a *nonspecific predisposition* to diverse forms of psychopathology.

Conclusion

In closing, we suggest that there is substantial utility in conceiving of neuroticism as a biologically based predisposition to experience NAS-mediated dysregulatory processes, and not simply as a susceptibility to negative affect. Specifically, neuroticism proximally affects cognition (viz., controlled information processing) via facilitation of dysregulatory attentional and motor processes. Therefore, this formulation implies that the negative affect

(e.g., anxiety, depression) and psychopathology associated with neuroticism do not represent direct effects of the neural substrates of neuroticism, but rather are relatively distal consequences of more fundamental attentional and motor processes (and their effects on controlled self-regulation). As we have emphasized above, we agree that neuroticism is, indeed, associated with negative affect, as well as maladaptive behavior. We simply view our suggestions regarding the nature of neuroticism as specifying processes that may underlie these observations. The specification of those processes, in turn, may serve as a useful guide to the formulation of interventions designed to reduce the psychopathological consequences of dysregulation in general and of neuroticism in particular.

REFERENCES

Abramson, L. Y., Metalsky, G. I., & Alloy, L. B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological Review, 96*, 358-372.
Bachorowski, J., & Newman, J. P. (1990). Impulsive motor behavior: The effects of personality and goal salience. *Journal of Personality and Social Psychology, 58*, 512-518.
Bargh, J. A. (1984). Automatic and conscious processing of social information. In R. S. Wyer & T. K. Srull (Eds.), *Handbook of social cognition* (Vol. 3, pp. 1-43). Hillsdale, NJ: Erlbaum.
Bargh, J. A. (1989). Conditional automaticity: Varieties of automatic influence in social perception and cognition. In J. S. Uleman & J. A. Bargh (Eds.) *Unintended thought* (pp. 3-51). New York: Guilford Press.
Beck, A. T. (1976). *Cognitive therapy and the emotional disorders*. New York: International Universities Press.
Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression: A treatment manual*. New York: Guilford Press.
Berridge, C. W., Arnsten, A. F. T., & Foote, S. L. (1993). Noradrenergic modulation of cognitive function: Clinical implications of anatomical, electrophysiological, and behavioural studies in animal models. *Psychological Medicine, 23*, 557-564.
Buck, R. (1984). The physiological bases of nonverbal communication. In W. Waid (Ed.), *Sociophysiology* (pp. 139-162). New York: Springer-Verlag.
Carver, C. S., & Scheier, M. F. (1986). Functional and dysfunctional responses to anxiety: The interactions between expectancies and self-focused attention. In R. Schwarzer (Ed.), *Self-related cognitions in anxiety and motivation* (pp. 111-141). Hillsdale, NJ: Erlbaum.
Clark, L. A., Watson, D., & Mineka, S. (1994). Temperament, personality, and the Mood and Anxiety Disorders. *Journal of Abnormal Psychology, 103*, 103-116.
Cloninger, C. R. (1987). Neurogenetic adaptive mechanisms in alcoholism. *Science, 236*, 410-416.
Costa, P. T., & McCrae, R. R. (1980). Influence of extraversion and neuroticism on subjective well-being: Happy and unhappy people. *Journal of Personality and Social Psychology, 38*, 668-678.
Derryberry, D., & Reed, M. A. (1994). Temperament and attention: Orienting toward and away from positive and negative signals. *Journal of Personality and Social Psychology, 66*, 1128-1139.
Devine, P. G. (1989). Stereotypes and prejudice: Their automatic and controlled components. *Journal of Personality and Social Psychology, 56*, 5-18.
Dodge, K. A., & Crick, N. R. (1990). Social information-processing bases of aggressive behavior in children. *Personality and Social Psychology Bulletin, 16*, 8-22.

Dodge, K. A., & Newman, J. P. (1981). Biased decision making processes in aggressive boys. *Journal of Abnormal Psychology, 90*, 375-379.
Eaves, L., Eysenck, H. J., & Martin, N. (1989). *Genes, culture and personality: An emperical approach*. New York: Plenum Press.
Ellis, A. (1962). *Reason and emotion in psychotherapy*. Secaucus, NJ: Lyle Stuart.
Eysenck, H. J. (1967). *Biological basis of personality*. Springfield, IL: Thomas.
Eysenck, H. J., & Eysenck, M. W. (1985). *Personality and individual differences: A natural science approach*. New York: Plenum Press.
Eysenck, H. J., & Eysenck, S. B. G. (1975). *Manual of the Eysenck Personality Questionnaire*. San Diego, CA: Educational and Industrial Testing Service.
Eysenck, M. W. (1979). Anxiety, learning, and memory: A reconceptualization. *Journal of Research in Personality, 13*, 363-385.
Eysenck, M. W. (1981). Learning, memory and personality. In H. J. Eysenck (Ed.), *A model for personality* (pp. 169-209). New York: Springer-Verlag.
Fowles, D. C. (1980). The three arousal model: Implications of Gray's two-factor learning theory for heart rate, electrodermal activity, and psychopathy. *Psychophysiology, 17*, 87-104.
Fowles, D. C. (1984). Biological variables in psychopathology: A psychobiological perspective. In H. E. Adams & P. B. Sutker (Eds.), *Comprehensive handbook of psychopathology* (pp. 77-110). New York: Plenum Press.
Fowles, D. C. (1988). Psychophysiology and psychopathology: A motivational approach. *Psychophysiology, 25*, 373-391.
Gilbert, D. T. (1989). Thinking lightly about others: Automatic components of the social inference process. In J. S. Uleman & J. A. Bargh (Eds.), *Unintended thought* (pp. 189-211). New York: Guilford Press.
Gilbert, D. T., Krull, D. S., & Pelham, B. W. (1988). Of thoughts unspoken: Social inference and the self-regulation of behavior. *Journal of Personality and Social Psychology, 55*, 685-694.
Gilbert, D. T., Pelham, B. W., & Krull, D. S. (1988). On cognitive busyness: When person perceivers meet persons perceived. *Journal of Personality and Social Psychology, 54*, 733-740.
Gray, J. A. (1975). *Elements of a two-process theory of learning*. London: Academic Press.
Gray, J. A. (1981). A critique of Eysenck's theory of personality. In H. J. Eysenck (Ed.), *A model for personality* (pp. 246-276). New York: Springer-Verlag.
Gray, J. A. (1982a). *The neuropsychology of anxiety*. New York: Oxford University Press.
Gray, J. A. (1982b). Precis of the neuropsychology of anxiety. *The Behavioral and Brain Sciences, 5*, 469-534.
Gray, J. A. (1987). *The psychology of fear and stress* (2nd ed.). New York: Cambridge University Press.
Gray, J. A. (1991). Neural systems, emotion and personality. In J. Madden IV (Ed.), *Neurobiology of learning, emotion and affect* (pp. 273-306). New York: Raven Press.
Gray, J. A., Owen, S., Davis, N., & Tsaltas, E. (1983). Psychological and physiological relations between anxiety and impulsivity. In M. Zuckerman (Ed.), *Biological bases of sensation seeking, impulsivity, and anxiety* (pp. 189-217). Hillsdale, NJ: Erlbaum.
Gray, J. A., & Smith, P. T. (1969). An arousal-decision model for partial reinforcement and discrimination learning. In R. Gilbert & N. S. Sutherland (Eds.), *Animal discrimination learning* (pp. 243-272). New York: Academic Press.
Hollon, S. D., & Garber, J. (1988). Cognitive therapy. In L. Y. Abramson (Ed.), *Social cognition and clinical psychology: A synthesis* (pp. 204-252). New York: Guilford Press.
Hollon, S. D., & Garber, J. (1990). Cognitive therapy for depression: A social cognitive perspective. *Personality and Social Psychology Bulletin, 16*, 58-73.
Ingram, R. E. (1990). Self-focused attention in clinical disorders: Review and a conceptual model. *Psychological Review, 107*, 156-176.
Kanfer, F. H., & Gaelick, L. (1986). Self-management methods. In F. H. Kanfer & A. P. Goldstein (Eds.), *Helping people change: A textbook of methods* (3rd ed., pp. 283-345). New York: Pergamon Press.

Kirschenbaum, D. S. (1987). Self-regulatory failure: A review with clinical implications. *Clinical Psychology Review, 7*, 77-104.
Kramer, A., Schneider, W., Fisk, A., & Donchin, E. (1986). The effects of practice and task structure on components of the event-related brain potential. *Psychophysiology, 23*, 33-47.
Larsen, R. J., & Ketelaar, T. (1989). Extraversion, neuroticism and susceptibility to positive and negative mood induction procedures. *Personality and Individual Differences, 10*, 1221-1228.
Marlatt, G. A., & Gordon, J. R. (1985). *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors*. New York: Guilford Press.
McCrae, R. R., & Costa, P. T. (1987). Validation of the five-factor model of personality across instruments and observers. *Journal of Personality and Social Psychology, 52*, 81-90.
Newman, J. P. (1987). Reaction to punishment in extraverts and psychopaths: Implications for the impulsive behavior of disinhibited individuals. *Journal of Research in Personality, 21*, 464-485.
Newman, J. P., & Wallace, J. F. (1993a). Diverse pathways to deficient self-regulation: Implications for disinhibitory psychopathology in children. *Clinical Psychology Review, 13*, 699-720.
Newman, J. P., & Wallace, J. F. (1993b). Three pathways to impulsive behavior: Implications for violence and aggression. In L. Klose (Ed.), *Proceedings of the Fourth Symposium on Violence and Aggression* (pp. 95-113). Saskatoon, Saskatchewan, Canada: Extension Division, University of Saskatchewan.
Newman, J. P., Wallace, J. F., Strauman, T. J, Skolaski, R. L., Orelind, K. M., Mattek, P. W., Elder, K. A., & McNeely, J. (1993). Effects of motivationally significant stimuli on the regulation of dominant responses. *Journal of Personality and Social Psychology, 65*, 165-175.
Nichols, S. L., & Newman, J. P. (1986). Effects of punishment on response latency in extraverts. *Journal of Personality and Social Psychology, 50*, 624-630.
O'Boyle, M., & Holzer, C. (1992). DSM-III-R personality disorders and Eysenck's personality dimensions. *Personality and Individual Differences, 13*, 1157-1159.
Patterson, C. M., & Newman, J. P. (1993). Reflectivity and learning from aversive events: Toward a psychological mechanism for the syndromes of disinhibition. *Psychological Review, 100*, 716-736.
Pihl, R. O., Peterson, J., & Finn, P. (1990). An heuristic model for the inherited predisposition to alcoholism. *Psychology of Addictive Behaviors, 4*, 12-25.
Posner, M. I. (1988). Structures and functions of selective attention. In T. Bolls & B. K. Bryant (Eds.), *Clinical neuropsychology and brain function: Research, measurement, and practice* (pp. 173-202). Washington, DC: American Psychological Association.
Posner, M. I., & Peterson, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience, 13*, 25-42.
Posner, M. I., & Snyder, C. R. R. (1975). Attention and cognitive control. In R. L. Solso (Ed.), *Information processing and cognition: The Loyola Symposium* (pp. 55-85). Hillsdale, NJ: Erlbaum.
Pritchard, W. S. (1989). P300 and EPQ/STPI personality traits. *Personality and Individual Differences, 10*, 15-24.
Pyszczynski, T., & Greenberg, J. (1987). Self-regulatory perseveration and the depressive self-focusing style: A self-awareness theory of reactive depression. *Psychological Review, 102*, 122-138.
Schneider, W., Dumais, S. T., & Shiffrin, R. M. (1984). Automatic and control processing and attention. In R. Parasuraman & D. R. Davies (Eds.), *Varieties of attention* (pp. 1-27). New York: Academic Press.
Schneider, W., & Shiffrin, R. M. (1977). Controlled and automatic human information processing: I. Detection, search, and attention. *Psychological Review, 84*, 1-66.
Shapiro, D. (1965). *Neurotic styles*. New York: Basic Books.
Sher, K. J., & Trull, T. J. (1994). Personality and disinhibitory psychopathology: Alcoholism and antisocial personality disorder. *Journal of Abnormal Psychology, 103*, 92-102.

Shiffrin, R. M. (1988). Attention. In R. C. Atkinson, R. J. Herrnstein, G. Lindzey, & R. D. Luce (Eds.), *Stevens' handbook of experimental psychology* (pp. 739-811). New York: Wiley.

Shiffrin, R. M., & Schneider, W. (1977). Controlled and automatic human information processing: II. Perceptual learning, automatic attending, and a general theory. *Psychological Review, 84*, 127-190.

Steele, C. M, & Josephs, R. A. (1988). Drinking your troubles away II: An attention-allocation model of alcohol's effect on psychological stress. *Journal of Abnormal Psychology, 97*, 196-205.

Stelmack, R. M., Houlihan, M., & McGarry-Roberts, P. A. (1993). Personality, reaction time, and event-related potentials. *Journal of Personality and Social Psychology, 65*, 399-409.

Tarter, R. E. (1978). Etiology of alcoholism: Interdisciplinary integration. In P. E. Nathan, G. A. Marlatt, & T. Loberg (Eds.), *Alcoholism: New directions in behavioral research and treatment* (pp. 41-70). New York: Plenum Press.

Tellegen, A. (1985). Structures of mood and personality and their relevance to assessing anxiety, with an emphasis on self-report. In A. H. Tuma & J. Maser (Eds.), *Anxiety and the anxiety disorders* (pp. 681-706). Hillsdale, NJ: Erlbaum.

Tiffany, S. T. (1990). A cognitive model of drug urges and drug-use behavior: Role of automatic and nonautomatic processes. *Psychological Review, 97*, 147-168.

Walker, J. L., Lahey, B. B., Russo, M. F., Frick, P. J., Christ, A. G., McBurnett, K., Loeber, R., Stouthamer-Loeber, M., & Green, S. M. (1991). Anxiety, inhibition, and conduct disorder in children: I. Relations to social impairment. *Journal of the American Academy of Child and Adolescent Psychiatry, 30*, 187-191.

Wallace, J. F., Bachorowski, J., & Newman, J. P. (1991). Failures of response modulation: Impulsive behavior in anxious and impulsive individuals. *Journal of Research in Personality, 25*, 23-44.

Wallace, J. F., & Newman, J. P. (1990). Differential effects of reward and punishment cues on response speed in anxious and impulsive individuals. *Personality and Individual Differences, 11*, 999-1009.

Wallace, J. F., & Newman, J. P. (1996). *Neuroticism and the automatic orienting of attention.* Submitted for publication.

Watson, D., & Clark, L. A. (1984). Negative affectivity: The disposition to experience aversive emotional states. *Psychological Bulletin, 96*, 465-490.

Widiger, T. A., & Costa, P. T. (1994). Personality and personality disorders. *Journal of Abnormal Psychology, 103*, 78-91.

Zuckerman, M., Kuhlman, D. M., Joireman, J., Teta, P., & Kraft, M. (1993). A comparison of three structural models for personality: The big three, the big five, and the alternative five. *Journal of Personality and Social Psychology, 65*, 757-768.

Cognitive Therapy and Research, Vol. 21, No. 2, 1997, pp. 157-184

Cognitive Processing Style, Mood, and Immune Function Following HIV Seropositivity Notification[1]

Susan K. Lutgendorf, Michael H. Antoni,[2] Gail Ironson, Nancy Klimas, Mary Ann Fletcher, and Neil Schneiderman

The relations among cognitive processing of stressful emotional material, mood, and immune functioning were examined in 30 asymptomatic gay men during the stress of HIV-1 seropositivity notification. We administered the Impact of Event Scale, and immunological and mood data were collected 5 weeks before, 1 week after, and 5 weeks after notification of HIV-1 seropositivity. Consistent elevations of avoidance or intrusion levels during the study period did not predict distress at 5 weeks postdiagnosis; rather, increased levels of both avoidance and intrusion over the study period were related to significantly greater anxiety, depression, and total mood disturbance by the end of the study. Increased intrusion, but not avoidance, during the period from study entry to 1-week postdiagnosis was related to higher levels of distress 1 week after HIV serostatus notification. In contrast, in the weeks following serostatus notification, increased avoidance predicted worse mood outcomes. Increased avoidance over the 10-week study period significantly predicted poorer proliferative response to pokeweed mitogen as well as trends toward lower T-helper-inducer lymphocyte (CD4+) percentages. Increased intrusion over this time period significantly predicted lower CD4+ percentages, controlling statistically for baselines. Mood change during the 10-week study

[1]We would like to acknowledge the contributions of all of the graduate students, technicians, and support staff who were responsible for the recruitment of subjects, data collection and processing, and conduct of this study. We would especially like to thank Sharon August, Lane Baggett, John Simoneau, Lynn Helder, David Goldstein, and Arthur LaPerriere. Most of all we wish to acknowledge the contributions of the men who donated their time and energy in participating in this study in the hope of generating knowledge on the factors associated with the course of HIV disease.
This work was supported by National Institute of Mental Health (NIMH) grants MH42455, T32-MH18917, and P01-MH49548.

[2]Address all correspondence to Michael H. Antoni, University of Miami, Department of Psychology, P.O. Box 248185, Coral Gables, Florida 33124-2070.

0147-5916/97/0400-0157$12.50/0 © 1997 Plenum Publishing Corporation