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Affect Experience and Expression and Headache

By

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ABSTRACT

This study examined the extent to which emotional experience and expression covaried with headache susceptibility. Subjects were obtained from a university population. Both within-headache and between group (headache vs. headache-free) comparisons were undertaken. The former entailed comparing 1) episodic headache sufferers with those that experienced continuous or near-continuous pain, and 2) vascular subjects with tension headache sufferers. In terms of content of emotional experience, measures reflecting anger, anxiety, and intensity of affect experience were employed. Emotional inhibition was assessed by means of the Anger Expression Scale. Subjects were required to monitor daily headache activity over a two-week period (Headache Frequency Record; HFR). Daily headache hours derived from the HFR were used to classify subjects as either continuous (greater than 10 hours/day) or episodic (less than 10 hours/day). A headache symptom questionnaire was employed to determine diagnostic status; subjects with vascular headache conditions including migraine, combined headache, and cluster were grouped together.

Headache sufferers in general exhibited higher scores on measures of negative emotionality. Anxiety proved to be the most significant factor, however, elevations in trait anger were also noted. A tendancy to inhibit overt

anger expression in situations involving peers was also observed. Measures of emotionality did not differentiate more severe headache sufferers from those with less severe conditions. Rather, degree of anger suppression (peer and family) proved to be an important discriminator. Subjects with vascular-type headaches and/or continuous pain showed marked deficits in anger expression in both peer and family contexts, whereas less severe subjects suppressed anger only in the former. These data suggest that a more pervasive deficit in emotional expression is characteristic of severe headache sufferers, regardless of typology.

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To my family,

for their love, support,

and patience - God knows

they needed it!

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INTRODUCTION

The purpose of the present study was to examine the relationship between emotional experience/expression and headache activity. Although recent efforts to link emotionality to headache have been promising, confusion exists as to the precise nature of emotional experience among headache sufferers. Two issues are of particular relevance to the present study. The first issue involves the extent to which heightened emotionality characterizes university students with problem headache. Some question exists as to the validity of claims of heightened emotionality among non-clinic headache sufferers as much of the empirical support comes from studies utilizing patient populations (eg. Rojhan and Gerhards, 1986; Blanchard, Andrasik & Arena, 1984; Andrasik, Blanchard, Arena, Teders, Teevan & Rodichok, 1982). A second issue involves within-headache variability in emotional experience and expression. While headache sufferers in general may be subject to high levels of anger and anxiety, there are some data pointing to a distinct subset of headache sufferers who are not highly emotional and, in fact, are characterized by reduced mood variability and constricted affect (Demjen, 1986; Bakal and Demjen, 1984; Harrigan, Kues, Ricks & Smith, 1984).

These individuals have been variously described as continuous, near-continuous or "chronic daily" headache sufferers (Saper, 1986; Demjen, 1986; Drummond, 1985). One of the goals of the present research was to determine if such individuals could be found within a relatively young sample of headache sufferers, and if so, whether they were psychologically similar or dissimilar to their peers with episodic headache.

An examination of between group (headache vs. headache-free) and within headache differences in emotional experience and expression may clarify some of the confusion surrounding psychological factors in headache. In terms of content of emotional experience, both anxiety and anger were examined, with a particular emphasis on characteristic modes of expressing anger. Anger and anxiety were selected as these emotions have been consistently implicated in headache evolution and maintenance.

Historical Perspective

Current psychobiologic formulations of headache pathogenesis reflect an empirical reinterest in the concepts put forth by the early psychosomatic theorists. Flanders Dunbar (1943) and Alexander (1943) were among the first to postulate an association between personality/emotional styles and illness. Alexander proposed that specific personality traits, in conjunction

with a physiological predisposition or "organ weakness" predisposed persons to develop specific psychosomatic disorders. Hypertension, for example, was viewed as the physiological expression of unrelieved emotional tension, in this case suppressed anger. In a variant of this hypothesis, Harold Wolff (1937) postulated that while constitutional factors (ie. genetic disposition) render individuals susceptible to migraine, emotional reactions actually precipitate headache attacks. He held that specific personality traits primed migraineurs to experience sustained pernicious emotional states that ultimately provoked migraine attacks. In his classic paper, Wolff (1937) outlined features characteristic of the "migraine personality." These included a preoccupation with success, obsessiveness, orderliness, repressed hostility and an overconcern with time and efficiency.

Following the publication of Wolff's clinical observations, numerous efforts were made to establish the significance and/or existence of pathological personality traits in both migraine and tension headache sufferers (Blanchard et al, 1984; Harrison, 1975; Bakal, 1975). Growing disenchantment with Wolff's early formulations emerged in the late sixties and seventies as efforts to empirically isolate "migraine" personality characteristics failed to produce consistent results. While some studies supported the clinical observational literature (Paulley

and Haskell, 1975; Furmanski, 1952; Bihldroff, King & Parnes, 1971), others argued against unique personality traits (Friedmann, von Storch & Merritt, 1954; Lucus, 1971), and still others identified traits not typically associated with the headache personality (Price and Blackwell, 1980). In reviewing the personality research of the last few decades both Harrison and Bakal concluded that the relationship between personality and headache is typically low in magnitude and nonspecific in nature.

The failure to isolate personality traits unique to headache sufferers has not lead to a disinterest in the psychological components of headache. Current research reflects a shift in focus from the specific personality dimensions emphasized in early research to an evaluation of the role of emotion in headache onset and maintenance. Two factors in particular have been implicated in the genesis of headache conditions: emotionality or emotional over-reactivity and inhibited emotional expression.

Emotional Experience and Headache

Emotionality has been defined as the susceptibility to become easily and often intensely distressed; high levels of trait anxiety and/or hostility are indicative of emotionality (Buss and Plomin, 1975). Numerous studies attest to the presence of emotionality among persons prone to recurrent headache. Elevations in trait anxiety, for example, have been observed consistently in episodic

headache sufferers drawn from both clinic (Blanchard et al, 1984; Andrasik et al, 1982) and community samples (Jones and Page, 1986; Price and Blackwell, 1980; Andrasik and Holroyd, 1980). High levels of hostility have also been reported in several studies. Henryk-Gutt and Rees (1973) found significant elevations in hostility (as measured by Buss-Durkhee Hostility Guilt Inventory) among migraine sufferers attending a treatment clinic. Blaszczynski (1983) also reported higher hostility among migraine and tension headache sufferers as compared to controls.

Studies examining "subjective stress sensitivity" offer additional support for heightened emotionality. In a recent study, Rojahn and Gerhards (1986) had subjects rate the extent to which they reacted "emotionally" to a variety of physical and psychosocial stressors. Migraine sufferers achieved significantly higher "general stress sensitivity" scores than control subjects. Another study examining perceived severity of life events revealed that headache sufferers rate stressful life events as more distressing and disturbing than do headache-free controls (Holm, Holroyd, Hursey & Penzien, 1986). It is important to note that these observations do not reflect differential exposure to stressful life events; no differences in exposure to stressful events have been found between headache sufferers and headache-free controls (Andrasik et al, 1982; Andrasik and Holroyd, 1980; Ivernizzi and

Sacchetti, 1985). Taken together, these observations support the prevailing view of the typical headache sufferer as an emotionally over-reactive stress-sensitive individual.

Emotional Experience and Headache Severity

While increased emotionality has been reported fairly consistently in studies comparing headache sufferers in general with headache-free controls, the relationship between emotionality and within-headache variability is less obvious. A number of studies have pointed to a continuum of psychological disturbance within headache samples, ranging from little or no pathology among migraine and cluster subjects to more severe disturbance among tension and combined headache sufferers (Blanchard et al, 1984; Andrasik et al, 1982; Kudrow and Sutkus, 1979). Headache frequency has been cited as a possible determinant of degree of pathology as subjects with tension or combined headache typically report greater headache activity. In the Blanchard et al (1984) study tension headache sufferers reported an average of only 1.96 pain-free days per week as compared to 3.69 headache-free days among the less psychologically distressed migraine group.

While these studies appear to point to a concomitant increase in emotionality with increasing headache severity, the use of MMPI scores (in each study) to define emotional disturbance among headache sufferers is questionable. The

MMPI was devised for use with psychiatric patients, presumably free of chronic pain disorders. As a result, several of the subscales, hypochondriasis and hysteria in particular, consist largely of items reflecting somatic complaints. Rather than reflecting pathological absorption with bodily symptoms, responses to these MMPI subscales may actually represent an accurate depiction of physical symptoms associated with a chronic pain disorder. Headache sufferers that experience more frequent headache activity may therefore yield results that appear more pathological simply because they report more symptoms.

Moreover, at least two additional studies, employing measures other than MMPI subscales, have failed to demonstrate any association between headache severity and emotionality. Jones and Page (1986) examined the relationship between anxiety and perceived severity of headache among subjects experiencing weekly headache. Although no information was obtained regarding actual headache frequency, perceptions of headache severity likely reflected the frequency with which subjects experienced headache. No association was found between perceived severity of headache and trait anxiety. Similarily, Demjen (1986) found no relationship between anxiety and average daily headache hours, another indice of headache severity. Harrigan et al (1984) reported lower mood variability among subjects with high frequency headache than was evident

among normal controls. The authors suggested that constricted affect experience and emotional over-control may actually characterize these individuals.

Conflicting reports of the presence/absence of emotionality among headache sufferers may reflect not only the use of different assessment instruments but also the utilization of subjects with significantly different headache problems. A large percentage of subjects in the Demjen study, for example, experienced pain that was of a continuous or near-continuous nature. The subjects in the other studies (eg. Blanchard et al, 1984; Andrasik et al, 1982) were episodic headache sufferers. Research examining patients with various unremitting pain conditions has shown that these individuals typically deny both emotional difficulties in general, and the possibility that psychological factors may contribute to illness (Blumer and Heilbronn, 1982; Pilowsky and Spence, 1976). DeGood et al (1985) further noted that chronic pain patients tend to endorse somatic and not cognitive anxiety descriptors, a pattern that is reversed among healthy controls. (DeGood, Buckelew & Tait, 1985). The authors suggested that for these patients, the ability to communicate private affective distress may be limited almost entirely to the somatic sphere.

Similar characteristics have been found to differentiate continuous headache sufferers from those that

experience episodic headache. Demjen and Bakal (1981) noted that subjects with continuous pain exhibited higher scores on measures of denial of emotional difficulties, and viewed their disorder in somatic as opposed to psychological terms. While the continuous headache sufferers resembled the intractable pain patients in the Pilowsky and Spence (1976) study, episodic headache sufferers had a stronger psychological focus for their disorder and a greater willingness to discuss emotional difficulties. Poor responsivity to treatment has also been observed among daily headache sufferers, and attributed, at least in part, to an unwillingness to either acknowledge or confront personal problems (Demjen, Bakal & Kaganov, 1984; Featherstone and Beitman, 1984).

These observations are consistent with recent data pointing to an association between the alexithymic coping style and headache severity. Alexithymia has been described as a cluster of cognitive and behavioral deficits related to the experience and expression of affect (Sifneos, 1973). Restricted affect experience and inhibited emotional expression are central to this construct. In the Demjen (1986) study, the presence of alexithymic characteristics (as measured by the Schalling-Sifneos Personality Scale) correlated positively with daily headache hours whereas other measures, including trait anxiety, did not. Repressive or self-deceptive

tendencies were further noted among subjects with severe headache activity. Collectively, these studies suggest that relative to episodic headache sufferers, subjects with continuous headache may, in fact, be less emotional, or less willing to acknowledge personal distress to self or others. Lower scores on measures of anxiety and anger would therefore be expected among these individuals.

Emotional Expression

The notion that headache activity and inhibited emotional expression may be related is consistent with other theoretical and empirical observations in the psychosomatic literature. According to the inhibition-disease framework (Pennebaker, Hughes & O'Heeron, 1987), the act of inhibiting overt expression of emotion is physiologically stressful. This model postulates that since brief instances of emotional inhibition are associated with increased physiological activity, long-term inhibition should correlate with higher overall autonomic and somatic levels (eg. chronic muscle tension) and the incidence of stress-related disorders. The assumption implicit in this model is that sustained elevations in physiological activity render one susceptible to psychosomatic symptom development.

Considerable support exists for an association between short-term inhibition of emotional expression and increased physiological reactivity (Buck, 1984; Anderson, 1980; Waid

and Orne, 1981). The long-term consequences of chronic emotional inhibition are less clear, however, heightened tonic and phasic physiologic activity have been reported among subjects with repressive or non-expressive coping styles. Weinberger and colleagues (1979) for example, found higher forehead muscle tension and skin resistance responses among repressive subjects, during a phrase association task, than was evidenced by either low anxious or high anxious persons (Weinberger, Schwartz & Davidson, 1979). Martin and Pihl (1986) similarily reported increased levels of tonic physiologic activity, and a dissociation between subjective and physiological stress responses among subjects high in alexithymic characteristics. In a series of experiments designed to assess expressivity and its physiological correlates, Buck (1984) concluded that subjects who report actively inhibiting emotional expression evidence higher skin conductance levels than do expressive subjects.

Investigations of non-expressive coping styles among various illness groups provide further support for the deleterious consequences of emotional inhibition. Jensen (1987), for example, found higher recurrence of breast cancer among women classified as repressors. McClelland (1979) also reported higher blood pressure among suppressors. These observations suggest that the tendency to inhibit emotional expression may contribute to illness

susceptibility.

Anger Expression/Suppression

Recent efforts to examine the association between emotional inhibition and illness susceptibility have focused on specific patterns of emotional expression (eg. anger-in/anger-out) rather than dispositional measures of denial, repression, or alexithymia. Non-expressiveness in this context refers to the tendency to characteristically inhibit overt anger expression; that is, to not express felt anger towards the source of frustration. What is being assessed in these studies is not repression or denial per se, but rather suppression or inhibition of overt emotional expression.

Anger suppression, in particular, has been linked to psychosomatic symptom development and exacerbation. Dembroski and associates (1985) reported a significant relationship between anger-in and severity of coronary heart disease (Dembroski, MacDougall, Williams, Haney & Blumenthal, 1985). Anger-in has been conceptualized as a reluctance or inability, across a variety of situations, to overtly express feelings of anger/hostility (Dembroski et al, 1985). Subjects high on the anger-in dimension manifested more severe heart disease. An association between anger-in and severity of hypertension among borderline hypertensives has also been demonstrated. Subjects with more severe hypertension evidenced higher

levels of anger suppression than less severe hypertensives (Schneider, Egan, Johnson, Drobny & Julius, 1986). In this particular study the Anger Expression Scale (Speilberger, Johnson, Russell, Crane, Jacobs & Worden, 1984) was used to assess characteristic patterns of anger expression (ie. overt expression/inhibition).

Research indicates that the tendency to inhibit overt anger expression may also characterize chronic headache sufferers. Grothgar and Scholz (1987) noted that in comparison to controls, recurrent migraine sufferers exhibited significantly less overt anger behavior during an anger induction procedure, despite increased physiological reactivity. The migraine group showed a much higher increase in pulse pressure during the anger-provoking condition as compared to both healthy controls and pain patients. This observation is consistent with the inhibition/disease framework in that inhibition of anger expression has been shown to account, on the physiological level, for the persistence of stressor-induced activation states, whereas openly expressed anger reduces these states (van Egeren, Abelson, Thorton, 1978).

Similar deficits in overt emotional expression have been reported among subjects with tension headache. Traue and colleagues (1985) compared compared healthy controls and tension headache sufferers' physiological and behavioral responses to a situational stressor during a

symptom-free state. Headache subjects exhibited both reduced emotional expressiveness and increased EMG levels in both frontalis and trapezius muscles (Traue, Gottwald, Henderson & Bakal, 1985). To date, none of these studies have determined the extent to which anger suppression may be associated with headache severity. The present study investigated this possibility.

In summary, studies have demonstrated both emotional over-reactivity and restricted affect among subjects with recurrent headache. Suppression of overt anger expression has also been implicated in headache development and maintenance. The purpose of this study was to isolate the headache parameters that determine the nature of these varied relationships. Both within headache variability and between group differences (headache vs. headache-free) were examined. The primary within headache dimension was based on daily headache hours: subjects reporting episodic headache were compared with those experiencing continuous, or near-continuous pain. Comparisons based on diagnostic status were also undertaken as vascular headache has traditionally been viewed as more severe than tension headache.

Figure 1 illustrates the hypothesized relationships between the psychological variables and the group contrasts. Relative to headache-free controls, subjects with recurrent headache were expected to report increased

levels of negative affect, specifically anger and anxiety. The intensity with which these individuals experienced emotion was also expected to be higher, as was the degree to which anger expression was actively inhibited. Relative to episodic headache sufferers, the continuous headache sufferers were expected to report infrequent arousal of negative affect, less intense emotional experience, and increased anger suppression. Since previous research has demonstrated self-deceptive tendencies among these individuals, it seemed unlikely that intense negative emotionality would be acknowledged on self-report measures.

A particular strength of this study lay in its assessment of relatively young headache sufferers who were not self-selected (eg. selected on the basis of having sought medical attention). Subjects in the present study were solicited by means of a survey questionnaire, and may, therefore, be more representative of headache sufferers in general.

Linear structural relations analysis (LISREL) was used to examine between and within group differences in emotional experience and expression. Figure 1 specifies the hypothesized relationships between the four exogenous/independent latent constructs (Anxiety, Anger, Anger Suppression, Emotional Intensity) and the two endogenous/dependent latent constructs (group contrasts). LISREL incorporates both a structural model and a

measurement model. The former specifies the degree of association among exogenous latent variables (eg. Anxiety/Anger), and between exogenous and endogenous latent variables (eg. Anxiety and Group Contrasts). As the arrows in Figure 1 indicate, an association was expected between each of the exogenous latent variables and the group contrasts. In addition to specifying structural relations, LISREL also examines the degree of relationship between each theoretical construct (eg. Anxiety) and the observable indicators of that construct (eg. Trait Anxiety Inventory Scores).

The degree to which the proposed model is able to replicate the original patterns in the data can be determined by an examination of three measures: the overall chi-square statistic, the goodness of fit index, and the root mean square. The program also provides T-Values for each of the specified parameters, and indicates where improvements could be made to an initially poor fitting model (residuals, modification indices, etc.).

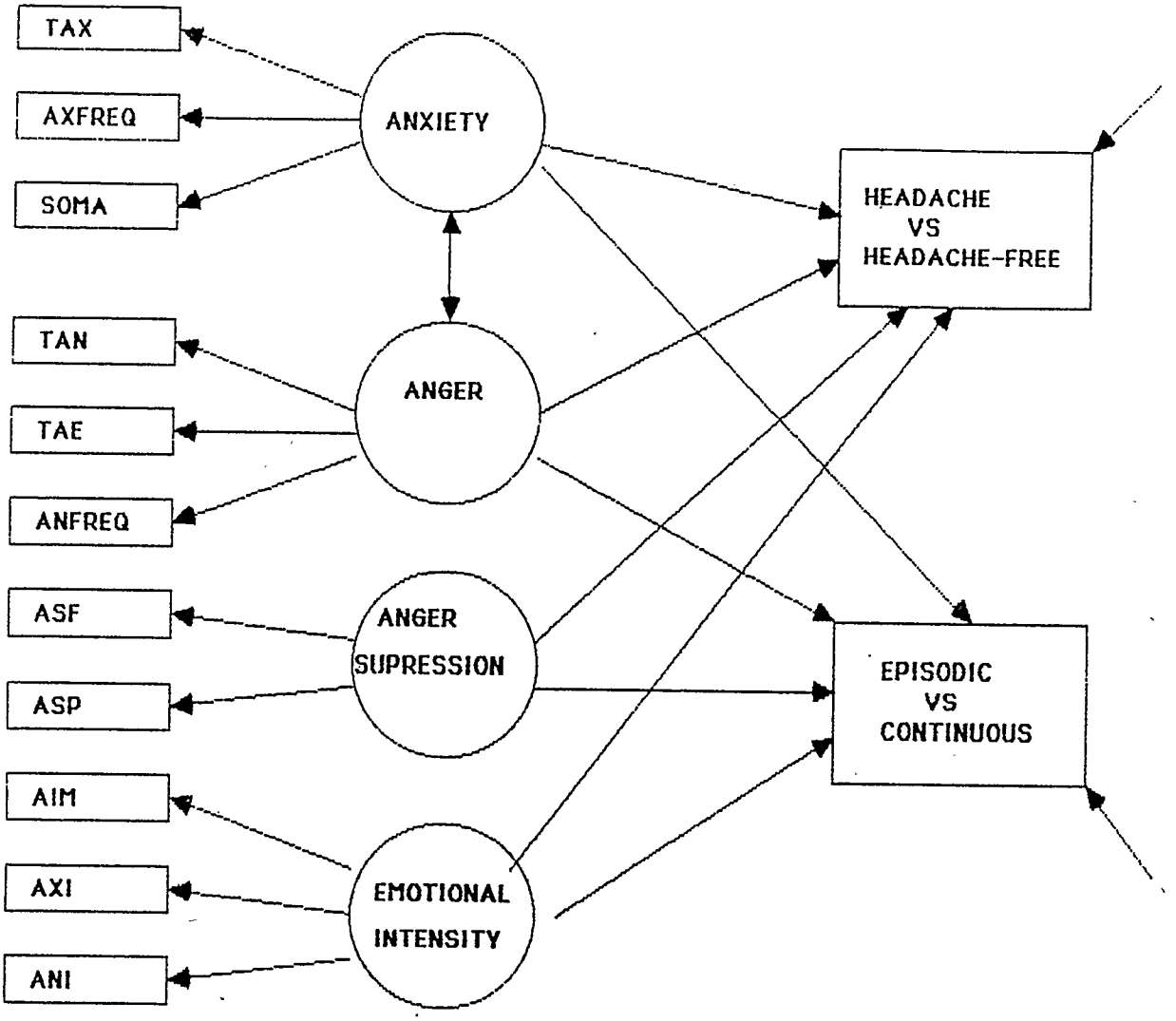

Figure 1. Hypothesized relationships between latent variables and group contrasts. TAX: Trait Anxiety; AXFREQ: Anxiety Frequency; SOMA: Somatic Anxiety; TAN: Trait Anger; TAE: Total Anger Expressed; ANFREQ: Anger Frequency; ASF: Anger Suppression-Family; ASP: Anger Suppression-Peers; AIM: Affect Intensity; AXI: Anxiety Intensity; ANI: Anger intensity.

METHOD

Subjects.

Forty recurrent headache sufferers and forty headache-free controls were recruited from a sample of 497 university students who completed a headache screening questionnaire (see Appendix J). Individuals who reported regularily (minimum six months duration) experiencing at least three headaches per week were identified as recurrent headache sufferers, and invited to participate in the study. Students that reported experiencing less than one headache per month were identified for inclusion in the control group.

Measures

Headache Activity

Headache Symptom Questionnaire (HSQ; Epstein and Abel, 1977).

The HSQ includes 16 headache symptoms that are considered relevant to the diagnosis of migraine, combined migraine-muscle contraction, muscle-contraction, and cluster headache. Each item is scored from 0 to 4 (never, infrequently, sometimes, usually, always). When used as a brief 5-minute assessment instrument approximately 70% of headache sufferers are correctly classified (Arena, Blanchard, Andrasik & Dudek, 1982). Information pertaining to medication usage was also obtained from the HSQ.

Headache Frequency Record (HFR; Bakal and Kaganov, 1976).

The HFR is a self-monitoring device (diary) that

permits the daily recording of headache activity. Daily headache hours (HOURS) were derived from the HFR by summing hours across the two week period and dividing by 14. A composite measure, headache severity (SEVERITY), was also derived by multiplying intensity by hours of headache. Daily headache hours and/or severity are frequently used in the literature as measures of the overall severity of the condition. Additional measures computed from the HFR included: 1) average length of individual headache attacks (LA), 2) frequency of headaches (total number of headaches reported during the two week period; FREQUENCY), and 3) number of headache-present days (HDAYS). Previous research has demonstrated an inverse association between daily headache hours obtained from the HFR and response to cognitive behavioral intervention (Bakal, Demjen & Kaganov, 1981). A positive relationship between headache hours and musculoskeletal and vascular symptoms has also been noted (Demjen, 1986).

Headache history was determined by assessing subjects response to the following questions: 1. the age at which their headaches first became a problem, 2. whether or not they had seen a physician and/or neurologist regarding headache, and 3. if they had received any form of treatment for headache. Finally, subjects were asked to describe any factors that they believed were associated with onset of headache attacks.

Other Medical Problems

In order to ensure that groups did not differ on any health variables other than headache, a checklist of disorders that are thought to have a "psychosomatic" component was derived. Lipowski (1987) states that the term "psychosomatic" refers, or is related to, "the inseparability and interdependence of psychosocial and biologic (physiologic and somatic) aspects of humankind." While it is recognized that a host of conditions meet this definition, only the most commonly cited classic psychosomatic conditions were included: asthma, dermatitis, gastrointestinal conditions (eg. irritable bowel, ulcerative colitis), duodenal or peptic ulcer, rheumatoid arthritis, hypertension and chronic pain conditions. Subjects were also asked if they currently suffered from any neurological condition, or other medical illness. Other medical conditions that were of a psychosomatic nature (eg. sensitive stomach) were added to those checked on the questionnaire to yield a total score on psychosomatic illnesses (one individual indicated problems with a sensitive stomach). Other medical conditions cited were: hearing impairment (n=1), mitral valve prolapse (n=2), and allergies (n=2).

The Perceived Social Support Scale
(PSSS; Blumenthal, Burg, Barefoot, Williams, Haney & Zimet 1987)

The PSSS is a 24 item scale employing a 5-point Likert-type format. Eight items comprise each of three subscales: significant other, peer, and family support. The author's report Cronbach's alpha's of .88, .91, .87, and .85 for the total, significant other, family, and peer subscales respectively. The test-retest reliabilities (over a 2-3 month period) for the total and three subscales were .85, .72, .85, and .75, respectively.

Trait Anxiety Inventory (TAI; Speilberger, 1966).

This 20 item self-report measure assesses the trait component of anxiety. The subject is asked to indicate how he/she generally feels, and each item is rated for frequency using a 4-point Likert scale. Test-retest reliabilities range from .73 to .86, and several studies have demonstrated high concurrent validity with other measures of trait anxiety (eg. Spielberger, Gorsuch, and Lushene, 1970; Cattel and Scheier, 1961)

Cognitive Somatic Anxiety Questionaire
(CSAQ; Schwartz, Davidson & Goleman, 1978).

The CSAQ is a 14-item self-report measure in which the subject indicates, on a five-point scale, the extent to which they agree or disagree with each of the items. Seven of the items reflect the somatic expression of anxiety, and seven reflect anxiety that is expressed cognitively. The

correlation between subscales is .40, and the scale correlates signficantly with the trait form of the State-Trait Anxiety Inventory r=.67 (cognitive subscale) and r=.40 (somatic subscale).

Trait Anger Inventory (TAI; Speilberger, Jacobs, Russell & Crane 1983).

The Trait Anger Inventory is a 10 item self-report measure of subjects characteristic levels of anger. The authors report an internal consistency reliability coefficient of .87 for the scale as well as concurrent validity with other measures of anger.

College Events Inventory (CEI; Lopez and Thurman, 1987).

The CEI is a 15 item self-report questionnaire that assesses the frequency and intensity with which subjects' respond (with anger) to mildly aversive events typically associated with a university environment (eg. losing notes, parking problems). This measure was included because the author felt that subjects would be less apt to respond in a socially desirable manner to the items present in the CEI (as compared to the Trait Anger and Anxiety Inventories). Subjects are required to respond only to items that have elicited anger by rating on separate 7-point Likert scales a) the frequency with which that event occurs for them, and b) the intensity of anger that is usually provoked by the event. Because of the intrinsic relationship between anger and anxiety (see Spielberger et al, 1983), a second copy of

the CEI was created in which subjects were asked to respond only to items that elicited anxiety. Cronbach's alpha's for the anger frequency, anger intensity, anxiety frequency and anxiety intensity scales were .87, .87, .90, and .89, respectively.

Affect Intensity Measure
(AIM; Larson, Diener and Emmons, 1986).

The AIM is a 40 item self-report questionnaire that assesses the characteristic strength or intensity with which an individual typically experiences his/her emotions. Both positive (25 items) and negative (13 items) emotions are included; the remaining two items are neutral. The AIM has a 3-month temporal reliability of .81 and a coefficient alpha of .90. Cronbach's alpha for the scale comprising positive items only was .91, and for negative items only, .80.

Anger Expression Scale
(AX; Speilberger, Johnson, Russell, Crane,
Jacobs and Worden, 1984).

The AX is a self-report questionnaire that assesses subjects' method of expressing anger when provoked. Each item is rated for frequency on a 4-point Likert scale. The AX includes two eight item subscales (Anger-in, Anger-out) that measure whether anger is characteristically suppressed or exhibited. The authors report that internal consistency reliabilities for the two subscales range from .73-.85, and

that their intercorrelation is essentially zero. In its original form the AX requires subjects to indicate how often they behave in a particular manner (eg. I make sarcastic remarks), without reference to the source of anger. For the purposes of this study the AX was modified to yield separate scale scores for 1) anger expression in situations involving family, and 2) anger expression in situations involving friends/acquaintances. The decision to assess anger expression in this manner was based on clinical data indicating that feelings of anger/hostility experienced by headache sufferers are often directed towards loved ones (Khouri-Haddad, 1984). Alpha coefficients for the Anger-in (family), Anger-out (family), Anger in (peers) and Anger-out (peers) subscales were .83, .83, .78, and .58, respectively. Reliability coefficients for the total scales were somewhat lower with coefficient alphas of .69 and .73 for the anger-expression family and peer scales, respectively. Two measures were derived from each of the scales: 1) total anger expressed (anger-in plus anger-out); high scores on this measure are reflective of high trait anger (Lopez and Thurman, 1986), and 2) ratio of anger-in over total anger expression; this measure indicates the degree to which anger is characteristically suppressed.

Procedure

The headache screening questionnaire was used to

identify subjects who met inclusion criteria and were willing to participate in the study. Shortly after administering the screening instrument potential subjects were contacted by telephone and an appointment was set up for questionnaire administration. Subjects were required to complete all psychometric instruments in a single sitting. Headache subjects were also asked to monitor daily headache activity for a period of two weeks. Instruction in the use of the Headache Frequency Record was given immediately after questionnaire completion.

RESULTS

Sample Characteristics

Demographic and medical characteristics of the headache and headache-free samples are presented in Table 1. The majority of subjects were female (67.5% and 77.5% in the control and headache groups respectively). Gender differences were not observed on any of the demographic, medical, psychological or headache variables with the exception of affect intensity. Females experienced emotion more intensely than males ($t(78)=-3.47, p<.001$).

Control Group: The control group consisted of 13 males and 27 females with a mean age of 26.2 (range=18-43). Seventy-five percent of the subjects were single, 17.5% married, and 7.5% divorced or separated.

Table 1.

Demographic and Medical Characteristics

	Headache	Headache-free
Age		
Range	18-58	18-43
Mean		
Gender		
Female	31	27
Male	9	13
Marital Status		
Single	28	30
Married	9	7
Divorced	3	3
Social Support		
Range	34-83	48-84
Mean	65.2	70.0
Psychosomatic Illness		
Range	0-2	0-2
Mean	.35	.30

Headache Group: The headache group consisted of 9 males and 31 females with a mean age of 25.0 (range=18-58). Seventy percent of the subjects were single, 22.5% married, and 7.5% separated or divorced. The average age of onset of headache was 15.7 with a mean duration of 9.3 years. Based on responses obtained from the Headache Symptom Questionnaire, 4 subjects were classified as migraine, 26 as muscle-contraction, 7 as combined migraine muscle-contraction, and 3 as cluster. Two-tailed t-tests revealed that the headache group did not differ from the control group on any of the demographic variables, nor were group differences found with respect to total social support, social support peers, family, or significant others, or number of psychosomatic disorders reported (headache: $\underline{m}$=.30; headache-free: $\underline{m}$=.35).

Within Headache Groups - Episodic and Continuous: Average daily headache hours was used to divide headache sufferers into two groups: episodic and continuous or near-continuous. The original criterion for inclusion in the continuous group (ie. minimum fifteen hours/day) was relaxed due to difficulty finding students with recurrent headache. Using a modified version of the Kolmogorov-Smirnov Maximum Deviation Test the largest gap (diff.=.1915, $\underline{p}<.05$) in the continuum of headache hours was found between the 34th and 35th highest scores representing 7.2 hours/day and 10.0 hours/day respectively. Subjects

reporting an average of ten hours or greater per day were classified as continuous while those reporting less than ten hours were classified as episodic.

Episodic: The episodic group consisted of 7 males and 30 females with a mean age of 23.8 (range=18-39). Approximately 70% of the sample was single (70.6%), 23.5% married, and 5.9% separated or divorced.

Continuous: The continuous headache group consisted of 2 males and 4 females, ranging in age from 20 to 58 (m=32). Four individuals were single, one married, and one divorced or separated. Two-tailed independent t-tests revealed that the continuous group did not differ from the episodic group on the demographic, social support, or number of psychosomatic disorders variables. Group differences were found with respect to several of the headache indices including: hours ($t(38) = -10.47$, $p<.001$), headache days ($t(38)=-4.07$, $p<.001$), and severity ($t(38)=-4.96$, $p<.001$). Continuous subjects reported greater overall headache activity than those with episodic headache. Table 2 illustrates the indices of headache activity by group.

Table 2.

Indices of Headache Activity by Group: Episodic vs. Continuous

	Episodic		Continuous	
	Mean	SD	Mean	SD
HOURS	3.8	1.8	12.5	2.3**
SEVERITY	8.7	5.3	26.9	7.4**
LA	5.7	2.3	11.3	3.9**
FREQUENCY	9.6	3.7	17.2	6.8
INTENSITY	1.2	0.51	1.2	0.62
HDAYS	8.7	2.6	13.3	1.6**
MEDS	0.41	0.95	1.6	1.8
YEARS	8.7	6.9	13.0	8.2
AGEON	15.1	5.2	19.0	8.6

**: $p<.01$

Headache History

Approximately 50% of the sample had seen a physician regarding headache. Of those individuals, five had been referred to a neurologist. No significant neurological findings were reported by any subject. The majority (75%) of subjects who had seen a physician received some form of treatment, primarily pharmacologic. Two individuals had received biofeedback in addition to medication, and a third subject had been given acupuncture as well as medication. An additional 17% of the sample had been treated by non-medical health practitioners including chiropractors (n=4) and dentists (n=2). Two of the subjects had borderline TMJ which they felt contributed to headache and hence cited splints as a form of treatment. Chiropractic treatments entailed "spinal adjustments," the exact nature of which was unclear.

In terms of students perceptions regarding the causes of headache, the vast majority (92%) believed that stress or anxiety was a precipitant. Fatigue (55%) and studying or eyestrain (45%) were also frequently cited (see Table 3 for the distribution of headache triggers). These results are consistent with previous analyses of headache triggers among college students (Attanasio and Andrasik, 1987).

Table 3.

Perceived Headache Triggers

	Percent of Sample Endorsing
Stress/Anxiety	92.5
Fatigue	55.0
Studying/Eyestrain	45.0
Food Substances caffiene chocolate sugar acidic products	32.5
Other Illnesses TMJ low back pain neck problems	30.0
Eating Patterns hunger over-eating	27.5
Environmental Stressors noise bright lights	17.5
Alcohol	17.5
Hot Weather	15.0
Hormonal Changes	10.0
Allergies	5.0
Excessive Sleep	5.0

Indices of Headache Severity

Several indices of headache severity were derived from the headache frequency record: daily headache hours (HOURS), average length of attacks (LA), frequency (FREQ), number of headache days (HDAYS), intensity (INT), and severity (SEV). Given that any one of a number of indices of headache activity (other than hours) could have been used to group subjects, an effort was made to determine if hours was in fact the best predictor of perceived severity. A stepwise multiple regression analysis was conducted using standardized scores on headache indices as predictor variables, and the degree to which headache was perceived to be a problem as the criterion. Table 4 displays the correlations between the variables. The regression of headache variables on problem yielded an overall F value that was significant ($F(1,29)=7.72$, $p<.01$), accounting for 21% of the variance in the degree to which headache constitutes a problem. Daily headache hours made the only significant contribution to the prediction ($\beta=.46$; $t(29)=2.78$, $p<.01$). Correlational and t-test analyses revealed no significant associations between headache hours and any of the demographic (age, sex, marital status), social support, or medical (number of psychosomatic symptoms) variables.

Table 4.

Correlations among Headache Indices

		1.	2.	3.	4.	5.	6.	7.
LENGTH	1.	1.0						
HOURS	2.	0.77	1.0					
SEVERITY	3.	0.72	0.92	1.0				
FREQUENCY	4.	0.05	0.63	0.59	1.0			
INTENSITY	5.	0.08	0.11	0.43	0.17	1.0		
H. DAYS	6.	0.24	0.74	0.71	0.85	0.25	1.0	
PROBLEM	7.	0.44	0.46	0.42	0.18	0.11	0.23	1.0

Primary Analyses

In order to examine both between group (headache vs. headache-free) and within-headache (continuous vs. episodic) differences, two orthogonal contrasts were created. Vector 1 represented the between group comparison (headache versus headache-free) and vector 2 contrasted episodic with continuous headache sufferers (vectors were modified to control for group size; Pedhauzer, 1982).

Summary statistics of the distributions of scores for each of the independent measures revealed that a number of measures were highly skewed. The raw data for these variables (anger expression total-family, anger expression total-peer, proportion of somatic anxiety) were therefore transformed. Because all variables were positively skewed logarithmic transformations were undertaken (Tabachnik and Fidell, 1983). In all of the subsequent analyses logarithmically transformed scores were used in place of raw data for these variables.

The LISREL program (Joreskog and Sorbom, 1985, version 6.6) was used to test the model presented in the introduction and all subsequent models. In each of the models presented, all nonsignificant parameters were set to zero; each arrow linking indicator variables to latent variables and latent variables to one another thus constitutes a significant association (at the .05 level or greater). Arrows extending from endogenous latent

variables (circles) to observed indicator variables (rectangles) specify the correlation between indicators and and latent constructs. Arrows extending from exogenous to endogenous latent variables reflect the impact of exogenous variables on the group contrasts. This value can be analagized to a directional regression coefficient.

Model 1

Figure 1 specifies the hypothesized relationships between the psychological variables and the group constrasts. The model had six concepts--four exogenous (Anxiety, Anger, Anger Suppression, and Emotional Intensity) and two endogenous (Headache vs. Headache-Free and Episodic vs. Continuous). The variables hypothesized to measure each of these latent constructs are illustrated in Figure 1. The Anxiety latent variable, for example, had three indicator variables: trait anxiety (TAX), anxiety frequency (AXFREQ) and proportion of anxiety that is somatically expressed (SOMA). The Anger latent variable consisted of trait anger (TAN), anger frequency (ANFREQ), and total anger expression (TAE). Total anger behavior (expression) was derived by summing the total anger expression scores from the peer and family questionnaires. These scales were highly intercorrelated (r=.63). The third latent variable, Anger Suppression, had two indicator variables, anger suppression-family (ASF) and anger suppression-peers (ASP). The final latent variable,

Emotional Intensity, consisted of three measures of emotional intensity, anxiety intensity (AXI), anger intensity (ANI), and affect intensity (AIM). The correlation matrix of model components, and the group means for each of these variables are presented in tables 5, 6 (headache, headache-free) and 7 (episodic, continuous) respectively.

Table 5.

Correlation Matrix for Model 1 Components

		1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.	13.
V1	1.	1.0												
V2	2.	0.0	1.0											
TAX	3.	-0.55	0.08	1.0										
TAN	4.	-0.22	0.03	0.39	1.0									
ASF	5.	-0.06	-0.39	-0.04	-0.21	1.0								
ASP	6.	-0.36	-0.09	0.29	-0.13	0.24	1.0							
SOMA	7.	0.15	-0.09	-0.37	-0.18	0.05	0.09	1.0						
AIM	8.	-0.19	0.05	0.37	0.23	-0.16	-0.13	0.25	1.0					
AXFREQ	9.	-0.36	-0.02	0.51	0.31	0.16	0.27	-0.21	0.28	1.0				
TAE	10.	-0.35	-0.09	0.51	0.51	0.05	0.23	-0.13	0.07	0.34	1.0			
ANFREQ	11.	-0.11	0.03	0.35	0.37	0.07	0.03	0.22	0.24	0.81	0.35	1.0		
AXI	12.	-0.15	0.01	0.34	0.34	-0.09	0.08	-0.03	0.33	0.42	0.22	0.24	1.0	
ANI	13.	0.01	-0.02	0.27	0.37	-0.01	0.04	0.03	0.31	0.41	0.20	0.33	0.67	1.0

1: Headache vs. Headache-free
2: Continuous vs. Episodic
3: Trait Anxiety
4: Trait Anger
5: Anger Suppression-Family
6: Anger Suppression-Peers
7: Somatic Anxiety
8: Affect Intensity
9: Anxiety Frequency
10: Total Anger Expressed
11: Anger Frequency
12: Anxiety Intensity
13: Anger Intensity

Table 6.
Group Means for Independent Measures: Headache and Headache-Free

	Headache		Headache-free	
	Mean	SD	Mean	SD
TAX	44.6	9.1	34.4	6.2***
TAN	21.3	5.2	19.0	4.4*
ASF	0.49	0.07	0.47	0.09
ASP	0.56	0.09	0.49	0.07***
SOMA	-0.32	0.05	-0.30	0.07
AIM	3.9	0.48	3.7	0.52
NEGAFF	4.0	0.60	3.6	0.54**
POSAFF	3.8	0.52	3.7	0.58
AXFREQ	43.1	20.9	28.2	18.5***
TAE	3.0	0.14	2.9	0.16**
ANFREQ	35.9	21.1	31.7	16.8
AXI	4.3	1.3	3.9	1.4
ANI	3.8	1.3	3.8	1.1

Table 7.
Group Means for Independent Measures: Episodic and Continuous

	Episodic		Continuous	
	Mean	SD	Mean	SD
TAX	45.1	9.2	42.1	8.1
TAN	21.4	5.4	20.8	4.8
ASF	0.46	0.10	0.63	0.13***
ASP	0.55	0.08	0.58	0.12
SOMA	-0.32	0.05	-0.30	0.05
AIM	3.9	0.47	3.8	0.53
NEGAFF	4.0	0.61	3.7	0.58
POSAFF	3.8	0.52	3.7	0.47
AXFREQ	43.0	21.6	44.7	17.8
TAE	3.0	0.14	3.1	0.12
ANFREQ	36.3	20.9	34.3	23.7
AXI	4.3	1.4	4.2	1.3
ANI	3.8	1.6	3.9	1.4

* :$p<.05$; ** :$p<.01$; ***:$p<.001$

The results of the first analysis indicated that the original model yielded a very poor fit to the data ($\chi^2(67)=207.74$; $p<.000$). A significant chi-square is not desirable as it indicates that the differences between the model-implied relationships and the actual patterns in the data are not small enough to be sampling fluctuations. Examination of the modification indices associated with each parameter revealed a) that several of the constrained variables had been mis-specified (showed a strong association with latent constructs other than those to which they had initially been fixed), and b) the need to remove the constraint that the residual error terms for anxiety and anger frequency be independent of each other. Because the correlational structure of these two variables forced them to load on different latent variables, no acceptable fit could be obtained without allowing their residual errors to correlate. Rather than removing this constraint a decision was made to drop anger-frequency from subsequent analyses (anger frequency did not correlate with either of the group contrasts, while anxiety frequency did).

Because of the extreme number of high modification indices in the original model, a discriminant function analysis was conducted to determine how the model might be improved. Two significant functions emerged: the first discriminated headache sufferers from headache-free

controls, and the second discriminated episodic and continuous headache sufferers (see Table 8). Based on the pattern of correlations between the variables and the discriminant functions (see Table 9), and the modification indices, the following changes were made in the original model: 1) as each of the anger suppression variables contributed to different functions, two latent variables (anger suppression peers and anger suppression family) were created; 2) since negative affect intensity loaded significantly on the first function, and general affect intensity and positive affect intensity did not, this variable was introduced in place of general affect intensity; 3) the constraint restricting the intensity variables from loading on any latent variable other than emotional intensity was removed; both the modification indices in the original model and the discriminant function results suggested a strong association between these variables and Latent Variable 1.

Table 8.

Canonical Discriminant Functions Evaluated at Group Means

Group	Function 1	Function 2
Headache-free	-0.70834	-0.00586
Episodic	0.73244	-0.20775
Continuous	0.68613	1.45960

Table 9.

Correlations Between Discriminating Variables and Canonical Discriminant Functions

Variables	Function 1	Function 2
TAX	0.91559	-0.16296
ASP	0.52282	0.29852
AXFREQ	0.41892	0.11444
TAE	0.41136	-0.05458
NEGAFF	0.33448	-0.26137
ANI	0.30661	0.05376
ANFREQ	0.29618	0.02831
AXI	0.28831	-0.14638
SOMA	-0.23223	0.08607
ASF	0.05585	0.98762
TAN	0.18574	-0.29797
AIM	0.19233	-0.22419
POSAFF	0.06158	-0.20204

Model 2

The resultant solution consisted of five exogenous variables (Anxiety, Anger, Anger Suppression-Family, Anger Suppression-Peers, and Emotional Intensity) and the two group contrasts (see Figure 2). This model yielded an acceptable fit to the data ($\chi^2(50)=59.24$, $p=.17$). The corresponding adjusted goodness of fit index (.841) indicated that the model adequately accounted for the observed variances and covariances. In addition, the root mean square residual (.077) suggested relatively small residuals on average. As illustrated in Figure 2, only one latent variable, Anger Suppression Family, had a significant effect on the within headache contrast. Subjects with continuous headache were less expressive than episodic headache sufferers. Three latent variables were found to significantly discriminate headache sufferers from headache-free controls: Anger Suppression Peers, Anxiety, and Emotional Intensity. Relative to controls, headache sufferers showed greater anxiety and a stronger tendency to suppress anger towards peers. The Anxiety latent construct correlated positively with both the Anger construct and the Suppression Peers construct. The high correlation between the Anger and Anxiety latent variables, together with the stronger effect of Anxiety on the between groups contrast, precluded a direct effect of the Anger construct. Trait anger levels were, however, higher in the headache group.

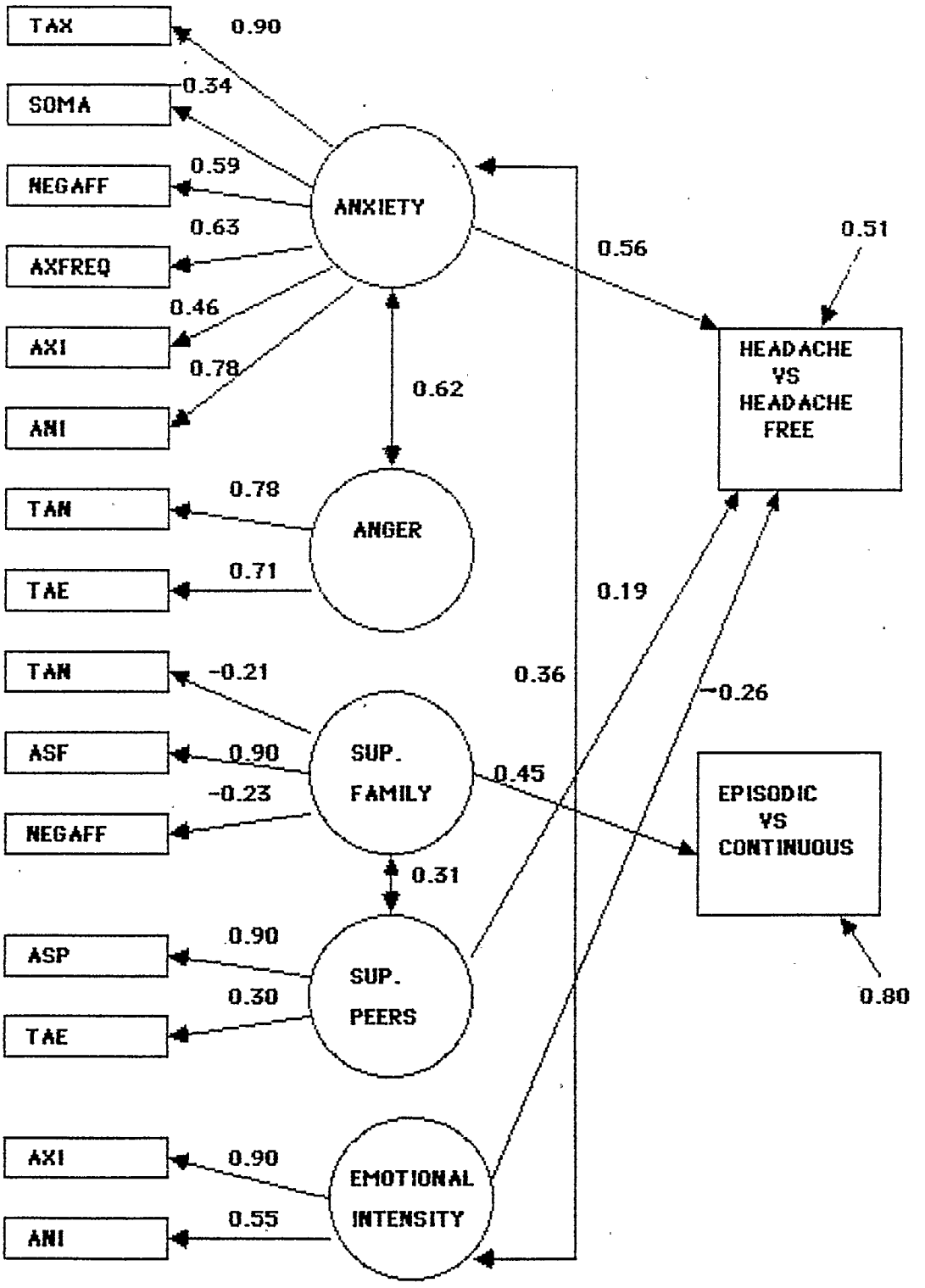

Figure 2. Model 2.

TAX: Trait Anxiety; SOMA: Somatic Anxiety; NEGAFF: Negative Affect Intensity; AXFREQ: Anxiety Frequency; AXI: Anxiety Intensity; ANI: Anger Intensity; TAN: Trait Anger; TAE: Total Anger Expressed; ASF: Anger Suppression-Family; ASP: Anger Suppression-Peers.

The significant negative association between the Emotional Intensity Latent Variable and the headache contrast appeared to be a statistical artifact as the two indicator variables (ANI, AXI) also loaded on the Anxiety latent variable, but in the opposite direction. Several steps were taken to determine if this was in fact the case. First the intensity indicators were prevented from loading on any latent construct other than Emotional Intensity. This resulted in an extremely poor fit. Similarily, deleting the construct itself from the model, and freeing the indicators to load on the Anxiety variable also resulted in a poor fit. These outcomes suggested that the the significant effect associated with Emotional Intensity was, in fact, a statistical artifact, occuring as a result of the conflicting pattern of correlations between trait anxiety, the intensity variables, and the between group contrast. As depicted in Table 10, trait anxiety and the intensity variables were correlated, trait anxiety and the between group contrast were correlated, but the intensity variables and the between group contrast were not correlated. As a result the intensity indicators were related to latent variables that had opposite relationships to the between group vector. The possibility that a real phenomenon was involved seemed unlikely given the failure of affect intensity (in model 1) and negative affect intensity (in model 2) to load significantly

Table 10.

Correlation Matrix for Model 2 Components

		1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
V1	1.	1.0											
V2	2.	0.00	1.0										
TAX	3.	-0.55	0.08	1.0									
TAN	4.	-0.22	0.03	0.39	1.0								
ASF	5.	-0.06	-0.39	-0.04	-0.21	1.0							
ASP	6.	-0.36	-0.09	0.29	-0.13	0.24	1.0						
SCMA	7.	0.15	-0.09	-0.37	-0.18	0.05	0.09	1.0					
NEGAFF	8.	-0.33	0.13	0.55	0.23	-0.19	-0.02	-0.12	1.0				
AXFREQ	9.	-0.36	-0.02	0.51	0.31	0.16	0.27	-0.21	0.33	1.0			
TAE	10.	-0.35	-0.09	0.51	0.51	0.05	0.23	-0.13	0.19	0.34	1.0		
AXI	11.	-0.15	0.01	0.34	0.34	-0.09	0.08	-0.03	0.43	0.42	0.21	1.0	
ANI	12.	0.01	-0.02	0.27	0.37	-0.01	0.04	0.03	0.29	0.41	0.26	0.67	1.0

1: Headache vs. Headache-free
2: Continuous vs. Episodic
3: Trait Anxiety
4: Trait Anger
5: Anger Suppression-Family
6: Anger Suppression-Peers
7: Somatic Anxiety
8: Negative Affect Intensity
9: Anxiety Frequency
10: Total Anger Expressed
11: Anxiety Intensity
12: Anger Intensity

on the Emotional Intensity construct. This latent variable and the two intensity indicators were therefore dropped from the model.

Model 3

The third model thus consisted of four exogenous variables (Anxiety, Anger, Anger Suppression-Peers and Anger Suppression-Family) and the between and within group contrasts. Model 3 yielded a good fit to the data with a chi-square of 31.28 with 31 degrees of freedom (p=.45), an adjusted goodness of fit index of .89, and a root mean square residual of .06. This was a significant improvement over the results obtained in model 2. Figure 3 illustrates the significant effects associated with each of the estimated coefficients. In this model, Anxiety and Suppressing Anger to Peers exerted a direct effect on the between group contrast. Headache sufferers were characterized by higher levels of anxiety, and a reduced ability to overtly express anger to friends or acquaintances. The Anxiety latent construct was positively correlated with both the Anger and Suppression Peers variables. Anxious subjects were more angry and more apt to suppress anger to peers. As in model 2, only Anger Suppression-Family has a significant effect on the within headache contrast. This construct has three indicators, high anger-suppression-family, low negative affect intensity, and low trait anger.

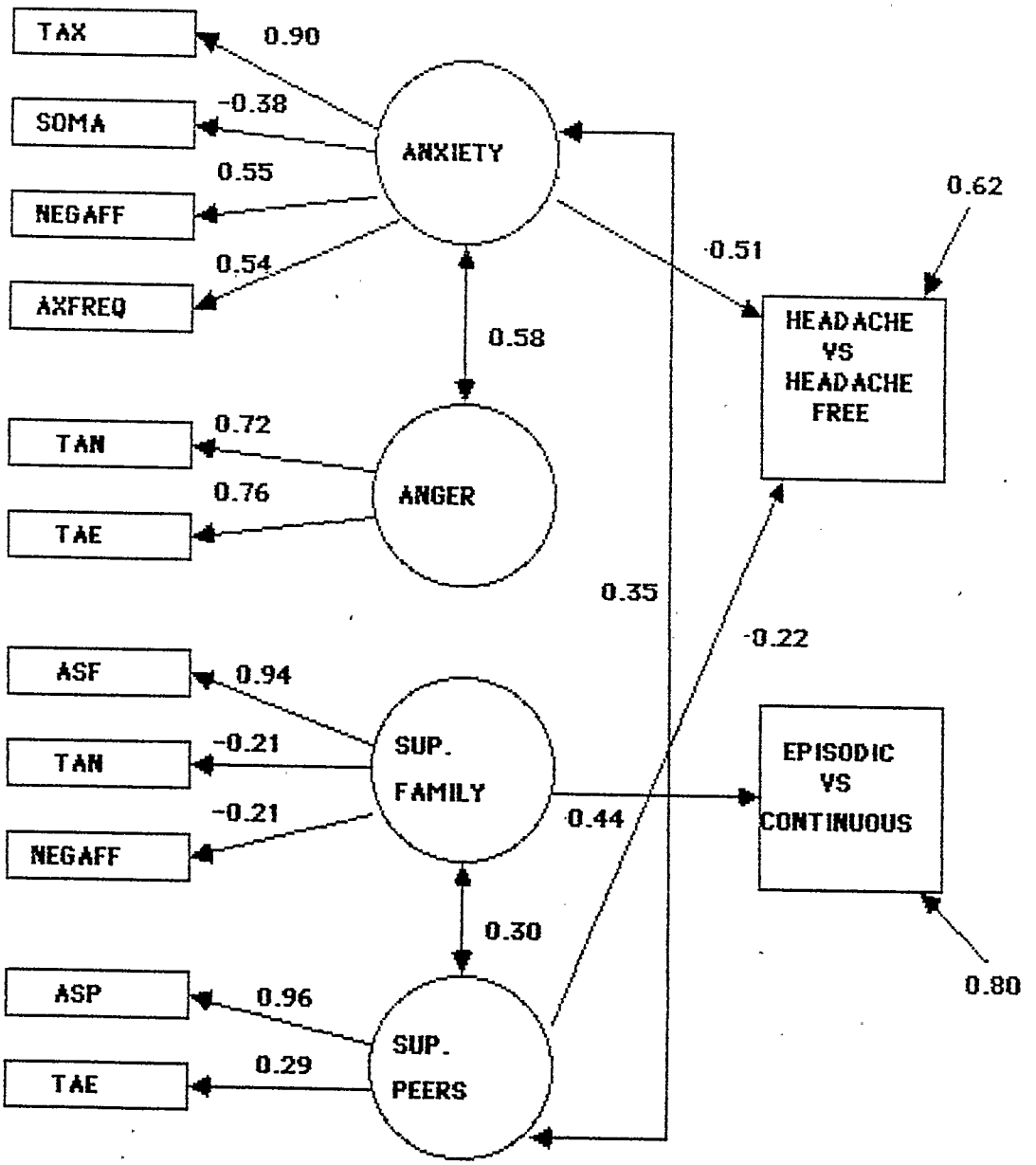

Figure 3. Model 3.

TAX: Trait Anxiety; SOMA: Somatic Anxiety; NEGAFF: Negative Affect Intensity; AXFREQ: Anxiety Frequency; TAN: Trait Anger; TAE: Total Anger Expressed: ASF: Anger Suppression-Family; ASP: Anger Suppression-Peers.

Subjects who suppress anger to family thus report feeling less anger, and reacting less intensely to negative moods in general. This pattern was characteristic only of continuous headache sufferers. No such association was found for the Suppression-Family construct and the between groups vector.

In summary, this model suggested that within a headache population the tendency to inhibit expression of anger to family members specifically, is associated with increasing severity. Continuous subjects did not differ from those with episodic headache in anxiety, anger, or suppression of anger towards friends or acquaintances. With respect to the between group comparisons, headache sufferers in general tended to be more emotional, and less able to express anger towards peers than headache-free controls. No between group differences were found in suppression of anger towards family, or the anger construct.

Psychological Variables and Headache Diagnosis

In order to determine if any of the exogenous variables were related to diagnostic categories, the episodic-continuous contrast was replaced by a vector contrasting tension headache sufferers with those classified as migraine, mixed, or cluster. The decision to group migraine, mixed and cluster headache sufferers was supported both by the literature, and by the outcome of a

discriminant function analyses. With respect to the former, mixed headache is believed to evolve from episodic migraine (Saper, 1986; Nappi, Facchinetti & Martignoni, 1985), and cluster headache is considered a rare variant of vascular headache (Diamond and Dallesio, 1978). Vascular symptoms thus underly each of these diagnostic categories. In addition, a discriminant function analysis using the measures incorporated in Model 3, yielded two functions, the first maximally discriminating control subjects from headache sufferers, and the second discriminating subjects with vascular symptoms (migraine, mixed cluster) from those that were classified as tension headache sufferers (see Table 11). Seventy percent of cases were classified correctly, and the pattern of classification errors supported grouping subjects in the aforementioned manner (see Table 12).

Table 11.

Canonical Discriminant Functions Evaluated at Group Means

Group	Function 1	Function 2
Headache-free	-0.69775	-0.18750
Tension	0.37212	0.61484
Vascular	0.40267	-0.65275

Table 12.

Classification Results for Discriminant Function Analysis

Actual Group	Predicted Group Membership		
	Headache-free	Tension	Vascular
Headache-free	32(80%)	3(7.5%)	5(12.5%)
Tension	10(38.5%)	11(42.3%)	5(19.3%)
Vascular	1(7.1%)	1(7.1%)	12(85.7%)

Tension Group

The tension headache group consisted of twenty-six headache sufferers (5 male, 21 female) ranging in age from 18 to 58 (m=24.5). Seventy-three percent of the subjects were single, 23% married, and 4% separated or divorced.

Vascular Group

Of the fourteen subjects (4 male, 10 female) in the vascular group, four were classified as migraine, seven as mixed muscle contraction-migraine, and three as cluster. Subjects ranged in age from 18 to 39, with a mean of 26.1. Sixty-five percent of the sample was single, 21% married, and 14% separated or divorced. Two-tailed t-tests revealed that the tension and vascular groups did not differ on the demographic, social support, or psychosomatic disorders variables. Differences were however found in several of the headache variables. Subjects with vascular symptoms reported a longer history of problem headache ($t(38)=-2.94$, $p<.005$), an earlier age of onset of headache ($t(38)=2.60$, $p<.02$), and greater medication usage ($t(38)=-3.17$, $p<.007$). Table 13 displays indices of headache activity by group.

Table 13.

Indices of Headache Activity by Group: Tension vs. Vascular

	Tension		Vascular	
	Mean	SD	Mean	SD
HOURS	4.2	2.3	6.7	4.9
SEVER	9.5	6.1	14.9	11.4
LA	5.6	2.4	8.2	3.9*
FREQ	10.3	3.0	11.5	6.6
INTEN	1.2	0.54	1.2	0.50
HDAYS	9.2	2.7	10.0	3.6
MEDS	0.11	0.43	1.5	1.6**
YRS	7.1	5.9	13.5	7.6*
AGEON	17.3	5.3	12.6	5.6*

**: $p<.01$
*: $p<.05$

Model 4

Model 4 consisted of the four endogenous variables described in model 3, and the diagnostic group comparison. The correlations between model components, and group means for the independent measures are presented in Tables 14 and 15, respectively.

The model yielded a good fit to the data ($\chi^2(31)=29.15$, $p=.56$), with an adjusted goodness of fit index of .94, and a root mean square residual of .06. As in model 3, only one significant effect was observed for the diagnostic group contrast. Subjects with vascular symptoms showed less overt anger expression towards family members. Figure 4 illustrates the significant association between the Anger Suppression Family construct and the diagnostic group contrast. Model 4 differs from the previous model only in the strength of the association between suppression family and the within headache contrast; a stronger effect is obtained for the diagnostic, as opposed to the episodic/continuous, group contrast.

Table 14.

Correlation Matrix for Model 4 Components

		1.	2.	3.	4.	5.	6.	7.	8.	9.	10.
V1	1.	1.0									
V2	2.	0.00	1.0								
TAX	3.	-0.55	-0.06	1.0							
TAN	4.	-0.22	0.14	0.39	1.0						
ASF	5.	-0.06	-0.48	-0.04	-0.21	1.0					
ASP	6.	-0.36	-0.18	0.29	-0.13	0.24	1.0				
SOMA	7.	0.15	0.08	-0.37	-0.18	0.05	0.09	1.0			
NEGAFF	8.	-0.33	0.18	0.55	0.23	-0.19	-0.02	-0.12	1.0		
AXFREQ	9.	-0.36	-0.12	0.51	0.31	0.16	0.27	-0.21	0.33	1.0	
TAE	10.	-0.35	-0.08	0.51	0.51	0.05	0.23	-0.13	0.12	0.34	1.0

1: Headache vs. Headache-free
2: Continuous vs. Episodic
3: Trait Anxiety
4: Trait Anger
5: Anger Suppression-Family
6: Anger Suppression-Peers
7: Somatic Anxiety
8: Negative Affect Intensity
9: Anxiety Frequency
10: Total Anger Expressed

Table 15.

Group Means for Independent Measures: Tension and Vascular

	Tension		Vascular	
	Mean	SD	Mean	SD
TAX	44.0	10.1	45.7	6.9
TAN	21.9	5.2	20.1	5.3
ASF	0.43	0.09	0.58	0.10***
ASP	0.54	0.09	0.59	0.09
SOMA	-0.31	0.05	-0.33	0.06
AIM	3.9	0.51	3.9	0.42
NEGAFF	4.1	0.65	3.8	0.45
POSAFF	3.8	0.52	3.9	0.53
AXFREQ	40.6	21.2	47.9	20.3
TAE	3.0	0.16	3.0	0.13
ANFREQ	35.2	20.9	37.5	22.2
AXI	4.3	1.3	4.3	1.5
ANI	3.8	1.3	3.8	1.4

***:$p<.001$

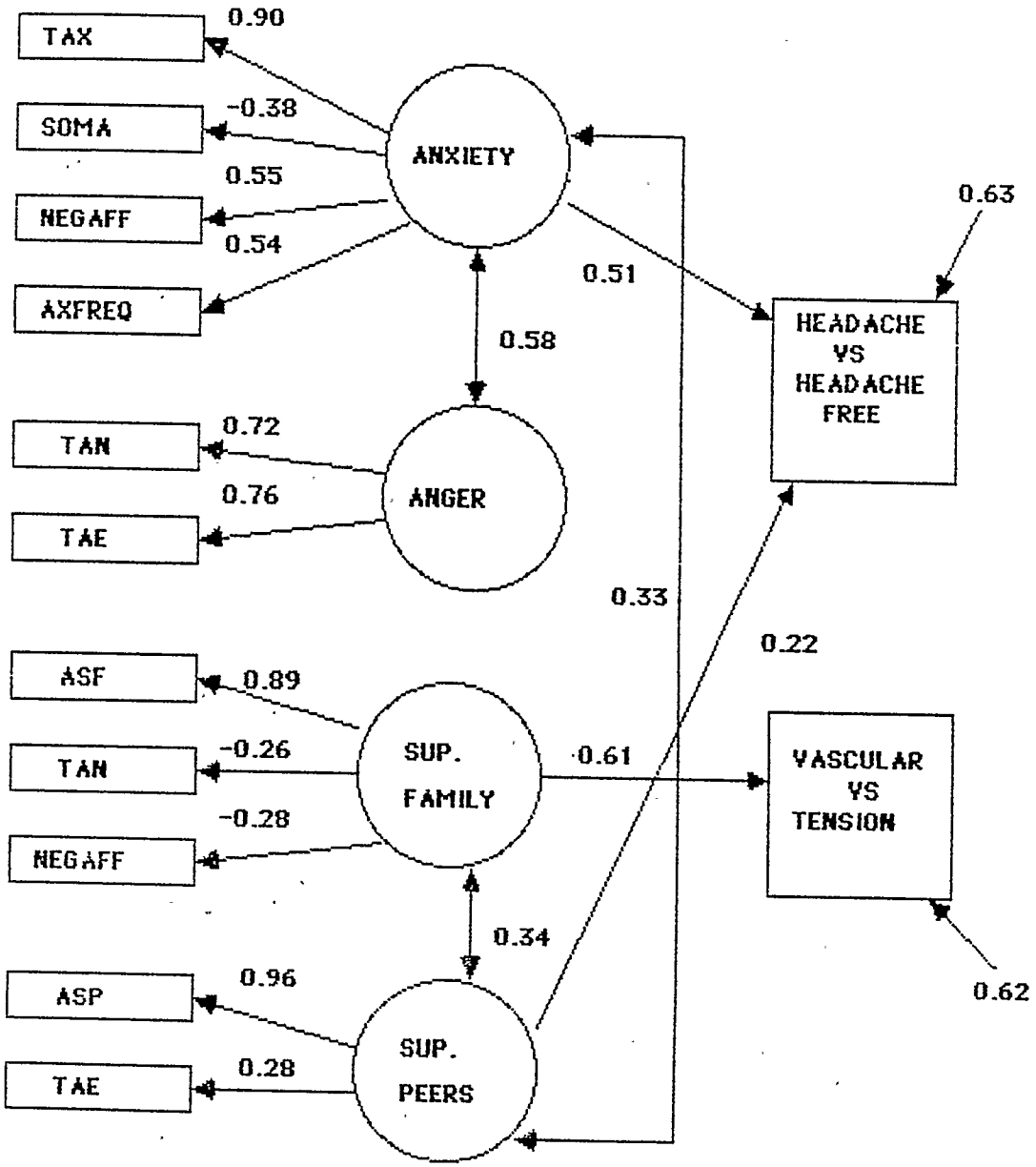

Figure 4. Model 4.

TAX: Trait Anxiety; SOMA: Somatic Anxiety; NEGAFF: Negative Affect Intensity; AXFREQ: Anxiety Frequency; TAN: Trait Anger; TAE: Total Anger Expressed: ASF: Anger Suppression-Family; ASP: Anger Suppression-Peers.

To summarize the LISREL results, several latent constructs were found to significantly discriminate headache sufferers from headache-free controls. The headache sufferers assessed in this study experienced more anxiety and were more inclined to inhibit overt anger expression in situations involving peers. The within-headache contrasts revealed that both continuous headache sufferers and subjects with vascular headache have considerably more difficulty expressing anger to family than do the less severe headache subjects. In terms of emotional experience, however (anger and anxiety), these subjects were no more or less emotional than the episodic group.

DISCUSSION

The goals of the present study were twofold: a) to determine the degree to which heightened levels of anxiety and anger, characterize headache sufferers drawn from a non-clinic college student population and b) to examine within-headache variability in emotional experience and expression. The within-headache analysis was adopted with the hope of clarifying a conflicting finding in the area, namely, that headache sufferers have been described as both highly emotional and non-emotional/non-expressive.

Headache constituted a significant problem for most of the headache sufferers in the present study. On average, students reported approximately five hours of headache per

day. Previous research has demonstrated similar levels of headache activity among college students (Andrasik and Holroyd, 1980), and subjects obtained from clinical settings (Blanchard et al, 1984). Students' perceptions regarding the cause of headache were consistent with that reported in a recent epidemiological study of headache in college students. Attanasio and Andrasik (1987) cited tension and eyestrain as the most common elicitors of headache. In the present study, stress/anxiety and fatigue/eyestrain were similarily identified most often as headache triggers. In terms of headache activity, these relatively young headache sufferers were experiencing headache as frequently as do the majority of subjects found in clinical samples (Blanchard et al, 1984).

Headache sufferers in general were found to be significantly more anxious than headache-free controls. Moreover, the degree of anxiety evidenced by headache subjects in this study approached levels observed in psychiatric populations (Spielberger, 1970). High levels of anxiety have been reported in previous studies utilizing both clinic and non-clinic populations. Blanchard et al (1984), for example, found clinically significant anxiety in a sizeable proportion of clinic based tension headache sufferers. Andrasik and Holroyd (1980) also reported marked elevations in anxiety among students with recurrent headache. The latter study provides a basis for comparison

as the students sampled, reported approximately the same degree of headache activity as was found in the present study. Mean levels of trait anxiety were virtually identical across these studies. Anxiety thus appears to be a robust correlate of recurrent headache.

The trait form of the State Trait Anger Inventory (Spielberger et al, 1983) was used to assess anger. Spielberger has conceptualized trait anger in terms of individual differences in the disposition to experience anger. Persons high in trait anger are believed to perceive a wider ranger of situations as anger-provoking, and hence experience anger more frequently than subjects low in this dimension. Higher levels of trait anger were found among headache sufferers relative to controls. This is consistent both with the clinical observational literature (Gill, Spruiell, & Spierings, 1988; Adler, Morrisey-Adler & Packard, 1987; Khouri-Haddad, 1984), and with empirical accounts of increased hostility in this population (Blaszczynski, 1983; Henryk-Gutt and Rees, 1973).

Although heightened anger was observed among headache sufferers, anxiety proved to be a more significant factor. Within the LISREL model, anger and anxiety were found to be highly interrelated but only anxiety actually discriminated headache sufferers from headache-free controls. The unique contribution of anger, independent of anxiety, thus remains

to be determined. It is important to note that the high degree of relationship found between these constructs is not peculiar to this particular sample. In a study examining anger among college students, Deffenbacher et al (1986) found that general anxiety was as predictive of various self-report and observational indices of anger as was trait anger (Deffenbacher, Demm & Brandon, 1986). Other investigators have reported significant associations between trait anxiety measures and measures of both anger and hostility (Costa and McCrae, in press; Siegman, Dembroski & Ringel, 1987).

The heightened levels of anxiety and anger found within this sample may, in fact, be reflective of a general disposition to experience negative mood states. Watson and Clark (in press) have demonstrated that emotional experience is dominated by two broad and largely independent dimensions, Negative Affect (NA) and Positive Affect (PA). Subjects high in NA are more likely to experience significant aversive mood states even in the absence of overt stress. The disposition to experience these states is unrelated to an individual's experience of positive emotions; that is, high NA does not necessarily imply a lack of positive emotional experience. In the present study, in addition to high levels of anxiety and anger, headache sufferers were found to experience negative emotions in general with greater intensity than controls

(negative affect intensity). Differences were not, however, observed in strength of positive emotional experiences (positive affect intensity). Taken together, these data suggest that headache sufferers as a group, are more disposed to experience negative mood states than are headache-free controls.

The primary contribution of the anger construct in headache may lie more in the domain of emotional inhibition or suppression than experience. Anger expression must be distinguished both conceptually and empirically from the experience of anger (Spielberger et al, 1984). Whereas measures of trait anger reflect the frequency with which angry feelings are experienced, measures of anger expression assess the nature and frequency of anger-related behavior.

Headache sufferers in general were found to typically inhibit overt expressions of anger to peers, but not family. These individuals were less likely than headache-free controls to direct their anger towards the source of frustration. Group differences in anger suppression could not be attributed to perceived differences in social support as headache sufferers did not differ from controls on this dimension. The frequency of anger behavior in general (eg. anger-in plus anger-out) also loaded positively on the Suppression-Peers construct. This indicates that the tendency to characteristically

suppress anger to peers is associated with a higher overall frequency of anger behavior. Similar inhibitory patterns have been reported in at least one other study involving headache sufferers. Grothgar and Scholz (1987) found that during an anger-provocation procedure headache subjects expressed significantly less anger than did controls. Nonassertive behavior has also been linked to headache activity (Jones and Page, 1986). A central feature of nonassertiveness is difficulty expressing negative feelings (Arrindell and van der Ende, 1985). Jones and Page (1986) found that college students with headache problems were significantly less assertive than those who did not suffer from headache. The ability to express feelings of anger openly and constructively thus appears lacking among headache sufferers, at least with respect to peers.

The present data indicated that anger suppression in peer interactions is related to trait anxiety. It seems likely that individuals who suppress anger to peers, do so, because the act of directly expressing anger is in some way threatening. Subjects high in trait anxiety may be particularly disposed to view anger expression as threatening, as these individuals perceive threat across a variety of situations (Spielberger et al, 1970). Jones and Page (1986) have similarly noted a high degree of relationship between assertive behavior and trait anxiety. Moreover, trait levels of anxiety have been found to

decrease following assertiveness training (Wakesman, 1984; Brooks and Richardson, 1980).

Group differences in characteristic modes of expressing anger to family were not observed, nor was general anxiety related to anger expression in the family context. When angered by family members, headache sufferers in general were no more or less likely to overtly express anger than headache-free controls. Within this sample, then, the tendency to inhibit overt anger expression appears limited to situations involving peers. College students may be particularly reticent about expressing anger to friends, as opposed to family, since more threat may be associated with anger expression in the former case.

Although headache sufferers in general did not differ from controls in their ability to express anger to family, considerable within-headache variability was found in this dimension. Subjects with continuous headache showed marked elevations on the Suppression Family factor. Again, group differences in suppression to family could not be attributed to differences in perceived supportiveness of family. Continuous headache sufferers perceived their families to be equally as supportive as did episodic headache sufferers. The Suppression Family factor was comprised of variables reflecting high suppression of anger towards family, low trait anger and low negative affect

intensity. Subjects who suppress anger in family conflicts thus report infrequent arousal of anger and low intensity of negative emotional experience in general. This pattern is quite different from that observed in the Suppression Peer factor. Why low levels of anger should be associated with high suppression to family, and not to peers, is unclear. It could be argued that persons who rarely feel anger are perhaps better able to inhibit overt anger expression on the few occasions when they are angered. If this were the case, however, a similar pattern would be expected in the Suppression Peer factor. A more reasonable explanation is that subjects were unwilling to acknowledge angry feelings within the family context.

The constellation of traits comprising the suppression to family factor (ie. high anger suppression to family, low trait anxiety, low negative affect intensity) are worthy of further study. Previous research has generally sought to examine emotional suppression in a general fashion, both in terms of content and in terms of headache sufferers. For example, there have been several attempts to isolate deficits associated with the alexithymic cognitive style among subjects with headache conditions. Efforts to demonstrate a higher incidence of alexithymic behavior among headache sufferers as compared to controls have largely failed (Demjen, 1986; Blanchard et al, 1984). Subjects with headache are no more or less alexithymic than

subjects without headache. This non-emotional/expressive style has, however, been linked to increasing headache severity. Subjects with more severe headache conditions demonstrate higher levels of alexithymic behavior (Demjen, 1986).

The continuous headache sufferers assessed in the present study were not 'alexithymic' in the strictest sense of the word. Alexithymic deficits are believed to involve not only the inhibition of felt emotion, but also limited awareness of, or ability to recognize, emotional states. Subjects with continuous headache seemed aware of distressing feelings in general, that is, high levels of anxiety and anger, but were less willing to acknowledge anger in the family. Moreover, stress was cited by each of these individuals as contributing to the headache problem.

In previous studies examining severe headache patients, denial of the influence of stress/anxiety on headache, repressive (self-deceptive) coping styles, and increased evidence of alexithymic behavior have been reported as characterizing the more continuous subjects (Demjen, 1986; Demjen and Bakal, 1984; Demjen, 1981). The continuous or near-continuous subjects in the present study neither denied the stress-headache relationship or evidenced self-deceptive tendencies (as reflected in their willingness to admit to feeling anxious and angry). These individuals were, however, more emotionally controlled;

they were less apt to express feelings of anger regardless of the context in which anger was evoked. The pattern observed in the present study may be a precursor to the clinical pattern.

Within-headache differences in nonexpressivity to family were also found when comparisons were made using traditional diagnostic criteria. Subjects with headache of a vascular nature (migraine, cluster, combined) scored higher on the Suppression Family factor than tension headache sufferers. Although five of the six continuous headache subjects were included in the vascular group, this outcome could not be attributed to group differences in headache activity. Vascular subjects did not differ from tension headache sufferers in headache hours, severity, intensity, frequency or days with headache. In fact, the only headache variable that differentiated groups was length of attacks, with vascular subjects experiencing longer headaches.

It is not known whether this pattern may be specific to a particular diagnostic group (eg. migraine, cluster, combined) as the sample sizes were too small to permit comparisons. More likely, this non-expressive style is non-specific and simply differentiates severe headache sufferers from those with less severe headache problems.

Although vascular subjects did not, on average, report greater headache activity, the symptoms associated with

vascular headache (eg. nausea and vomiting, visual disturbances, light sensitivity etc.) tend to be more severe than those associated with tension headache. In a series of studies, examining the symptom characteristics of headache sufferers, Bakal and colleagues found that frequency of migraine symptoms was the strongest predictor of perceived headache severity (Kaganov, Bakal & Dunn, 1981; Bakal and Kaganov, 1979; Bakal and Kaganov, 1977). The fact that vascular subjects relied more heavily on prescription medication further suggests that pain may have been more severe within this sample. Headache intensity ratings did not differ between groups, however, pain is idiosyncratic and subjective in nature, and subjects with only one type of headache typically do not have the experience of the other type as a basis for comparison. The vascular group, then, likely constituted a more severe sample of headache sufferers.

The present findings suggest that pervasive deficits in overt anger expression are characteristic of subjects who experience particularly severe headache related symptoms and/or continuous pain. A final model comparing severe headache sufferers (vascular and/or continuous), with those who reported neither continuous headache or vascular symptoms yielded an even stronger effect for the Suppression Family factor. This final comparison was not presented in the results section as it entailed the removal

of only a single subject (continuous headache sufferer) from the tension group into the vascular group. Suppression of anger in the family context thus appears characteristics of severe headache sufferers in general, regardless of typology.

Most theorists would agree that the degree and patterning of emotional expression is strongly influenced by the socialization process (eg. Buck, 1979). The family itself is likely to be the primary agent for socializing emotional expression as it is within the family that the individual first learns to communicate needs and concerns. Balswick and Avert (1977) have in fact demonstrated a strong relationship between student and parental expressiveness. Persons who are non-expressive then, likely come from parents who were also non-expressive.

Within a family systems perspective, emotional non-expressiveness is viewed as a means of avoiding interpersonal conflict. In the dynamic sense the symptom becomes the vehicle of emotional expression. Minuchin and colleagues work with 'psychosomatic children' indicates that specific family interactional patterns characterize families in which psychsomatic conditions develop (Minuchin, Baker, Rosman, Liebman, Milman, & Todd, 1975; Minuchin, 1974). Excessive togetherness and overprotection, rigidity, and conflict avoidance have been identified as consistent features. These characteristic

patterns, in conjunction with a physiological disposition, are believed to encourage symptom development and/or exacerbation, particularly if the symptom in some way serves to mask underlying conflict. In a review of the literature pertaining to family process and psychosomatic disorders, Meissner (1974) similarily cited 'emotional overinvolvement' and conflict avoidance as characteristic of these families.

The potential contribution of family conflict in the development of "chronic daily headache" has been alluded to by Featherstone and Beitman (1982; 1984). The majority of daily headache sufferers assessed in these studies reported significant personal stresses, defined as any condition, situation, or relationship that upset the patient (work-related stress was not included). Underlying family/marital conflict appeared to be the most prevalent feature. Drummond (1985) also cited "social problems at home" as a significant discriminator of constant versus episodic headache. Regardless of whether family conflict is masked or not, the act of suppressing angry feelings is both physiologically and psychologically stressful. Inhibiting anger in situations involving family as well as peers likely adds to the overall adverse effect of this behavior.

To summarize, headache sufferers in general were both highly emotional and non-expressive. As a group, these

individuals evidenced high levels of anger and anxiety, the latter of which was related, in part, to difficulties expressing anger to friends and acquaintances. Whereas no differences were found between groups in characteristic modes of expressing anger to family, the within-headache analyses revealed a significant association between inhibited expression of anger in this context (eg. family), and severity of headache. In comparison to less severe headache sufferers, subjects with vascular-type headaches and/or continuous pain showed a reduced ability to express anger to family members. This pervasive deficit in emotional expression was the only factor that differentiated these more severe headache sufferers from those whose conditions were not as distressing.

Although both heightened emotionality and emotional suppression appear to be important correlates of headache in general, the degree to which severe headache sufferers can be distinguished from subjects with less severe headache, depends not on emotionality, but rather, on the expression of felt emotion. The observation that emotionality is common to all headache sufferers, regardless of severity, indicates that this line of research is perhaps less useful than a specific focus on emotional suppression. While each of these factors may contribute to ongoing headache susceptibility, the severity of the symptom itself appears to correlate only with

non-expressiveness.

This association may be mediated by another variable such as persistent muscular hypertension. Both Bakal et al (1984) and Olesen (1978) have argued strongly that chronic muscle tension represents a significant component of severe headache. Interestingly, higher levels of musculoskeletal activity have been observed consistently among vascular as compared to tension headache sufferers ((Philips, 1978; Bakal and Kaganov, 1976; Pozniak and Patewicz, 1976). Moreover, frequency of musculoskeletal symptoms has been identified as a significant discriminator of episodic versus constant headache, irrespective of diagnostic status (Drummond, 1985). The presence of chronic muscle tension is theoretically consistent with the inhibition-disease model. According to this model sustained muscular hypertension develops across time as a result of the individual's failure to express felt emotion. The extent to which inhibited emotional expression and heightened musculoskeletal activity covary among subjects with severe headache disorders appears worthy of further investigation. Future research should address this issue with a specific focus on evaluating potential conflict within the family and/or family interactional patterns that might influence headache susceptibility and exacerbation.

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Appendix A

Headache Symptom Questionnaire

Please use the following rating scale to describe your headache activity.

0=never

1=infrequently

2=sometimes

3=usually

4=always

Place a number from the preceding scale in the blank space provided.

____ 1. I awaken with headaches.

____ 2. My headache ends within 24 hours.

____ 3. I have sudden attacks of headaches.

____ 4. My headache is worse at the end of the working day.

____ 5. My headache is throbbing or pulsating.

____ 6. My headache can be described as a feeling of tightness or external pressure on my head (band-like or cap-like).

____ 7. My headache begins on one side.

____ 8. My headache starts in the neck, shoulder, or back of the head.

____ 9. My headache is associated with visual changes like seeing stars; blind spots, double vision and/or intolerance to light.

____ 10. I have nausea and/or vomiting with my headache.

____ 11. My headache gets worse if I strain, cough or lift objects.

____ 12. My headache is better if I can loosen up my neck muscles.

____ 13. Aspirin, Anacin, Bufferin, Excedrin, Alka Seltzer, and similar drugs relieve my headaches.

____ 14. I take a prescribed medication to prevent a full-blown attack of headache.

____ 15. My headache starts during periods of relaxation.

____ 16. My headache starts during periods of stress.

How old were you when headaches first became a problem? _____

Please list any factors/triggers that you believe are associated with onset of your headaches.

_____

_____

_____

_____

_____

Appendix B

We are interested in how you feel about the following statements. Read each statement carefully. Please indicate how you feel about each statement by circling: the 1 if you Very Strongly Disagree
the 2 if you Strongly Disagree
the 3 if you Mildly Disagree
the 4 if you are Neutral
the 5 if you Mildly Agree
the 6 if you Strongly Agree
the 7 is you Very Strongly Agree

	Very Strongly Disagree	Strongly Disagree	Mildly Disagree	Neutral	Mildly Agree	Strongly Agree	Very Strongly Agree
1. There is a special person who is around when I am in need.	1	2	3	4	5	6	7
2. There is a special person with whom I can share joys and sorrows.	1	2	3	4	5	6	7
3. My family really tries to help me.	1	2	3	4	5	6	7
4. I get the emotional help and support I need from my family.	1	2	3	4	5	6	7
5. I have a special person who is a real source of comfort to me.	1	2	3	4	5	6	7
6. My friends really try to help me.	1	2	3	4	5	6	7
7. I can count on my friends when things go wrong.	1	2	3	4	5	6	7
8. I can talk about my problems with my family.	1	2	3	4	5	6	7
9. I have friends with whom I can share my joys and sorrows.	1	2	3	4	5	6	7
10. There is a special person in my life who cares about my feelings.	1	2	3	4	5	6	7
11. My family is willing to help me make decisions.	1	2	3	4	5	6	7
12. I can talk about my problems with my friends.	1	2	3	4	5	6	7

Appendix C

A number of statements which people have used to describe themselves are given below. Read each statement carefully and then circle the appropriate response to indicate how you generally feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

		Almost Never	Sometimes	Often	Almost Always
1.	I feel pleasant	1	2	3	4
2.	I tire quickly	1	2	3	4
3.	I feel like crying	1	2	3	4
4.	I wish I could be as happy as others seem to be	1	2	3	4
5.	I am losing out on things because I can't make up my mind soon enough	1	2	3	4
6.	I feel rested	1	2	3	4
7.	I am "calm, cool, and collected"	1	2	3	4
8.	I feel that difficulties are piling up so that I cannot overcome them	1	2	3	4
9.	I worry too much over something that doesn't really matter	1	2	3	4
10.	I am happy	1	2	3	4
11.	I am inclined to take things hard	1	2	3	4
12.	I lack self-confidence	1	2	3	4
13.	I feel secure	1	2	3	4
14.	I try to avoid facing a crisis or difficulty	1	2	3	4
15.	I feel blue	1	2	3	4
16.	I am content	1	2	3	4
17.	Some unimportant thought runs through my mind and bothers me	1	2	3	4
18.	I take disappointments so keenly that I can't put them out of my mind	1	2	3	4
19.	I am a steady person	1	2	3	4
20.	I get in a state of tension or turmoil as I think over my recent concerns and interests	1	2	3	4

Appendix D

Please rate the degree to which you typically or generally experience the following feelings/sensations when you are anxious.

1=not at all

2=not very much

3=sometimes

4=quite a bit

5=very much so

Place a number from the preceding scale in the blank space provided.

_____ I find it difficult to concentrate because of uncontrollable thoughts.

_____ I feel jittery in my body.

_____ I imagine terrifying scenes.

_____ My heart beats faster

_____ I worry too much over something that doesn't really matter.

_____ I nervously pace.

_____ I feel like I am losing out on things because I can't make up my mind soon enough.

_____ I perspire.

_____ I can't keep anxiety provoking thoughts out of my mind.

_____ I become immobilized.

_____ Some unimportant thought runs through my mind and bothers me.

_____ I feel tense in my stomach.

_____ I get diarrhea.

_____ I can't keep anxiety provoking pictures/images out of my mind.

Appendix E

A number of statements which people have used to describe themselves are given below. Read each statement carefully and then circle the appropriate response to indicate how you generally feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

		Almost Never	Sometimes	Often	Almost Always
1.	I have a fiery temper	1	2	3	4
2.	I am quick-tempered	1	2	3	4
3.	It makes me furious when I am criticized in front of others	1	2	3	4
4.	When I get mad I say nasty things	1	2	3	4
5.	I feel annoyed when I am not given recognition for doing good work	1	2	3	4
6.	I fly off the handle	1	2	3	4
7.	I get angry when I am slowed down by others mistakes	1	2	3	4
8.	When I get frustrated, I feel like hitting someone	1	2	3	4
9.	I am a hotheaded person	1	2	3	4
10.	I feel infuriated when I do a good job and get a poor evaluation	1	2	3	4

Appendix F

Below are a list of items related to feelings of anger among university students. As you read each item, decide if you experience anger with regard to that event. If you do not experience anger with regard to specific events listed below, leave the spaces to the left of the event blank. If you do experience anger with regard to any of the events listed below, please make two separate ratings of those events.

In Column A, please rate how frequently or infrequently you experience anger with regard to the event using the scale below.

1 2 3 4 5 6 7

infrequently frequently

In Column B, rate the level of intensity usually associated with the anger you experience related to the same event.

1 2 3 4 5 6 7

very low very high

Column A (frequency)	Column B (intensity)	
_____	_____	1. Receiving an unexpected low grade.
_____	_____	2. Registration/administrative procedures.
_____	_____	3. Being unable to find a parking place on or near campus.
_____	_____	4. Being unable to find desired library references.
_____	_____	5. "Pop" quizes/unannounced tests or assignments.
_____	_____	6. Having money/financial problems.
_____	_____	7. Losing notebooks/notes
_____	_____	8. Having car/transportation problems.
_____	_____	9. Heavy reading assignments.
_____	_____	10. Heavy writing assignments.
_____	_____	11. Someone borrowing something and not returning it.
_____	_____	12. Being turned down or stood up for a date.
_____	_____	13. Having personal belongings stolen.
_____	_____	14. Not being able to register for a desired course.
_____	_____	15. Professors lecturing too fast.

Appendix G

The items listed in the previous questionnaire may also elicit feelings of anxiety. This time, as you read each item, decide if you experience anxiety with regard to that event. If you do not experience anxiety with regard to a given event, leave the spaces to the left of the event blank. If you do experience anxiety with regard to any of the events listed below, please make two separate ratings of those events.

In Column A, please rate how frequently or infrequently you experience anxiety with regard to the event using the scale below.

1 2 3 4 5 6 7

infrequently frequently

In Column B, rate the level of intensity usually associated with the anxiety you experience related to the same event.

1 2 3 4 5 6 7

very low very high

Column A (frequency)	Column B (intensity)		
_____	_____	1.	Receiving an unexpected low grade.
_____	_____	2.	Registration/administrative procedures.
_____	_____	3.	Being unable to find a parking place on or near campus.
_____	_____	4.	Being unable to find desired library references.
_____	_____	5.	"Pop" quizes/unannounced tests or assignments.
_____	_____	6.	Having money/financial problems.
_____	_____	7.	Losing notebooks/notes
_____	_____	8.	Having car/transportation problems.
_____	_____	9.	Heavy reading assignments.
_____	_____	10.	Heavy writing assignments.
_____	_____	11.	Someone borrowing something and not returning it.
_____	_____	12.	Being turned down or stood up for a date.
_____	_____	13.	Having personal belongings stolen.
_____	_____	14.	Not being able to register for a desired course.
_____	_____	15.	Professors lecturing too fast.

Appendix H

The following questions refer to emotional reactions to typical life-events. Please indicate how YOU react to these events by placing a number from the following scale in the blank space preceding each item. Please base your answers on how YOU react, not on how you think others react or how you think a person should react.

NEVER	ALMOST NEVER	OCCASIONALLY	USUALLY	ALMOST ALWAYS	ALWAYS
1	2	3	4	5	6

1. _____ When I accomplish something difficult I feel delighted or elated.
2. _____ When I feel happy it is a strong type of exuberance.
3. _____ I enjoy being with other people very much.
4. _____ I feel pretty bad when I tell a lie.
5. _____ When I solve a small personal problem, I feel euphoric.
6. _____ My emotions tend to be more intense than those of most people.
7. _____ My happy moods are so strong that I feel like I'm in heaven.
8. _____ I get overly enthusiastic
9. _____ If I complete a task I thought was impossible, I am ecstatic.
10. _____ My heart races in anticipation of some exciting event.
11. _____ Sad movies deeply touch me.
12. _____ When I'm happy it's a feeling of being untroubled and content rather than being zestful and aroused.
13. _____ When I talk in front of a group for the first time my voice gets shaky and my heart races.
14. _____ When something good happens, I am usually much more jubilant than others.
15. _____ My friends might say I'm emotional.
16. _____ The memories I like the most are those of times when I felt content and peaceful rather than zestful and enthusiastic.
17. _____ The sight of someone who is hurt badly affects me strongly.
18. _____ When I'm feeling well it's easy for me to go from being in a good mood to being really joyful.
19. _____ "Calm and cool" could easily describe me.
20. _____ When I'm happy I feel like I'm bursting with joy.
21. _____ Seeing a picture of some violent car accident in a newspaper makes me feel sick to my stomach.
22. _____ When I'm happy I feel very energetic.
23. _____ When I receive an award I become overjoyed.
24. _____ When I succeed at something, my reaction is calm contentment.
25. _____ When I do something wrong I have strong feelings of shame and guilt.
26. _____ I can remain calm even on the most trying days.
27. _____ When things are going good I feel "on top of the world."
28. _____ When I get angry it's easy for me to still be rational and not overreact.
29. _____ When I know I have done something very well, I feel relaxed and content rather than excited and elated.
30. _____ When I do feel anxiety it is normally very strong.
31. _____ My negative moods are mild in intensity.
32. _____ When I am excited over something I want to share my feelings with everyone.
33. _____ When I feel happiness, it is a quiet type of contentment.
34. _____ My friends would probably say I'm tense or high-strung."
35. _____ When I'm happy I bubble over with energy.
36. _____ When I feel guilty, this emotion is quite strong.
37. _____ I would say my happy moods are closer to contentment than to joy.
38. _____ When someone compliments me I feel so happy that I could "burst."
39. _____ When I am nervous I get shaky all over.
40. _____ When I am happy the feeling is more like contentment and calm than exhilaration and excitement.

Appendix I

A number of statements which people have used to describe themselves when they are angry are listed below. Read each statement carefully. How you feel or behave when you are angry may depend on who the source of anger is, or who is around when you are angry.

In Column A, please rate the degree to which each statement characterizes you when you are angry and a family member is involved.

In Column B, please rate the degree to which each statement characterizes you when you are angry and a friend or acquaintance is involved.

Place a number from the following scale in the blank space provided.

1=almost never

2=sometimes

3=often

4=almost always

Column A (family member)	Column B (friend or acquaitance)	
_____	_____	1. I argue with others.
_____	_____	2. I am irritated a great deal more than people are aware of.
_____	_____	3. I lose my temper.
_____	_____	4. I keep things in.
_____	_____	5. I say nasty things.
_____	_____	6. I boil inside, but don't show it.
_____	_____	7. I someone annoys me, I am apt to tell him or her how I feel.
_____	_____	8. I pout or sulk.
_____	_____	9. I am secretly quite critical of others.
_____	_____	10. I strike out at whatever infuriates me.
_____	_____	11. I express my anger.
_____	_____	12. I withdraw from people.
_____	_____	13. I do things like slam doors.
_____	_____	14. I make sarcastic remarks to others.
_____	_____	15. I am angrier than I am willing to admit.
_____	_____	16. I tend to harbour grudges that I don't tell anyone about.

Appendix J: Headache Screening Questionnaire

Please circle the answer that applies to you.

1. Do you consider your headache a problem?

   a. never
   b. seldom
   c. sometimes
   d. often
   e. always

2. How often do you get a headache?

   a. less than once a month
   b. approx. 1-2 a month
   c. approx. 1-2 a week
   d. approx._3-4 a week
   e. almost ever day

3. On average how long do your headaches last?

   a. less than one hour
   b. 1-2 hours
   c. 2-4 hours
   d. 5-6 hours
   e. all day

4. Do you know what causes your headaches?

   a. never
   b. seldom
   c. sometimes
   d. often
   e. always

If so please list cause(s) of headache:

6. If you are willing to participate in a headache research study involving self-observation of symptoms, please provide your name and telephone number below. Note: Headache-free controls are also needed. Thank you.