Research report

Affective intensity and lability: Heritability in adult male twins

Emil F. Coccaro a,⁎, Anthony D. Ong c, A.D. Seroczynski b, C.S. Bergeman b

a Clinical Neuroscience & Psychopharmacology Research Unit, Department of Psychiatry and Behavioral Neuroscience, University of Chicago, Chicago, IL, USA
b Department of Psychology, University of Notre Dame, South Bend, Indiana, USA
c Department of Human Development, Cornell University, Ithaca, NY 14853

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Abstract

Background: Inability to monitor and self-regulate heightened levels of affect lability and affect intensity is associated with a range of mood, anxiety, and personality disorders, psychosomatic symptoms and socially maladaptive behaviors. Despite the importance of these aspects of affective regulation, there are no twin study data to shed light on the genetic and environmental components of these constructs.

Methods: Affective Lability Scale (ALS) and Affect Intensity Measure (AIM) questionnaires were administered to 796 male twins in the Vietnam Era Twin Registry and subjected to twin and model-fitting analyses. Complete data were available from 182 monozygotic and 119 dizygotic twin pairs.

Results: Biometrical genetic model-fitting estimates indicated that additive genetic influence accounted for 40% of the variance in affect intensity and 25% of the variance in the ALS subscale assessing anxiety-depression mood shifts. Nonadditive genetic influence was indicated for ALS subscales measuring shifts between normal mood and depression (29%) and anger (27%), respectively. There was negligible evidence of shared environmental influence on affect measures. In contrast, estimates of nonshared environmental influences ranged from 52% to 74%.

Limitations: Female were not included in this study due to the nature of the twin cohort. Data from subjects in a population cohort may not generalize to clinical populations. Measures of environment were not included.

Conclusions: These results provide evidence for moderate heritability of affect intensity and specific measures of affect lability. Individual differences in mood regulation may represent phenotypic variation in a core psychobiologic vulnerability (e.g., neurotransmitter systems).

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1. Introduction

Individuals are known to differ, reliably, in the lability and intensity of their affective experiences (cf. Eid and Diener, 1999; Schimmack and Diener, 1997) and these constructs of affect are key to various theories of personality integration and complexity (Buss and Plomin, 1975; Frijda et al., 1992), models of affect structure (Russell, 1980; Thayer, 1989), motivational arousal (Brehm and Self, 1989; Gray, 1967), studies of emotion regulation (Carver et al., 1989; Lazarus, 1991), and dysregulation (Diener et al., 1985; Larson et al., 1980). Affect lability refers to the degree of intraindividual variability in affective states, whereas affect intensity refers to the strength of affective responsiveness. Although covarying across individuals (Larsen and Diener, 1987), lability and intensity represent distinct dimensions of affective experience (Harvey et al., 1989; Larsen and Diener, 1985) that are separable from personality dispositions (cf. Eid and Diener, 1999; Schimmack and Diener, 1997) and show notable levels of continuity across the lifespan (Buss and Plomin, 1975; Campos et al., 1989; Strelau, 1991).

Both lability and intensity represent basic processes of affect regulation that may indicate either resilience or vulnerability to...
psychopathology (Larsen, 2000). Inability to monitor and self-regulate heightened levels of affect lability (particularly in negative moods) and affect intensity is associated with a range of personality and mood disorders, psychosomatic symptoms, socially maladaptive behaviors (Harvey et al., 1989; Herpertz et al., 1997; Larsen and Diener, 1987), and individual differences in emotional development (Rothbart and Bates, 1998). In adults, tonically labile and intense affective states are related to a profile of distinctly negative mental health outcomes. Rapid shifts between euthymia and negative affective states such as anger, anxiety, depression, and hypomania show strong positive associations with measures of current dysphoric mood (Costello et al., 1991; Harvey et al., 1989), impulsivity and borderline personality disorders (Cowdry et al., 1991; Koenigsberg et al., 2002), and neuroticism (Eid and Diener, 1999; Hepburn and Eysenck, 1989). Whereas affect lability is signaled largely by negative affective experiences (Eid and Diener, 1999; Harvey et al., 1989), affect intensity appears to cut across both positive and negative emotions, reflecting the general quality of affective experience rather than its frequency or hedonic valence (Diener et al., 1991; Diener and Lucas, 1999). Affect intensity, nevertheless, shares a positive manifold with measures of alcohol abuse (Flett and Hewitt, 1995), cyclothymia and bipolar affective disorder (Diener et al., 1985; Flett and Hewitt, 1995), emotionality (Larsen et al., 1986), emotion- and avoidant-oriented coping styles (Flett et al., 1996), negative mood regulation expectancies (Catanzaro, 1993), neuroticism (Schimmack and Diener, 1997), and psychosomatic distress (Larsen and Diener, 1987).

Predisposing genetic factors may moderate individual differences in both the physiological arousal and somesthetic perception of labile and intense affective states. Evidence supporting genetic influence comes from family, twin, and adoption studies of affective disorders, which illustrate the significant comorbidity of affect lability and affect intensity with major depression and bipolar disorder (Henry et al., 2001; Merikangas et al., 2002). The risk of developing major depressive disorder is between threefold (McGuffin and Katz, 1986) and ninefold (Farmer et al., 2000) in the families of depressed probands compared with the general population. In comparison, the relative risk of bipolar disorder in first-degree relatives of bipolar probands is between 3% and 19% (Cradock and Jones, 1999). The preponderance of studies of twins of unipolar probands (Beirut et al., 1999; Kendler et al., 1995; Kendler and Prescott, 1999; Lyons et al., 1998; McGuffin et al., 1996) show a genetic effect with significantly higher concordance rates in monozygotic (MZ) twins than in dizygotic (DZ) twins. Moreover, additive genetic effects, on average, are only slightly higher for bipolar depression (Jones et al., 2004). Finally, in studies of depression and bipolar disorder, probands who were adopted had a significantly higher rate of affective disorder in their biological compared to their adoptive parents (Mendlewicz and Rainer, 1977; Sullivan et al., 2000). Taken together, the evidence from family, twin, and adoption studies suggests an important genetic contribution to both unipolar and bipolar affective disorder.

It has been suggested that the dimensional traits of affect lability and affect intensity, rather than affective disorder per se, have a heritable component (Silverman et al., 1991; Torgersen, 2000). Moreover, given that genetic effects have been found for neuroticism (Plomin et al., 1997) and pessimism (Plomin et al., 1992), emotionality and sociability (Plomin et al., 1988), and aggression (Coccaro et al., 1997), it seems plausible that a similar mechanism may underlie individual differences in affect lability and affect intensity. Apart from the few previous studies (i.e., Baker et al., 1992; Lykken and Tellegen, 1996; Plomin et al., 1988; Royse et al., 2002; Tellegen et al., 1988) that have reported substantial heritabilities for both positive and negative affect, the influence of genetic and environmental factors on shaping the phenotypic expression of affect lability and affect intensity have been largely unexplored.

The purpose of the present study is to estimate the contribution of (a) additive factors, (b) nonadditive genetic effects, (c) shared environment, and (d) nonshared environment to variation in adult affect lability and affect intensity using a classical twin study. Heritability is a descriptive statistic that is defined as the proportion of phenotypic variance in a population that is due to genetic variance. The genetic variance can also be partitioned into two types—additive and nonadditive. Additive genetic variance is the degree to which genotypic values combine linearly in their effect on the phenotype, whereas nonadditive genetic variance embodies hereditary influences that are due to dominance (interactions between alleles at a single locus), as well as the variance due to higher order interactions, termed epistasis. Environmental differences among individuals can also lead to phenotypic differences. Environmental influences can be separated into those which are shared among members of a family (referred to in a variety of ways, including common, shared, within, E2) and those which are not (termed specific, nonshared, between, E1). Shared environmental influences are defined as any nongenetic influence that contributes to the phenotypic resemblance among family members, whereas the nonshared environmental component contains environmental factors that make family members different from one another.

2. Methods

2.1. Participants

Participants were 182 monozygotic (MZ) and 119 dizygotic (DZ) twin pairs from the Vietnam Era Twin (VET) Registry. Ascertainment and characteristics of the entire twin registry (Eisen et al., 1987, 1989; Goldberg et al., 1987) and this sample specifically (Coccaro et al., 1997; Seroczyński et al., 1999) are described elsewhere. The protocol for this study was approved by both the IRB of the first author’s institution (MCP Hahnemann University, Philadelphia, PA) and by the IRB associated with the VET Registry at the time of study (Hines VAMC, Hines, IL). Informed consent was obtained by VET Registry staff. Briefly, questionnaires were mailed to 1208 individuals; 796 (65.9%) returned the completed packet. Of the non-responders, two individuals were identified as being deceased (0.2%), 169 individuals refused participation (16.2%), and 214 (18%) were lost to follow-up. Of those who responded, 628 (78.9%) were members of a twin pair, resulting in a pairwise response rate of 52%. The response rate for MZ (60.6%) twins was greater than DZ (39.4%) twins. Similar imbalance in zygosity response has been noted in other studies using the VET Registry (Goldberg et al., 1990; Henderson et al., 1990) and this discrepancy might be due to the increased likelihood that both members of a male MZ twin pair will
volunteer for research (Lykken et al., 1987). Mean age for the sample was 44.1 ± 2.9 years, range = 36–54. Participating twins were predominately Caucasian (94.1%) who were married, and had completed an average of 14.2 ± 2.5 years, range = 8–20 years of education. This distribution reflects that of the entire VET Registry (Henderson, et al., 1990).

2.2. Measures

2.2.1. The Affect Lability Scale

The Affect Lability Scale (ALS; Harvey et al., 1989) is a 54-item self-report inventory designed to assess changeability or shifts in affect. Scales of the ALS include shifts between euthymia (normal mood) and depression, anxiety, anger, and hypomania, as well as changes between hypomania and depression and anxiety and depression. For this study, only the depression, anger, and anxiety-depression scales were used, resulting in a total of 26 items. Items are scored on a four-point scale ranging from 1 = not like me at all to 4 = very much like me. Higher scores reflect greater affect lability.

Previous studies indicate that the various subscales of the ALS have adequate to good test–retest reliabilities, specifically, .64 for depression, .86 for anger, and .81 for anxiety-depression (Harvey et al., 1989). In this study, Cronbach’s alpha for the subscales of depression, anger, and anxiety–depression were: .86, .87, and .91.

2.2.2. The Affect Intensity Measure

The Affect Intensity Measure (AIM; Larsen and Diener, 1987) is a 40-item questionnaire designed to assess the strength or intensity of emotional experiences. The AIM questions were originally based on a definition of affect intensity that emphasizes the distinction between frequency and intensity of emotional experiences (e.g., “I am happy quite often.”) and intensity of emotion (e.g., “When I am happy, the feeling is one of intense joy”). The AIM was constructed to assess the latter.

Larsen et al. (1986) report that the AIM discriminates well from scales measuring lying, social desirability, defensiveness and infrequency, faking good and faking bad, and an extreme response set. In addition, the AIM also showed no significant relationship to measures of depression or psychological well-being. Stability across 1-, 2-, and 3-month retests was .80, .81, and .81, respectively, and strong internal consistency, with Cronbach’s alphas ranging from .90 to .94 over different samples (Larsen and Diener, 1987). Cronbach’s alpha for the AIM in this sample was .88.

3. Results

3.1. Descriptive statistics

All three ALS scales correlated significantly with each other (range: r = .68 to r = .82, p < .001) and with the AIM (range: .32 to .36, p < .001). Only correlations with age and these scores were notable as significant negative correlations between age and all three ALS scales (range: r = −.14 to r = −.22, p < .001), but not with AIM scores, were found. Accordingly, all ALS scale scores were standardized using a regression technique typically used in analyses of this type (McGue and Bouchard, 1984). Finally, because there were no significant mean or variance differences found by zygosity a basic assumption of the twin method was met and allowed further twin analyses to be conducted (Lykken, et al., 1987). Intraclass coefficients for MZ and DZ twin pairs, respectively, were: ALS-Depression: ICC = .31/.09, h² = .44; Anxiety-Depression: ICC = .22/.14, h² = .16; Anger: ICC = .30/.09, h² = .34; AIM: ICC = .46/.29, h² = .34. Accordingly, simple estimates of heritability (h²) were: ALS-Depression: h² = .44; Anxiety-Depression: h² = .16; Anger: h² = .34; AIM: h² = .34. While combat experience may affect twin similarity for affect intensity and lability in individuals ascertained from the VET Registry, use of a Combat Index score (created to reflect the variability in combat experiences: Janes et al., 1991), in a Hierarchical Multiple Regression (Cohen and Cohen, 1983) indicated no significant differences in twin similarity for any of the scales of the ALS or the AIM as a function of combat exposure.

3.2. Model-fitting

Description of behavioral genetic model-fitting techniques has been detailed elsewhere (Boomsma et al., 1989; Neale and Cardon, 1992) and these analyses were performed using LISREL 8.52 (Jöreskog et al., 2000) to estimate genetic (additive and nonadditive) and environmental (shared and nonshared) influences on the Affect Lability Scales and the Affect Intensity Measure. Results of model-fitting analyses are presented in Table 1.

The results of these analysis revealed that ALS Depression and ALS Anger scores display significant nonadditive genetic influence: 29% for depression and 27% for anger. ALS Anxiety and AIM scores display a significant pattern of additive genetic influence: 25% for ALS Anxiety scale and 40% for AIM. These results also indicate that shared environment has little influence on the four measures, but nonshared environment shows significant influence, accounting for 52–74% of the variance of all four scales.

Two reduced models were used to provide a better test of the significance of the genetic and shared environment parameters. The change in chi-square was determined with two models: Model 1 in which the shared environmental parameter was set to zero, and Model 2 in which the genetic parameter was set to zero. For those measures that showed nonadditive genetic influence, the nonadditive parameter was set to zero; for those that showed additive genetic influence, the additive parameter was set to zero. None of the three scales of the ALS or the AIM showed a significant change in chi-square when the shared environmental parameter was set to zero. That is, being in the same family with someone does not lead to similarities in affect lability or intensity later in life. The ALS Depression scale and the AIM both showed significant changes in chi-square when the genetic parameters were dropped from the models. This suggests that genetic influence plays a significant role in individual differences for depressive lability and affective intensity. The change in chi-square for ALS Anger scale was only marginally nonsignificant when the nonadditive genetic parameter was dropped from the model, indicating that the nonsignificance may be an issue of power rather than a poor model. That is, a larger sample may be necessary to detect effects of this size. For all four measures, nonshared environment appears to most strongly influence individual differences (Table 2).
Table 1
Parameter estimates and the proportion of phenotypic variance due to additive genetics, nonadditive genetics, shared environment and unique environment for the Affect Liability Scales and the Affect Intensity Measure.

<table>
<thead>
<tr>
<th>Scale</th>
<th>Ga</th>
<th>Gd</th>
<th>Es</th>
<th>En</th>
<th>(\chi^2) (1 df)</th>
<th>(p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Affect Liability Scale</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td>–</td>
<td>.54±.14</td>
<td>.14±.44</td>
<td>.83±.04</td>
<td>.09</td>
<td>.77</td>
</tr>
<tr>
<td>Anger</td>
<td>–</td>
<td>.52±.15</td>
<td>.15±.41</td>
<td>.84±.04</td>
<td>.05</td>
<td>.83</td>
</tr>
<tr>
<td>Anxiety–depression</td>
<td>.49±.23</td>
<td>(25%)</td>
<td>.08±.12</td>
<td>.86±.04</td>
<td>3.87</td>
<td>.05</td>
</tr>
<tr>
<td>Affect intensity</td>
<td>.63±.16</td>
<td>(40%)</td>
<td>.29±.31</td>
<td>.73±.04</td>
<td>.54</td>
<td>.46</td>
</tr>
</tbody>
</table>

Note: Ga = additive genetic; Gd = nonadditive genetic; Es = shared environment; En = nonshared environment.

4. Discussion

Individual differences in the ability to self-regulate tonically labile and intense affective states are central to determining resilience or vulnerability to psychopathology (Larsen, 2000). Such differences represent diatheses that are shaped by both genetic and environmental influences. Biogenetic influences may include genetic abnormalities (Plomin et al., 1990), neurochemical abnormalities (Gurvits et al., 2000; Steinberg et al., 1997), and acquired neurological abnormalities (Bechara et al., 1999). Environmental influences include shared (e.g., parenting) and nonshared (e.g., peer influence) environment (Haris, 1995).

The present research adds to the growing literature on the complexity of affective experience by investigating genetic and environmental influences on affect intensity and three specific measures of affect lability—reflecting euthymia–anger, euthymia–depression and depression–anxiety mood shifts. The results indicate significant genetic influence for all four affect measures. Additive genetic influence was indicated for affect intensity (40%) and for the ALS subscale reflecting anxiety–depression mood shifts (25%).

The results indicate significant genetic influence including for affect intensity and three specific measures of affect lability—reflecting euthymia–anger, euthymia–depression and depression–anxiety mood shifts. The results indicate significant genetic influence including for affect intensity and three specific measures of affect lability—reflecting euthymia–anger, euthymia–depression and depression–anxiety mood shifts.

Table 2
Reduced models testing the influence of shared rearing environment (Model 1) and genetic variance (Model 2).

<table>
<thead>
<tr>
<th>Scale</th>
<th>Model 1</th>
<th></th>
<th>Model 2</th>
<th></th>
<th>(\chi^2) (1 df)</th>
<th>(p)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Affect Liability Scale</td>
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</tr>
<tr>
<td>Depression</td>
<td>.02</td>
<td>ns</td>
<td>3.93</td>
<td>.05</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anger</td>
<td>.03</td>
<td>ns</td>
<td>3.33</td>
<td>ns</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anxiety–depression</td>
<td>.00</td>
<td>ns</td>
<td>1.20</td>
<td>ns</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Affect intensity</td>
<td>.24</td>
<td>ns</td>
<td>4.40</td>
<td>.05</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: Es = shared environmental variance; G = genetic variance; ns = nonsignificant.
showed that parents tend to treat their twins according to their actual zygosity and not their perceived zygosity, suggesting that it is the shared genes that contribute to the increased resemblance of identical twins and not just differential shared environmental effects. Third, the affect measures used in this study are based on self-report and are subject to all the limitations of self-report personality instruments (Hofstee, 1994). Future studies should strive to add measures of affect intensity and lability derived from parental and peer reports.

Fourth, specific measures of the environment were not examined in this study and so important risk factors such as postnatal trauma, inconsistent family environments, and abuse could not be added to the analyses. Finally, although genetic studies may be of fundamental importance in understanding the etiology of specific affective disorders and targeting treatment in clinical populations, these data are based on participants from a twin registry. Accordingly, these data may not generalize to clinical populations. Future studies of high-risk populations will be needed to identify which affect measures are relevant to vulnerability, which are epiphenomenal, and which are adaptive and compensatory.

Effective regulation of affective states may be achieved ultimately through interventions designed to increase intra-personal awareness of emotions. Recent research and theory on emotional intelligence (Salovey et al., 1995) illustrate that the ability to identify, distinguish, and appropriately label emotional experiences has interpersonal and mental health benefits that are evident even in later life (Ong and Bergeman, 2004). The capacity, however, to recruit appropriate regulatory processes, particularly under conditions of stress, may be based on predisposing genetic factors. The relevance of such factors to affect associations (i.e., lability, intensity, reactivity, complexity) represents important directions for future research.

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Conflict of interest
None of the authors have any conflict of interest in the context of this work.

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