

Does Resting Electroencephalograph Asymmetry Reflect a Trait? An Application of Latent State–Trait Theory

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Recent research on brain asymmetry and emotion treated measures of resting electroencephalograph (EEG) asymmetry as genuine trait variables, but inconsistency in reported findings and modest retest correlations of baseline asymmetry are not consistent with this practice. The present study examined the alternative hypothesis that resting EEG asymmetry represents a superimposition of a traitlike activation asymmetry with substantial state-dependent fluctuations. Resting EEG was collected from 59 participants on 4 occasions of measurement, and data were analyzed in terms of latent state–trait theory. For most scalp regions, about 60% of the variance of the asymmetry measure was due to individual differences on a temporally stable latent trait, and 40% of the variance was due to occasion-specific fluctuations, but measurement errors were negligible. Further analyses indicated that these fluctuations might be efficiently reduced by aggregation across several occasions.

When Hans Berger (1929) described the human electroencephalograph (EEG) for the very first time, he immediately expressed the hope that the waxing and waning of brain waves would give information about the neural basis of individual differences. Nearly 4 decades later, the arousal theory of extraversion by Eysenck (1967) stimulated an increased interest in the relationship between personality and spontaneous resting EEG, but initial find-

ings were inconsistent and soon raised questions about the definition of resting states during recording of the EEG (Gale, 1973, 1983). The state dependence of the spontaneous EEG made it difficult to use this measure as a marker for consistent and stable individual differences in tonic brain activation. This problem finally led to a decline of this research paradigm and to a shift of interest to the arousability concept of extraversion and its measurement of phasic brain activity (Bartussek, 1984; Stelmack, 1990; Stemmler & Meinhard, 1990).

Recently, the diathesis-stress model of anterior asymmetry and emotion by Davidson (1984, 1998b) and colleagues (e.g., Davidson & Tomarken, 1989) has aroused a new interest in using resting EEG as a measure of a biologically based stable individual differences (for recent applications, see Hagemann et al., 1999; Pauli, Wiederman, & Nickola, 1999; Wiedermann et al., 1999). However, apparent inconsistencies in findings (Hagemann, Naumann, Becker, Maier, & Bartussek, 1998; Heller & Nitschke, 1998; Reid, Duke, & Allen, 1998) and modest retest correlations of resting EEG asymmetry (Sutton & Davidson, 1997; Tomarken, Davidson, Wheeler, & Kinney, 1992) have raised concerns about the trait property of the EEG measures (Davidson, 1998b). The present article reports on an examination of some assumptions that underlie the empirical investigation of the model. To provide the relevant background, we review the model and its findings and outline the latent state–trait (LST) theory of Steyer (1987, 1988) and colleagues (Steyer & Schmitt, 1990) as the methodological framework for this investigation.

The Model of Anterior Asymmetry and Emotion and Basic Research Paradigms

Neuropsychological observations of affective consequences following brain lesions led to the hypothesis that both hemispheres

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are differently engaged in emotional functions (e.g., Gainotti, 1972; Robinson, Kubos, Starr, Rao, & Price, 1984). On the basis of these observations and additional psychophysiological evidence from experiments with normal participants, Davidson (1984, 1992b) and colleagues (e.g., Davidson & Tomarken, 1989) proposed a model of emotion in which each anterior hemisphere is part of a specific affective/motivational brain system. In this framework, the left anterior hemisphere is a major component within a neural network that is specialized for approach behavior; activation of this system is associated with the experience and expression of positive emotions. Conversely, the right anterior hemisphere is a major component within a brain circuit that is specialized for withdrawal behavior; activation of this system is associated with the experience and expression of negative emotions (Davidson, 1993; Wheeler, Davidson, & Tomarken, 1993).

One prediction derived from this model is that individual differences in asymmetrical activation levels of these systems might be a contributory factor in traitlike affective/motivational behaviors that constitute a person's affective style (Davidson, 1998a; Schaffer, Davidson, & Saron, 1983). A person with relative left-sided tonic activation of the anterior cortex might be characterized by a lowered threshold for the experience of positive emotions when an appropriate elicitor is present and by a stronger approach/activation motivation. In contrast, a person with relative right-sided activation might show an increased vulnerability for the experience of negative emotional states and a stronger withdrawal/inhibition tendency. In this view, the baseline asymmetry of anterior cortical activation is a trait that acts as a diathesis for emotion elicitors.

The empirical examination of this model has most frequently used the EEG as a measure of brain activation, with alpha activity (8–13 Hz) serving as the indicator of cortical deactivation (e.g., Creutzfeldt, 1995). Two different research paradigms have been used to study brain asymmetry and emotion. In the *state paradigm*, the EEG is recorded while different emotional states are elicited, and the subsequent analysis examines the shift of asymmetry between the emotional conditions. The results of several studies of this type supported the model (e.g., Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Fox & Davidson, 1988; Tucker, Stenslie, Roth, & Shearer, 1981; for reviews, see Davidson, 1995; Hagemann et al., 1998). In the *trait paradigm*, a baseline resting EEG is recorded first, and affective reactivity or related emotional traits are assessed in a second step. The subsequent analysis relates the EEG to the affective traits. A number of pioneering studies supported the diathesis-stress version of the model (e.g., Davidson & Fox, 1989; Schaffer et al., 1983; Tomarken, Davidson, & Henriques, 1990). Recent reviews of the research on resting EEG asymmetry and affective dispositions have suggested a need for reconsideration of the evidence (Hagemann et al., 1999; Heller & Nitschke, 1998; Reid et al., 1998; but see Davidson, 1998a). In the following, we provide an overview of empirical studies with a focus on the trait paradigm.

Resting EEG Asymmetry and Affective Dispositions

The last 2 decades have provided numerous reports of the association between the resting EEG and affective dispositions; the findings of 33 independent samples are presented in Table 1. All studies analyzed lateralized anterior activation and negative trait

affect/motivation tendency. Of the 33 samples, 14 showed a clear association between right-sided cortical activation and increased negative affectivity and withdrawal-inhibition strength (Allen, Iacono, Depue, & Arbisi, 1993; Bruder et al., 1997; Calkins, Fox, & Marshall, 1996; Davidson & Fox, 1989; Fox, Bell, & Jones, 1992; Henriques & Davidson, 1990; Henriques & Davidson, 1991; Jacobs & Snyder, 1996; Petruzzello & Landers, 1994; Petruzzello & Tate, 1997; Saletu et al., 1996; Tomarken et al., 1990; Wiedermann et al., 1999; and the large sample study with selected subjects of Tomarken & Davidson, 1994; Tomarken, Davidson, Wheeler, & Kinney, 1992; and Wheeler et al., 1993). The remaining 19 samples showed either no association, a significant relationship that was opposite to the prediction, or a mixture of confirming and disconfirming association patterns. Indicators of positive trait affect/motivation tendency were collected in 12 of the 33 samples, but only 4 samples showed a clear association between left-sided anterior cortical activation and increased positive affect and approach/activation strength (Calkins et al., 1996; Harmon-Jones & Allen, 1997; Petruzzello & Tate, 1997; and, again, the large sample with selected subjects of Tomarken, Davidson, Wheeler, & Kinney, 1992; and Wheeler et al., 1993). Variations in sample characteristics such as sex, age, or sample size do not appear to account for differences in the outcome of the studies (see Table 1).

To better understand the apparent inconsistency in findings, recent studies have focused on methodological factors such as length of the EEG database, the EEG reference, and the definition of the affective trait variables. Hagemann et al. (1998) recorded resting EEG and presented affective slides to obtain ratings of participants' emotional reactions. After applying different analysis procedures to their raw data, they observed that the choice of the particular EEG reference (common vertex vs. linked mastoids), the length of the EEG database (30 s vs. 8 min), and the computation procedure of the affectivity measures (with vs. without a global rating bias statistically removed) were important moderators of the asymmetry-affectivity relationship. Complementing this study, Reid et al. (1998) recorded resting EEG in two independent samples, each consisting of a group of depressed and a group of control participants. Their analyses indicated that the definition of the patient group (extreme scores on the Beck Depression Inventory, Beck, Ward, Mendelson, Mock, & Erbaugh, 1961; vs. clinical diagnosis), the length of the EEG database (30 s vs. 8 min), and the choice of the EEG reference (common vertex vs. linked mastoids vs. average reference) could also moderate the asymmetry-depression relationship. These studies pointed to particular procedural factors that might contribute to the inconsistency of findings in this area.

Differences in the reliability of asymmetry measures that were based on 30 s versus 8 min resting EEG may be one important contributor to such inconsistencies. Cronbach's alphas for the asymmetry measures are in the range of .80 to .90 when asymmetry is assessed with an 8-min resting EEG, which indicates good reliability for these measures (Hagemann et al., 1998; Reid et al., 1998; Sutton & Davidson, 1997; Tomarken, Davidson, Wheeler, & Kinney, 1992). On the other hand, an EEG database of 30 s yields asymmetry measures with mean parallel test correlations in the .50s, suggesting insufficient reliability for this particular EEG database (Hagemann et al., 1998). Such findings suggest that different analysis protocols can lead to different results when

Table 1
Overview of Studies on Resting EEG Asymmetry and Affective/Motivational Traits

Authors	Participants (mean age or range in y or m)	Recording condition (EEG reference, length)	Affective trait measures	EEG retest	Results	
					positive affect/approach	negative affect/withdrawal
Allen et al. (1993)	8 women	Eyes closed (Cz; 32 s)	Depression vs. CG (CD)	No		ant: R post: <i>ns</i>
Bell et al. (1998) ^a	21 women (<i>M</i> = 39 y)	Eyes open and closed (A1 + A2; 2 min)	Depression vs. CG (CD; SCL-90-R)	2 occasions (1-week interval)		Occasion 1: ant: R Occasion 2: ant: <i>ns</i>
Bruder et al. (1997)	35 men 35 women (<i>M</i> = 37 y)	Eyes open and closed (Cz, nose reference; 6 min)	Anxious depression vs. nonanxious depression (CD)	No		ant: R post: R
Calkins et al. (1996)	66 participants (both sexes; <i>M</i> = 4 and 9 m)	Eyes open (AR; 2 min; 4-6 Hz)	Nonanxious depression vs. CG (CD)	No		ant: <i>ns</i> post: L
Davidson & Fox (1989)	13 girls (<i>M</i> = 10 m)	(Cz; 30 s; 6-8 Hz)	Positive and negative affectivity (BO)	No		ant: R post: <i>ns</i>
Debener et al. (2000) ^b	12 men 25 women (<i>M</i> = 47 y)	Eyes open and closed (A1 + A2; 8 min)	Affective reaction after separation from mother (BO)	No		ant: R post: <i>ns</i>
Flor-Henry & Koles (1984) ^c	197 participants (half men; half women; <i>M</i> = 34 y)	Eyes open or closed (Cz; 2 × 2-3 min)	Depression vs. CG (CD)	2 occasions (2-4 weeks interval)		Aggregate: ant: R post: <i>ns</i> Single occasions: ant: <i>ns</i> post: <i>ns</i>
Fox et al. (1992) ^{d, e}	Sample 1: 8-10 boys 4-13 girls (<i>M</i> = 14 and 24 m)	Eyes open (Cz; 4 min; 6-9 Hz)	Depression vs. mania (CD; <i>N</i> = 137; eyes open) Mania vs. CG (CD; <i>N</i> = 135 eyes closed)	No		ant: R post: L ant: L post: <i>ns</i>
	Sample 2: 6 boys, 7 girls (<i>M</i> = 7 to 12 m)	Eyes open (Cz; 2 min; 6-9 Hz)	Affective reaction after separation from mother (BO)	2 occasions (10 m interval)		Occasion 1: ant: R post: <i>ns</i> Occasion 2: ant: <i>ns</i> post: <i>ns</i>
Gotlib et al. (1998)	77 women	Eyes open and closed (Cz; 8 min)	Affective reactivity to music (<i>N</i> = 59)	No		ant: <i>ns</i>
Hagemann et al. (1998)	15 men, 22 women (<i>M</i> = 25 y)	Eyes open and closed (Cz, A1 + A2; 8 min)	Depression vs. CG (CD; <i>N</i> = 77) Affective reactivity to slides	No		ant: R ant: L post: <i>ns</i>

(table continues)

Table 1 (continued)

Authors	Participants (mean age or range in y or m)	Recording condition (EEG reference, length)	Affective trait measures	EEG retest	Results	
					positive affect/approach	negative affect/withdrawal
Hagemann et al. (1999)	12 men 24 women ($M = 23$ y)	Eyes open and closed (A1 + A2; 8 min)	PANAS	No	ant: <i>ns</i> post: <i>ns</i>	ant: L post: <i>ns</i>
Harmon-Jones & Allen (1997)	36 women	Eyes open and closed (Cz; 4 min)	BIS/BAS by E, N	No	ant: <i>ns</i> post: <i>ns</i>	ant: <i>ns</i> post: <i>ns</i>
Harmon-Jones & Allen (1998) ^f	15 boys, 11 girls ($M = 13$ y)	Eyes open and closed (Cz; 6 min)	BIS/BAS Scales Aggression Questionnaire PANAS-C	No	ant: L post: <i>ns</i> ant: L post: <i>ns</i> ant: <i>ns</i> post: <i>ns</i>	ant: <i>ns</i> post: <i>ns</i> ant: L post: <i>ns</i> ant: <i>ns</i> post: <i>ns</i>
Heller et al. (1997)	18 men, 22 women	Eyes open and closed (A1 + A2; 8 min)	STAI-Trait	No	ant: <i>ns</i> post: <i>ns</i>	ant: <i>ns</i> post: <i>ns</i>
Henriques & Davidson (1990)	3 men, 11 women ($M = 36$ y)	Eyes open and closed (Cz, A1 + A2, AR; 1 min)	Depression vs. CG (CD)	No	ant: R post: L	ant: R post: L
Henriques & Davidson (1991)	11 men, 17 women ($M = 40$ y)	Eyes open and closed (Cz, A1 + A2, AR; 1 min)	Depression vs CG; (CD)	No	ant: R post: <i>ns</i>	ant: R post: <i>ns</i>
Jacobs & Snyder (1996)	40 men (range 18-53 y)	Eyes closed (A1 + A2; 5 min)	PANAS	No	ant: <i>ns</i>	ant: R
Kemali et al. (1981)	87 participants	(ipsilateral A1, A2; 120-160 s)	BDI Depression vs. CG (CD)	No	ant: <i>ns</i> post: R	ant: R post: <i>ns</i>
Nitschke et al. (1999)	21 men 33 women ($M = 18$ y)	Eyes open and closed (A1 + A2; 8 min)	Anxiety arousal vs. anxious apprehension (MASQ, PSWQ) Depression vs. CG (MASQ)	No	ant: R post: <i>ns</i>	ant: R post: <i>ns</i>
Pauli et al. (1999) ^e	3 men, 5 women ($M = 24$ y)	Eyes open or closed (A1 + A2; 2 × 2 min)	BDI (eyes open)	No	ant: <i>ns</i>	ant: R
Perris et al. (1981)	24 men 36 women ($M = 46$ y)	Eyes closed (ipsilateral A1, A2; ≥ 5 min)	BDI (eyes closed)	No	ant: R post: <i>ns</i>	ant: R post: <i>ns</i>
Petruzzello & Landers (1994)	19 men ($M = 23$ y)	Eyes open (ipsilateral A1, A2; 66 s)	Depression vs. CG (CD)	No	ant: <i>ns</i> post: L	ant: <i>ns</i> post: L
Petruzzello & Tate (1997)	15 men 5 women ($M = 23$ y)	Eyes closed (A1 + A2; 8 min)	STAI-Trait Affective reactivity to exercise (STAI-State)	No	ant: R	ant: R
Pollock & Schneider (1990)	16 men, 16 women ($M = 64$ y)	Eyes closed (A1 + A2, 3 min)	Affective reactivity to exercise (ADACL)	No	ant: L post: <i>ns</i>	post: <i>ns</i> ant: <i>ns</i>
Reid et al. (1998) ^d	Sample 1: 36 women ($M = 19$ y)	Eyes open and closed (Cz, A1 + A2, AR; 8 min)	Depression vs. CG (CD)	No	ant: <i>ns</i> post: <i>ns</i>	ant: <i>ns</i> post: <i>ns</i>
Saletu et al. (1996)	Sample 2: 27 women ($M = 28$ y) 118 women ($M = 51$ y)	Eyes closed (A1 + A2, 3 min)	Sample 1 Depression vs. CG (BDI) Sample 2 Depression vs. CG (CD) Depression vs. CG (CD)	No	ant: <i>ns</i> post: <i>ns</i>	ant: <i>ns</i> post: L ant: R post: <i>ns</i>

Table 1 (continued)

Authors	Participants (mean age or range in y or m)	Recording condition (EEG reference, length)	Affective trait measures	EEG retest	Results	
					positive affect/approach	negative affect/withdrawal
Schaffer et al. (1983) ^c	5 men, 10 women,	Eyes open or closed (Cz; 4 × 30 s)	Depression vs. CG (BDI, eyes open)	No		ant: <i>ns</i> post: <i>ns</i> ant: R post: <i>ns</i>
Sutton & Davidson (1997) ^g	23 men, 23 women (range 18–22 y)	Eyes open and closed (A1 + A2; 8 min)	PANAS	2 occasions (6 weeks interval)	ant: <i>ns</i>	post: <i>ns</i> ant: <i>ns</i>
Tomarken & Davidson (1994); Tomarken, Davidson, Wheeler & Doss (1992); Wheeler et al. (1993) ^h	initially 90 women (range 17–21 y)	Eyes open and closed (A1 + A2; 8 min)	BIS/BAS Scales	2 occasions (3 weeks interval)	ant: L post: <i>ns</i>	ant: R post: <i>ns</i> ant: R post: <i>ns</i>
Tomarken et al. (1990)	32 women (range 17–54 y)	Eyes open (Cz; 30 s)	STAI-Trait (N = 42)	2 occasions (3 weeks interval)	ant: L	ant: R post: <i>ns</i>
Wiedermann et al. (1999)	9 men, 39 women (M = 36 y)	Eyes open and closed (Cz; 4 min)	PANAS (N = 15–36)		ant: L	post: <i>ns</i> ant: R post: <i>ns</i>
			Affective reactivity to films (N = 26)	No	ant: L	post: <i>ns</i> ant: R post: <i>ns</i>
			Affective reactivity to films	No	ant: <i>ns</i>	ant: R post: <i>ns</i>
			Panic disorder vs. CG (CD)	No	post: <i>ns</i>	ant: R post: <i>ns</i>

Note. The total sample size is reported for each study. The electroencephalograph (EEG) was always recorded in a resting situation with eyes open or closed. The asymmetry measurement was always based on the alpha band (8–13 Hz) power of an EEG unless otherwise noted. The EEG was referenced either to common vertex (Cz), linked ears or mastoids (A1 + A2), ipsilateral ears or mastoids (A1, A2), or average reference (AR). The asymmetry measure was always based on EEG data aggregated across the recording condition (eyes open and closed) unless otherwise noted. Affective traits were assessed either by a clinical diagnosis (CD) of affective disorder, by behavioral observations (BO), or with standard self-report instruments such as the Activation Deactivation Adjective Check List (ADACL; Thayer, 1986), Aggression Questionnaire (Buss & Perry, 1992), Beck Depression Inventory (BDI; Beck et al., 1961), Behavioral Inhibition/Behavioral Activation Scales (BIS/BAS Scales; Carver & White, 1994), Mood and Anxiety Symptom Questionnaire (MASQ; Watson et al., 1995), Penn State Worry Questionnaire (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990), the trait version of the Positive and Negative Affect Scales (PANAS; Watson, Clark, & Tellegen, 1988), the trait version of the State-Trait Anxiety Inventory (STAI-Trait; Spielberger et al., 1970), or the Symptom Check List—90—Revised (SCL-90-R; Derogatis, Lipman, & Covi, 1973). Significant ($\alpha = .05$) results are reported separately for positive and negative dispositions and for anterior (ant) and posterior (post) regions; L indicates a positive relation between emotional/motivational trait magnitude and relative left-sided cortical activation, and R indicates a positive relation between trait and relative right-sided activation. y = years; m = months; CG = control group; E = extraversion; N = neuroticism.

^a EEG was recorded on two occasions, and data were analyzed separately for each occasion. ^b EEG was recorded on 2 occasions, and data were either aggregated across both occasions for analysis or analyzed separately for each occasion. ^c Data were analyzed separately for the eyes open and closed resting conditions. ^d This work comprised two independent studies. ^e In Sample 1, EEG was recorded on two occasions and data were analyzed separately for each occasion; in Sample 2, EEG was recorded on six occasions and data were aggregated across occasions. ^f The Aggression Questionnaire is assumed to measure a negative affect that is associated with an approach tendency; hence, the same finding was listed in the categories for positive affect/approach and negative affect/withdrawal. ^g EEG was recorded on 2 occasions and data were aggregated across both occasions for analysis. ^h The EEG of initially 90 participants was recorded on 2 occasions. From this pool, Wheeler et al. (1993) selected 26 participants and Tomarken, Davidson, Wheeler, and Doss (1992) selected 15–36 participants with stable asymmetry for analysis; only 47 unselected participants were available for this particular analysis of Tomarken and Davidson (1994). All analyses were performed on EEG data that were aggregated across both occasions.

applied to the same raw data, and methodological or technical factors may contribute to the inconsistency of the evidence. However, the identified methodological factors cannot fully explain the discrepancy of previous findings. Table 1 suggests that none of these factors (EEG reference, EEG database length and asymmetry measure reliability, operationalization of the affect disposition) can reliably predict the outcome of a study. Heller and Nitschke (1998) have recently emphasized the importance of considering subtypes of depression and anxiety and their comorbidity when confronted with conflicting results (see also Reid et al., 1998, for a discussion of this issue). The divergence of findings in nonclinical samples suggests that factors other than diagnostic problems might also contribute to the variability of results.

State Dependence of Resting Alpha Asymmetry

The alpha rhythm of the EEG is sensitive to seemingly minor manipulations of the recording situation. For example, alpha is most pronounced when a person is in a resting state with eyes closed but may be blocked when the person merely opens his or her eyes (see Creutzfeldt, 1995). When the eyes are kept closed, the alpha rhythm shows a characteristic waxing and waning (alpha spindles) that may be interpreted as continuous shifts of cortical activation. The apparent dynamics of the spontaneous EEG are not surprising, as the brain is thought to be a complex system that regulates its oscillating activities in a nonlinear fashion (see Basar & Haken, 1998; Nunez, 1995, for dynamic theories of brain waves).

Stable individual differences in the pattern of human alpha activation may be revealed by spectral analysis of the spontaneous EEG. The alpha peak frequency, bandwidth, and various spatial properties tend to be constant characteristics of the individual. These parameters have been associated with a spectral fingerprint of the individual brain (Nunez, 1981, pp. 25–34) and may serve as an index of individual differences. A number of recent studies have examined the temporal stability of resting asymmetry.

Tomarken, Davidson, Wheeler, and Kinney (1992) assessed the EEG alpha asymmetry measure on two occasions 3 weeks apart and reported a retest correlation of midfrontal asymmetry in the range of .53 to .66 and corresponding correlations for anterior temporal asymmetry in the range of .71 to .72. A similar finding was obtained later by Sutton and Davidson (1997), who collected data with a retest interval of 6 weeks and reported, on average, retest correlations of resting asymmetry measures of .57 across the whole scalp. Notably, both of these studies reported high reliability of these measures, suggesting that the low retest correlations are not due to measurement errors. Papousek and Schuller (1998) collected resting EEG for 4 min on two occasions spaced between 2 and 4 weeks apart and reported retest correlations for the alpha band in the range of .22 to .35 for frontopolar, .51 to .53 for midfrontal, .52 to .67 for anterior temporal, and .67 to .72 for parietal sites. Debener et al. (2000) recorded resting EEG from depressive and normal control participants on two occasions 2 to 4 weeks apart. For depressed patients, the retest correlations of asymmetry measures were around zero (between $-.22$ and $.05$) for anterior sites and .57 for the parietal region. For nondepressed individuals, the correlations were between .46 and .69. Finally, Rosenfeld, Baehr, Baehr, Gotlib, and Ranganath (1996) reported day-to-day fluctuations of the resting asymmetry of depressed

outpatients, and Fox et al. (1992) reported a low stability of resting EEG asymmetry in children during the 1st year of life. Compared with the temporal stability of many trait measures, these resting EEG asymmetry measures appear to be too low to reflect only a stable individual-differences variable (see the discussions in Davidson, 1993, 1998b; and Tomarken, Davidson, Wheeler, & Kinney, 1992). Most personality traits, corrected for unreliability, have annual stability coefficients of .98–.99 (see Costa & McCrae, 1992).

In this context, it is interesting to note that some of the researchers who collected the EEG on two occasions analyzed their data separately for each occasion but failed to replicate their results within the same sample. For example, Bell, Schwartz, Hardin, Baldwin, and Kline (1998) recorded resting EEG from depressive and normal control participants on two occasions 1 week apart. Whereas depressed participants showed relative right-sided anterior asymmetry compared with controls on the first occasion, no such difference occurred on the second occasion. A similar observation was also reported by Fox et al. (1992) in a sample of normal children who were repeatedly tested at age 14 and 24 months. Whereas children with right-sided anterior resting EEG asymmetry cried after separation from their mother at the first occasion of measurement, no such association was present at the second occasion. Complementing these findings, Debener et al. (2000) recorded resting EEG from depressive and normal control participants on two occasions between 2 and 4 weeks apart. Although there was an association between right-sided anterior activation and depression if the asymmetry measures were aggregated across both occasions, no such relation could be observed within each separate occasion. Heller and Nitschke (1998) recently suggested that the identification of subtypes of depression and anxiety and their comorbidity may explain some of the inconsistencies in the literature. However, the finding that the associations in question are difficult to replicate within the same sample suggests that additional factors might also contribute to the inconsistency.

The inconsistency of findings and the rather low temporal stability of the asymmetry measure raise concerns about the utility of frontal brain asymmetry, measured with a baseline EEG, as an index of a trait underlying stable individual differences in emotion. In an attempt to explain these findings, Tomarken, Davidson, Wheeler, and Kinney (1992) suggested that there might be a latent trait asymmetry of frontal activation that is superimposed on occasion-specific fluctuations of lateralized activity, or, as they noted, “resting EEG asymmetry may reflect the joint contribution of both stable individual differences and more situational state-like factors” (p. 589; this view was also expressed by Davidson, 1992a, 1993; and Wheeler et al., 1993; a similar proposal was recently put forward by Rosenfeld, 2000). In total, the divergence of findings and the low retest correlations of resting asymmetry suggest that the state dependence of the EEG could be a distorting factor for the trait paradigm.

However, only a few pioneering studies have paid attention to this potential source of error in their experimental and analytical designs. For one large study on resting brain asymmetry, Kang et al. (1991), Tomarken, Davidson, Wheeler, and Kinney (1992), Tomarken and Davidson (1994), Tomarken, Davidson, Wheeler, and Doss (1992), and Wheeler et al. (1993) collected the resting EEG of, initially, 90 participants on two occasions 3 weeks apart. To reduce the state variance and to increase the trait variance of

asymmetry, Wheeler et al. (1993) selected for their analysis of affective reactivity those participants whose standardized resting asymmetry measures on both occasions showed no difference greater than one third of a standard deviation. These participants were labeled as the stable group ($n = 26$), and the asymmetry measures were averaged across both occasions to further reduce their state variance. Tomarken and Davidson (1994) selected for an analysis of coping styles only those persons whose asymmetry measure scored in the top or bottom 25th percentile on both occasions. These participants were assigned to stable and extreme groups ($n_s = 12$ and 13), and the asymmetry measures were also averaged across both occasions. Finally, Tomarken, Davidson, Wheeler, and Doss (1992) used both selection procedures for their analysis of affective dimensions. In other investigations, Sutton and Davidson (1997) and Debener et al. (2000) averaged the resting asymmetry measure across two occasions (but no further selection procedures were applied). Finally, Fox et al. (1992) averaged the resting asymmetry of children across a total of six measurement occasions to compensate for instabilities due to maturation effects. Recently, Davidson (1998b) suggested that the lack of control of state-dependent fluctuations in studies using only one occasion for the measurement of resting asymmetry may explain null findings or contradictory results; consequently, he stressed the importance of assessing the asymmetry measure on several occasions to obtain conclusive findings.

Such approaches to controlling for occasion-specific fluctuations of the resting EEG asymmetry are an important first step in developing valid experimental and analytical designs for the investigation of resting EEG asymmetry. However, the procedures used—and their underlying assumptions—might be problematic.

1. All these procedures—like all other attempts to investigate the diathesis-stress model of anterior asymmetry and emotion with the resting EEG—rely on the assumption that resting EEG asymmetry measures reflect, at least in a substantial part, a *stable trait*. This basic assumption has not been adequately tested yet but is challenged by the inconsistency of findings and the low retest correlation of the asymmetry measure.

2. To maintain the assumption of a stable trait asymmetry in spite of the emerging evidence of modest stability of measures, researchers introduced a second postulate. The control procedures rely on the explicit assumption that, in addition to the stable latent trait, there are also substantial *statelike factors* that contribute to the measure (e.g., Tomarken, Davidson, Wheeler, & Kinney, 1992; Wheeler et al., 1993). However, the low retest correlations of the measures might alternatively be explained by a temporal instability of the trait, without the additional assumption of occasion-specific effects. In this particular case, the selection and aggregation procedures would be inadequate.

3. In the case of substantial occasion-specific variance in the data in addition to a stable latent trait, aggregation across several occasions may reduce the state variance and increase the trait variance (e.g., Epstein, 1983; Schmitt & Steyer, 1990). As it is not known how much of the variance of the asymmetry measure would be determined by such state-specific influences in this particular case, it is not obvious that an aggregation across only two occasions (e.g., Debener et al., 2000; Sutton & Davidson, 1997; Tomarken & Davidson, 1994) would be sufficient to control it.

4. Finally, the selection of participants with similar asymmetry measures on two occasions (e.g., Tomarken, Davidson, Wheeler,

& Doss, 1992; Wheeler et al., 1993) does not necessarily imply that these participants have a stable asymmetry; alternatively, the asymmetry of these persons may show a fluctuation across time similar to the asymmetry of unstable persons but may have just happened to be similar on two occasions by chance; thus, this selection procedure may be inadequate. The implicit assumption underlying this procedure is that the stability of frontal asymmetry is a stable property of the person. This assumption has not been examined, either.

Thus, the two main assumptions of the trait paradigm are (a) that there is a stable latent trait of resting EEG asymmetry and (b) that this trait is superimposed on occasion-specific fluctuations of lateralized activation. The subsidiary assumptions of the procedures to control the occasion specificity are that aggregation across two occasions is sufficient and that the change of asymmetry between two occasions is a stable property of the person. As these assumptions have not yet been examined, the validity of the trait paradigm—and the control procedures to reduce the presumed occasion specificity of asymmetry—has not been fully established.

If there is a stable trait component of the asymmetry measure but if it is rather small compared with the occasion-specific fluctuations, then the typical experimental design with only one occasion of measurement may often lead to invalid conclusions, which in turn may explain the inconsistency of findings. Alternatively, if the latent asymmetry trait is temporally unstable, then it might not be suited for the explanation of stable and consistent affective behavior; an instability of trait asymmetry might explain the critical findings as well. Thus, the available data are not conclusive, and these problems need to be addressed in empirical research. The necessary methodological framework to decompose a variable into its latent state and trait components and, hence, to investigate most of these questions was developed by Steyer (1987, 1988) and colleagues (Steyer & Schmitt, 1990) and was labeled the LST theory. In the next section, we give a brief overview of some basic LST models that can be used to address a number of the questions concerning the viability of resting EEG asymmetry as an index of a trait.

Models of LST Theory

The LST theory is an extension of classical test theory and allows the decomposition of a manifest (observed) random variable into latent (not observed) constituents such as state, trait, and measurement error components. To identify the variances of latent variables in empirical applications of this theory, the manifest variables have to be measured with at least two indicators, (i), on at least two occasions of measurement, (k). On the basis of theoretical considerations, different assumptions on the nature of the manifest variable may be represented with different models. Figures 1, 2, and 3, illustrate some basic models for the case of two indicators on four occasions.

In a *latent trait model* (Figure 1), all eight manifest variables (Y_{ik}) are decomposed into a common latent trait (T) and into measurement errors (e_{ik}) that are specific for each variable (Y_{ik}). Hence, this model assumes that there are no effects of the situation or the person-situation interaction on the manifest variables. In contrast, an *LST model* (Figure 2) is based on a two-step decomposition. In a first step, the two manifest variables (Y_{ik}) within each

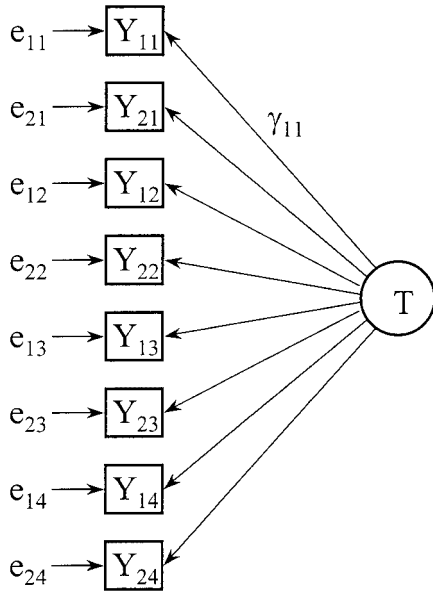


Figure 1. Latent trait model for two indicators (*i*) in four occasions (*k*) of measurement. Y_{ik} are manifest variables, e_{ik} are latent errors, and T is the latent trait. For simplicity, all factor loadings (γ_{ik}) are set equal to 1.

occasion of measurement (*k*) are decomposed into a common latent state (S_k) and into measurement errors (e_{ik}) that are specific for each variable (Y_{ik}). In a second step, all four states (S_k) are decomposed into a common latent trait (T) and into state residuals (SR_k) that are specific for each occasion. These residuals comprise the effects of the situation and the interaction effects of person and

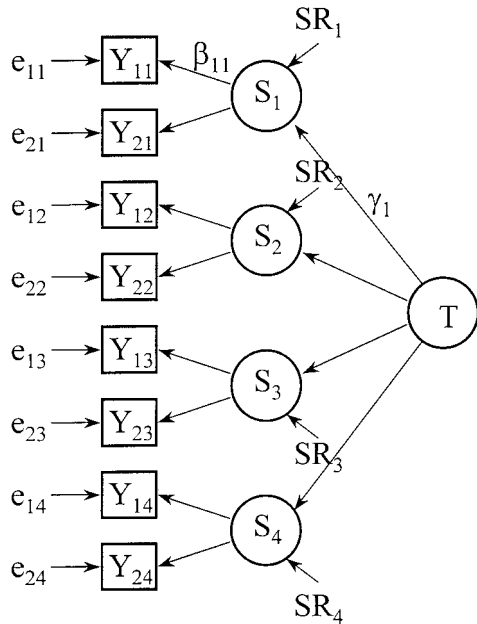


Figure 2. Latent state-trait model for two indicators (*i*) in four occasions (*k*) of measurement. Y_{ik} are manifest variables, e_{ik} are latent errors, S_k are latent states, T is the latent trait, and SR_k are latent state residuals. For simplicity, all factor loadings β_{ik} and γ_k are set equal to 1.

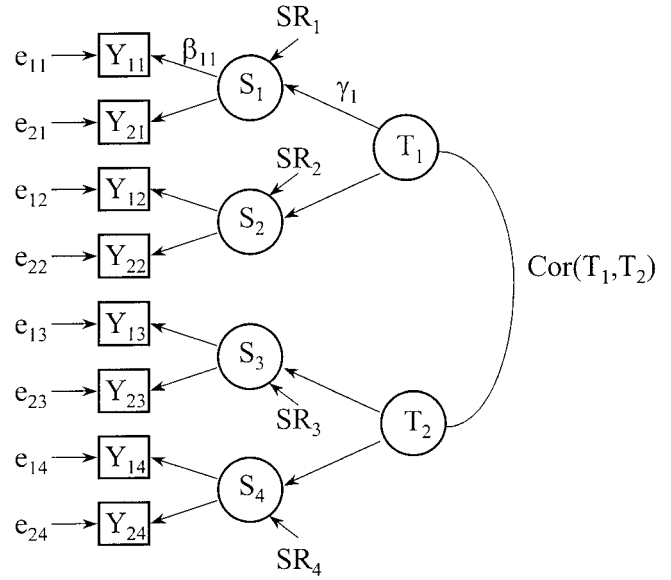


Figure 3. Latent state-trait change model for two indicators (*i*) in four occasions (*k*) of measurement. Y_{ik} are manifest variables, e_{ik} are latent errors, S_k are latent states, T_1 is the latent trait of the first two occasions and T_2 is the latent trait of the last two occasions, and SR_k are latent state residuals. For simplicity, all factor loadings β_{ik} and γ_k are set equal to 1. Cor = correlation.

situations on the latent state. Finally, an *LST change model* (Figure 3) is based on a similar two-step decomposition of the observed variable. As before, this model decomposes the manifest variables (Y_{ik}) into latent states (S_k) and measurement errors (e_{ik}). In contrast to the former model, however, it does not assume that the four latent states (S_k) measure a perfectly stable latent trait across the whole time period that is covered by the measurement occasions. Rather, it allows for different but correlated traits for the first and last two occasions.

These competing models and their parameters may be identified and tested on the basis of empirical data with standard techniques of structural equation modeling (for detailed formal representations of the LST framework, see Schmitt & Steyer, 1993; Steyer, Ferring, & Schmitt, 1992; Steyer & Schmitt, 1990; for recent developments, see Eid, 2000; Eid & Hoffmann, 1998; for the latest reviews, see Steyer, 1998; Steyer, Schmitt, & Eid, 1999).

The Present Study

In the majority of studies on tonic activation asymmetry and affective style, EEG was recorded on only one occasion of measurement, and the assumption was made that the resulting measures of asymmetry would be a sufficient proxy for a trait (e.g., Hagemann et al., 1998; Reid et al., 1998; Tomarken et al., 1990; see Table 1). Hence, these studies implicitly assumed that occasion-specific fluctuations (state residuals) would be of negligible importance, and thus a *single-trait model* of resting EEG asymmetry may represent the underlying assumption. In contrast, the few studies that measured resting EEG on two occasions explicitly stated that occasion-specific shifts of lateralized activation were of concern but that a temporally stable latent trait is

reflected by the resting asymmetry as well (e.g., Tomarken & Davidson, 1994; Tomarken, Davidson, Wheeler, & Kinney, 1992; Wheeler et al., 1993). Thus, this assumption may be represented by an LST model of resting EEG asymmetry. Finally, these authors assumed that the low retest correlations of asymmetry measures were due to statelike fluctuations that are superimposed on a stable latent trait. However, these correlations may also be explained by temporal instability of the trait; this latter assumption may be represented by an LST change model of resting asymmetry. The first aim of the present study is to apply the LST theory to EEG data collected on four occasions and to test these three models of resting asymmetry.

If these analyses suggest that there is a substantial occasion specificity of resting EEG asymmetry, these fluctuations would have to be controlled in empirical research. The second aim of the present study is to examine the efficacy of control procedures such as the selection of particular asymmetry measures on two occasions (e.g., Tomarken, Davidson, Wheeler, & Doss, 1992; Wheeler et al., 1993) or the aggregation of EEG data across several occasions (e.g., Debener et al., 2000; Sutton & Davidson, 1997; Tomarken & Davidson, 1994).

Method

Participants

The sample for the present study was drawn from the student population of the Universität Trier, Trier, Germany. All participants were recruited through E-mail advertising. A total of 63 right-handed participants were scheduled for data acquisition. Because of technical problems, the data sets of 4 participants could not be completed. Because no missing values were accepted in any of the analyses, these participants were excluded from the sample. The remaining 59 participants defined the sample of the present study, which consisted of 29 men (mean age = 25 years, $SD = 3.2$ years, range = 21–36 years) and 30 women (mean age = 23 years, $SD = 3.4$ years, range = 19–34 years). All participants were compensated with DM 200 (~\$100) after the data acquisition was completed.

Procedure

All participants were assessed individually on four occasions of measurement each separated by 4 weeks. After arriving at the laboratory on the first occasion, participants were informed that the purpose of the experiment was to investigate brain waves of persons in resting states and persons who are looking at visual stimuli. The participants gave written consent and were informed that they could leave the experiment at any time. Subsequently, handedness of the participants was assessed with the Edinburgh Inventory (Oldfield, 1971).

The general procedure was identical on each measurement occasion. At the beginning of the session, each participant was seated in an electrically shielded EEG cabin, and electrodes were applied for the measurement of electrooculogram (EOG) and EEG. The respective signals were recorded during twelve 1-min resting baseline periods. Six baselines were recorded with eyes open, and six with eyes closed. There was a 1-min break after four subsequent baseline recordings. On the first occasion of measurement, each participant was randomly assigned to one of two counterbalanced orders of the eyes open (O) and eyes closed (C) conditions (OCCO–COOC–OCCO or COOC–OCCO–COOC). On the second and third occasions, the respective other eyes order was assigned, and on the fourth occasion, the same order as on the first occasion was applied. The participants were instructed by microphone to be restful and when to open and close their eyes. EOG and EEG recording of each single baseline was

initiated by the experimenter after the instruction was given. After the EEG was recorded, the participants completed subsequent tasks involving the presentation of visual stimuli, which will be reported elsewhere (Hagemann, Hewig, Seifert, Naumann, & Bartussek, 2002).

EEG Recording and Quantification

EEG was recorded with the ECI-Electrocap system (Electro-Cap International, Inc., Eaton, OH; Blom & Anneveldt, 1982) from 32 sites of the international 10/10 system (Chatrian, Lettich, & Nelson, 1985), including the earlobes (A1, A2). All sites were referenced to vertex (Cz). A bipolar horizontal EOG was recorded from the epicanthus of each eye, and a bipolar vertical EOG was recorded from supra- and infraorbital positions of the right eye. The EEG was recorded with tin electrodes, and the EOG was recorded with silver/silver chloride (Ag/AgCl) electrodes (Polich & Lawson, 1985). Prior to the placement of electrodes, the expected electrode sites on the participant's scalp and face were cleaned with alcohol and gently abraded. All impedances of the EEG electrodes were below 5 k Ω , and the differences in impedance between homologous sites were below 1 k Ω .

EEG and EOG were amplified with two 32-channel SynAmps Model 5083 amplifiers (input impedance: 10 M Ω ; Neuroscan, Inc., Sterling, VA) in alternating-current mode. The pass band was set to 0.3–40 Hz (–12 dB/octave rolloff); the signals were digitized at 200 Hz and stored to hard disk for later analysis.

After data acquisition was accomplished, each combined EOG and EEG record was subjected to an off-line artifact control procedure. Because neuropsychological evidence suggests that the severity of poststroke depression is correlated with the lesion's proximity to the frontal poles (Robinson et al., 1984) and because recent research has reported on frontopolar data in investigations of EEG resting asymmetry (Papousek & Schuler, 1998; Sutton & Davidson, 1997), frontopolar sites were included in the present study. Because of the proximity of these sites to the eyes, even minor blinks and eye movements may result in substantial distortions of frontopolar alpha activity (Hagemann & Naumann, 2001); thus, a conservative control procedure was used in the present study. The continuous EOG and EEG recordings were visually inspected, and each portion of the EEG data that showed ocular, muscular, or technical artifacts in any channel was rejected for this and all simultaneously recorded channels (Barlow, 1986). This procedure resulted in a mean rejection rate of 47%, 43%, 45%, and 49% of all EEG epochs (see below) for the first, second, third, and fourth measurement occasions, respectively.

After the data were edited, the EEG was transformed to current source density (CSD) derivations to provide a reference-free measurement of local site activity (Hjorth, 1975, 1980; Katznelson, 1981). The rationale for this procedure was the recent observation that the measures of frontal EEG asymmetry are specific for the particular reference montage (e.g., Hagemann et al., 1998; Reid et al., 1998). To account for this problem, some authors have recommended the CSD derivation for asymmetry research (Hagemann et al., 1998; Nunez, 1981; Tomarken, Davidson, Wheeler, & Kinney, 1992). To compute Hjorth's (1980) estimates of the radial current density at each site, we measured the interelectrode distances from the ECI-Electrocap's actual electrode positions when a medium-sized real electrocap was positioned on an average-sized realistic model head (for a similar procedure, see Curran, Tucker, Kutas, & Posner, 1993). Each scalp electrode was subsequently defined to be a target site in the following computation procedure.

All sites within a radius of 10 cm around a target site were defined as next neighbors of the target (see Hjorth, 1980). Separately for the set of each target site and its next neighbors, the interelectrode distances between target and neighbors defined a distance vector, which was inverted and rescaled so that the sum of its elements equaled one. We computed the CSD of the target site by subtracting all vector-weighted neighbor site channels from the target site channel of the original Cz referenced data.

This procedure was repeated for each scalp electrode as target, yielding CSD derivations (μV) of the EEG that may be interpreted as the potential difference between the target electrodes and a weighted average of their surrounding next neighbor electrodes (weighted with the reciprocal of the distance between target and neighbor). Hjorth's (1980) procedure accounts for (a) different distances between target and neighbors and (b) different numbers of neighbors for each target.

In addition to the CSD derivations and to facilitate a numerical comparison with previous findings, we computed a computational linked ears (A1 + A2) reference by averaging the A1-Cz and A2-Cz channels and subtracting this average from the data of the Cz-referenced EEG channels (Davidson, 1988). This procedure prevents a shift of the effective reference site from midway between the ears to the ear with the lower electrode resistance, which may be introduced with a physically linked ears reference and might distort EEG asymmetry (Miller, Lutzenberger, & Elbert, 1991). All further analyses were performed separately for the CSD and the A1 + A2 derivations and separately for each occasion of measurement.

Prior to spectral analysis, the new EEG derivations were broken down to nonoverlapping epochs of 2.56 s, and an odd-even split of the epochs was performed. This procedure resulted in two parallel data sets for both the CSD and the A1 + A2 derivations and facilitated a later LST analysis of all measures. All epochs were extracted through a Hanning window (10%) to prevent spurious estimates of spectral power (Dummermuth & Molinari, 1987). A fast Fourier transform was performed on all epochs to compute spectral power (μV^2) spaced at intervals of 0.39 Hz. Power values were averaged across all epochs and across the alpha band (8–13 Hz) and converted to power density ($\mu\text{V}^2/\text{Hz}$) separately for both epoch sets of the CSD and of the A1 + A2 derivations on each occasion of measurement. Alpha power density values were natural logarithmically (\ln) transformed to normalize the data (Gasser, Bächer, & Möcks, 1982), and an asymmetry metric was computed as the right minus left difference of \ln power density of homologous sites (e.g., \ln power density F4 – \ln power density F3; see Davidson, 1988). Because asymmetry measures tend to show low absolute values and variances, they were multiplied by $10^{0.5}$ to prevent an excess of places behind the decimal point in the presentation of results.¹ Finally, this procedure resulted in two *parallel asymmetry measures* for each scalp region, separately for the CSD and the A1 + A2 derivations and separately for each occasion of measurement. To obtain measures of asymmetry that are aggregated on subsequent levels, (a) we averaged both parallel asymmetry measures within each occasion to yield *total asymmetry measures* for each occasion, and (b) we averaged the total measures across the first two or the last two occasions of measurement to obtain *aggregated asymmetry measures*. The artifact screening, rereferencing, and spectral analysis were performed with Scan 3.0 (Module Edit386, Version 3.2; Neurosoft, 1993); the further computation of asymmetry measures was performed with SPSS for Windows (Version 7.5.2G; SPSS, 1997).

Data Analysis

The main statistical analyses were performed for the frontopolar (Fp1, Fp2), lateral frontal (F7, F8), midfrontal (F3, F4), anterior temporal (T3, T4), central (C3, C4), posterior temporal (T5, T6), parietal (P3, P4), and occipital (O1, O2) regions. In a first step, the latent trait model, the LST model, and the LST change model were analyzed to confirm the hypothesized composition of the asymmetry measure as a compound of a transsituationally consistent and temporally stable trait and occasion-specific components. In a second step, LST parameters were computed to quantify the portion of variance of the manifest variables that was determined by their latent components. In a third step, the procedure for selecting persons with stable asymmetry and the effects of aggregation across various measurement occasions were examined.

1. The SEM analyses were based on the covariance matrices of the parallel asymmetry measures. We fitted the latent trait model, the LST model, and the LST change model to each covariance matrix by minimiz-

ing the generalized least squares (GLS) discrepancy function (Hu & Bentler, 1995). Each model was analyzed in different versions, beginning with a maximally restricted model version (equal variances of the measurement errors [e_{ik}], equal variances of the state residuals [SR_k], equal variances of the trait [T_k], equal effects [β_{ik}] of states [S_k], equal effects [γ_{ik} or γ_k] of trait [T]). If the model could not be accepted, a stepwise liberalization of these restrictions was introduced (Schmitt & Steyer, 1993). Note that these liberalizations are options of the prespecified general models. No a posteriori model liberalizations were introduced to prevent nonreplicable and erroneous model solutions (MacCallum, 1995).

General model fit was evaluated with chi-square statistics and goodness-of-fit indices (comparative fit index [CFI], Bentler, 1990; root-mean-square error of approximation [RMSEA] and the probability of the observed RMSEA under the null hypothesis $RMSEA \leq .05$ [p_{RMSEA}], Browne & Cudeck, 1993). Concurrent model fit was evaluated with chi-square difference tests. The significance ($\alpha = .05$) of single model parameters was evaluated with the critical ratio (C.R.) of a parameter and its standard error (C.R. = Var/SE).

2. On the basis of the accepted models, LST parameters were computed (for formulae, see Steyer et al., 1992; and Steyer & Schmitt, 1990). A *coefficient of reliability* specifies the portion of variance of the manifest variable that is due to all error-free latent components. A *coefficient of trait specificity* represents the percentage of variance of the manifest variable that is determined by transsituationally consistent and temporally stable individual differences.² In contrast, a *coefficient of occasion specificity* reflects the percentage of variance of the manifest variable that is determined by effects of the situation and the person-situation interaction. Finally, a *coefficient of trait stability* was introduced as the retest correlation of the latent traits. The LST parameters were reported for the parallel asymmetry measures and for the total asymmetry measures (the mean of both parallel measures). In principle, this aggregation increases reliability, trait specificity, and occasion specificity due to the decrease of error variance (see Steyer & Schmitt, 1990). The analysis of the trait change model was accomplished by a retest correlation analysis of aggregated asymmetry measures.

3. The examination of the procedure to select persons with stable asymmetry was based on a similar procedure that was used in previous research (Tomarken & Davidson, 1994; Wheeler et al., 1993) and was restricted to the midfrontal and parietal asymmetry measures for the current analysis. The total asymmetry measures were z standardized separately for each occasion. For each person, difference scores of the standardized measures were computed between each two consecutive occasions of measurement. The absolute value of this difference score defined a stability index between the consecutive occasions. On the basis of the stability index of the first two occasions, the 15 participants with the lowest values were assigned to a subgroup of initially stable persons, the 15 participants with the highest values were assigned to a subgroup of unstable persons, and the remaining 29 participants were assigned to a middle group. A one-way analysis of variance with group (stable, middle, unstable) was performed for the stability index of the first two, middle two, and last two occasions

¹ This scalar transformation of variables equals a scalar transformation of their covariance matrix by 10 and cannot lead to alterations of the covariance structure.

² Previously, this coefficient had been termed *consistency* (e.g., Steyer et al., 1999). However, to specify the latent trait or LST model, the assumption has to be made that all manifest variables that were collected in different measurement situations that emerged on temporally distinct occasions k measure the same latent trait (see Figures 1 and 2). Thus, this latent trait is transsituationally consistent and temporally stable across all occasions. Therefore, the coefficient that reflects the portion of variance of the manifest variable that is determined by the latent trait reflects both consistency and stability, and we prefer to term it *trait specificity*.

of measurement. The resulting effect sizes were quantified with Hays's ω^2 (Hays, 1973). Note that in other research, participants were also selected as stable and extreme if their asymmetry measure was in the top or bottom 25th percentile on two occasions of measurement (Kang et al., 1991; Tomarken & Davidson, 1994).

The effects of aggregation across various occasions of measurement were examined on a theoretical basis. Using the model parameter estimates derived above, we recomputed the LST parameters for the case that the total asymmetry measures are aggregated across two, three, or four occasions of measurement. In principle, this aggregation increases reliability as a consequence of the assumption of uncorrelated measurement errors. Furthermore, it decreases occasion specificity as a consequence of uncorrelated state residuals and, hence, increases trait specificity (see Steyer & Schmitt, 1990, for formal proofs and computation formulae).

Further analyses examined the novelty effect of the first testing situation, the dependence of the results on normality assumptions and sample size, and the dependence of the results on the particular derivation scheme. The first occasion of measurement differs from all following occasions with respect to the novelty of the situation. At the initial occasion, the participants experienced an unknown experimental protocol that included sitting in an electrically shielded EEG cabin with electrodes attached to their head while awaiting further tasks. On later occasions, however, this testing procedure was familiar. The novelty of the first occasion may have affected the trait and occasion specificity compared with the following occasions. To evaluate this effect in the present sample, we respecified the restrictive LST model such that the latent state residual (SR_1) and the effect γ_1 of the latent trait were now unrestricted within the first measurement occasion. The modified model was fitted to the data and compared with the restrictive LST model.

The parameter estimates and the inferential statistics used to evaluate model or parameter fit in SEM analyses rely on normal theory assumptions and a sufficient sample size, but the normal theory assumptions are often violated in practice (West, Finch, & Curran, 1995), and no general recommendations concerning sample size can be made (Arbuckle, 1997). To account for this problem, we further analyzed all models with nonpara-

metric bootstrap simulation studies (Efron & Tibshirani, 1993; see Bollen & Stine, 1992; and Yung & Bentler, 1996). Finally, recent studies suggest that the results of EEG asymmetry studies may depend on the particular reference schemes (Hagemann et al., 1998; Reid et al., 1998). We examined this issue by repeating the main analyses with the asymmetry measures that were based on an A1 + A2 reference. All SEM analyses were performed with Amos (Version 3.6; Arbuckle, 1997) under SPSS for Windows (Version 7.5.2G; SPSS, 1997).

Results

Raw Data Description

To evaluate the transsituational consistency and temporal stability of frontal resting asymmetry, we plotted the total asymmetry measure of the midfrontal region separately for each participant and each occasion of measurement in Figure 4. A visual inspection of this plot suggests that the intraindividual variability of persons across the occasions is of a similar magnitude as the interindividual variability of persons within each occasion. Thus, the asymmetry measure of the present sample might reflect not only a consistent and stable trait but also substantial nontrait factors.

LST Models of Asymmetry

Latent trait model. The restrictive latent trait model (equal variances of the measurement errors [e_{ik}], equal effects [γ_{ik}] of trait [T]) could not be accepted for any of the asymmetry measures for any of the scalp regions, all χ^2 s(34, $N = 59$) ≥ 88.33 , all $ps = .000$, all CFIs $\leq .37$, all RMSEAs $\geq .17$, all $p_{RMSEAS} = .000$. A liberal version of this model (equal variances of the measurement errors [e_{ik}] within each occasion, but no restrictions for these variances between occasions, no restrictions for the effects [γ_{ik}] of

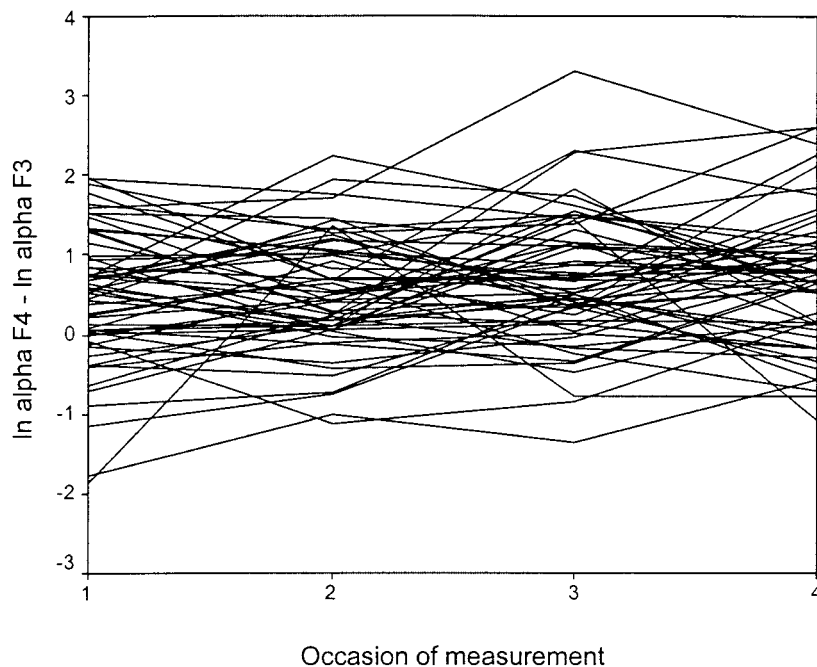


Figure 4. Total midfrontal asymmetry measures (ln alpha power F4 – ln alpha power F3; $\mu V^2/Hz$) of 59 participants on four occasions of measurement (current source density derivations).

trait $[T]$; further liberalizations resulted in not admissible model solutions) did not fit the data adequately either, all χ^2 s(24, $N = 59$) ≥ 78.64 , all p s = .000, all CFIs $\leq .31$, all RMSEAs $\geq .20$, all p_{RMSEA} s = .000. Hence, the assumption that the asymmetry measure only consists of latent trait and latent measurement errors had to be rejected.

LST model. The restrictive LST model (equal variances of the measurement errors $[e_{ik}]$, equal variances of the state residuals $[SR_k]$, equal effects $[\beta_{ik}]$ of states $[S_k]$, equal effects $[\gamma_k]$ of trait $[T]$) showed an acceptable fit to the data for all eight scalp regions, all χ^2 s(33, $N = 59$) ≤ 44.71 , all p s $\geq .084$, all CFIs $\geq .85$, all RMSEAs $\leq .08$, all p_{RMSEA} s $\geq .215$. Table 2 details the respective model test statistics for all regions and additionally presents estimated variances and their standard errors for the latent measurement errors, state residuals, and traits. For all regions, the latent error variances, the latent state residual variances, and the latent trait variances contributed significantly to the variance of the manifest asymmetry measure (all C.R.s ≥ 2.86), with one single exception: The latent trait variance of the frontopolar asymmetry measure failed to show a significant divergence from zero (C.R. = 1.87). With this exception, the assumption that the asymmetry measure is composed of a latent trait that is superimposed on significant state residuals and measurement errors was acceptable for all scalp regions.

LST change model. The restrictive LST change model (equal variances of the measurement errors $[e_{ik}]$, equal variances of the state residuals $[SR_k]$, equal variances of the traits $[T_z]$, equal effects $[\beta_{ik}]$ of states $[S_k]$, equal effects $[\gamma_k]$ of traits $[T_z]$) also showed an acceptable fit to the data for all eight scalp regions, all χ^2 s(32, $N = 59$) ≤ 44.70 , all p s $\geq .067$, all CFIs $\geq .86$, all RMSEAs $\leq .08$, all p_{RMSEA} s $\geq .181$. Table 3 details the respective model test statistics for all regions and also presents the estimated variances of the latent variables, the covariances of the latent traits, and their standard errors. For all regions, the variances of the latent variables contributed significantly to the variance of the manifest asymmetry measures (all C.R.s ≥ 2.43). For all but the frontopolar region, the latent trait covariances were positive and showed a significant divergence from zero (C.R. ≥ 2.83 for all regions except the frontopolar asymmetry, with C.R. = 0.73). Because the LST model of the frontopolar asymmetry measure did not reveal a significant latent trait component and the respective LST change model did not imply a significant latent trait covariance, it may be concluded that there is no consistent and stable trait component in this measure. Thus, frontopolar asymmetry was excluded from all further analyses.

In spite of the adequate model parameters for the remaining variables, the latent trait covariances of the posterior temporal and parietal regions exceeded their respective latent trait variances, implying latent trait retest correlations of 1.06 for the former and 1.07 for the latter region and, hence, not admissible model solutions.³ Such Haywood cases usually indicate a misspecification of the model, with an overparametrization of empirical data (Jöreskog & Sörbom, 1989). One may technically resolve this problem by restricting the latent trait covariance to be equal to the latent trait variances, which yields a model with a latent trait retest correlation fixed to 1.00, thus assuming perfect trait stability.⁴ This new model, however, is not only more parsimonious than the former one (the equality restriction of both trait variances and their covariance now

requires only the estimation of one single parameter instead of two) but is also equivalent to the restrictive LST model that was analyzed—and accepted—above. Hence, the representation of the same (perfectly stable) latent trait with two correlated trait factors may indeed be an overparametrization. For all following analyses of latent trait stability, the posterior temporal and parietal asymmetry measures were excluded and, on the basis of the acceptance of the LST model, perfect trait stability was assumed for these two regions.

For the remaining five regions, the difference between the latent trait covariance and the latent trait variance was small compared with their standard errors. This finding suggests that the trait covariance and trait variance have essentially the same size for these regions, which implies a correlation of approximately 1.00 and, hence, perfect temporal stability of the trait. In addition, a pairwise comparison of the LST model with the LST change model for the remaining five regions revealed no significant difference between these models, all $\Delta\chi^2$ s(1, $N = 59$) ≤ 3.69 , all p s $\geq .05$, although all LST change models showed a somewhat smaller chi-square value and thus better fit to the data.⁵ The sum of these findings suggests not only that all but the frontopolar asymmetry measures reflect significant measurement error and state residual and trait components but that the latent trait also has a high temporal stability.

LST Parameters of Asymmetry

Reliability, occasion specificity, and trait specificity. The parameter estimates for the LST model (see Table 2) allowed the computation of coefficients of reliability, occasion specificity, and trait specificity, which are presented in Table 4. For the parallel asymmetry measures, the reliability was in the range of 0.95 to 0.99 for all scalp regions ($Mdn = 0.95$). With the exception of

³ Although these model solutions are not admissible, they are of considerable diagnostic value for the evaluation of latent trait stability in this particular case. The GLS method yields unbiased estimates of population parameters because of the asymptotic multinormal distribution of the GLS estimates; see Bollen, 1989, pp. 104–123), which implies that a particular population parameter is equally underestimated and overestimated in different estimation trials (samples). If the population parameter has an extreme value (e.g., a correlation that is close to 1.00), then the GLS method can yield estimates that fall outside the admissible range (e.g., a correlation above 1.00), as in the present case. Although the estimated correlations are not admissible, they may be meaningful and suggest very high population correlations that were just overestimated with the current sample.

⁴ The equality restriction of both latent trait variances $\text{Var}(T_1)$ and $\text{Var}(T_2)$ has been part of the analyzed model. The additional restriction of the latent trait covariance $\text{Cov}(T_1, T_2)$ to be equal to these variances, $\text{Cov}(T_1, T_2) = \text{Var}(T_1) = \text{Var}(T_2)$, implies a latent trait correlation of 1.00, because $\text{Cor}(T_1, T_2) = \text{Cov}(T_1, T_2)/\text{St}(T_1)\text{St}(T_2) = \text{Var}(T_1)/\text{St}(T_1)\text{St}(T_1)$. Note that this restriction reduces the total number of estimated parameters from 4 to 3 and thus increases the degrees of freedom from 32 to 33.

⁵ The same analyses for the excluded posterior temporal and parietal regions also showed a substantial similarity of latent trait variance and covariance magnitude and no significant difference between the competing models, both $\Delta\chi^2$ s, (1, $N = 59$) ≤ 1.74 , both p s $\geq .10$. These findings give further support to the assumption of perfect latent trait stability for these regions.

Table 2
Chi-Square Statistics, Goodness-of-Fit Indices, and Estimated Model Parameters of a Latent State-Trait Model of Resting Asymmetry for Eight Scalp Regions

Region	$\chi^2(33, N = 59)$	p	Goodness of fit			Estimated model parameters			
			CFI	RMSEA	p_{RMSEA}	Var(Y)	Var(e)	Var(SR)	Var(T)
Frontopolar	34.35	.403	.99	.03	.619	0.467	0.025	0.370	0.072
SE							0.003	0.046	0.038
Lateral frontal	44.71	.084	.87	.08	.215	0.485	0.021	0.215	0.249
SE							0.002	0.029	0.063
Midfrontal	40.87	.163	.91	.06	.343	0.511	0.023	0.195	0.292
SE							0.003	0.028	0.069
Anterior temporal	23.70	.883	1.00	.00	.954	1.618	0.049	0.546	1.023
SE							0.005	0.068	0.235
Central	44.54	.087	.85	.08	.220	0.876	0.039	0.314	0.523
SE							0.004	0.042	0.142
Posterior temporal	32.53	.490	1.00	.00	.697	2.078	0.036	1.202	0.840
SE							0.004	0.151	0.294
Parietal	37.14	.284	.95	.05	.496	1.249	0.047	0.323	0.879
SE							0.005	0.045	0.234
Occipital	20.64	.954	1.00	.00	.985	3.232	0.042	1.047	2.143
SE							0.004	0.123	0.472

Note. All reported statistics are based on parametric generalized least squares estimates. The same restrictive latent state-trait model was analyzed for the asymmetry measure of each region (equal variances of the measurement error e_{ik} , equal variances of the state residuals SR_k , equal effects β_{ik} of states S_k , equal effects γ_k of trait T). All asymmetry measures are based on current source density derivations. $N = 59$. CFI = comparative fit index; RMSEA = root-mean-square error of approximation; p_{RMSEA} = probability of the observed RMSEA under the null hypothesis $RMSEA \leq .05$.

the frontopolar asymmetry, the occasion specificity was in the range of 0.26 to 0.58 ($Mdn = 0.36$) across all regions, and the trait specificity was in the range of 0.40 to 0.70 ($Mdn = 0.60$). The aggregation across both parallel asymmetry measures yielded total asymmetry measures that showed only marginally increased LST parameters (for all scalp regions, the median of reliability was 0.98, and, except for the frontopolar region, the medians for occasion specificity and trait specificity were 0.37 and 0.61, respectively).

Trait stability. The parameter estimates for the LST change model (see Table 3) allowed the computation of stability coefficients for the latent trait for all but the posterior temporal and parietal regions. For these latter two regions, a latent trait retest correlation of 1.00 was assumed (see above). These stability estimates were supplemented by retest correlations of the aggregated asymmetry measures (averaged across the first or last two occasions) and by retest correlations of the total asymmetry measures between all four occasions. The results are presented in Table 5. With the exception of the frontopolar asymmetry, the estimated retest correlations of the latent trait were in the range of 0.80 to 1.00 ($Mdn = 0.96$) across all regions, the respective retest correlations of the aggregated asymmetry measure were in the range of 0.63 to 0.88 ($Mdn = 0.72$), and the retest correlations of the total measures for the four occasions were in the range of 0.22 to 0.82 ($Mdn = 0.60$ or 0.61).⁶ With the exception of the frontopolar region, the sum of these findings suggests that about 40% of the variance of all asymmetry measures was determined by occasion-specific fluctuations and about 60% of the manifest variance was due to a latent trait with nearly perfect temporal stability. Measurement errors, however, contributed only slightly to the manifest variance.

Selection of Stable Persons

To evaluate the temporal course of frontal resting asymmetry for participants who were selected to show either a stable or an unstable frontal asymmetry on the initial two occasions of measurement, we plotted the asymmetry measures of these two groups separately for all four occasions in Figure 5. A visual inspection of this plot suggests that these two groups showed no obvious difference in their between-occasions variation of frontal asymmetry for all succeeding occasions of measurement. This observation was further confirmed by a one-way analysis of variance of the stability indices, which was performed separately for each measurement interval with group (stable, middle, unstable) as the independent variable. The resulting test statistics and the group means and standard errors are presented in Table 6. A significant and large main effect of group for the first measurement interval indicated that the selection procedure was efficient insofar as the stable group showed the smallest and the unstable group showed the greatest mean stability index, with the middle group falling in between, $F(2, 56) = 47.69, p = .000, \omega^2 = .61$. However, there were no group differences for the stability index of the second and the third measurement intervals (with $p = .184$ and $\omega^2 = .02$ for

⁶ To explore sex differences in temporal stability of resting trait asymmetry, we also analyzed retest correlations of the aggregated asymmetry measure (averaged across the first or last two occasions) separately for each sex. Across the whole scalp, except the frontopolar region, the retest correlations were in the range of .55 to .80 ($Mdn = .67$) for women and in the range of .61 to .91 ($Mdn = .71$) for men. In total, these findings suggest that there is no substantial sex effect on the stability of resting trait asymmetry.

Table 3
Chi-Square Statistics, Goodness-of-Fit Indices, and Estimated Model Parameters of a Latent State-Trait Change Model of Resting Asymmetry for Eight Scalp Regions

Region	$\chi^2(32, N = 59)$	p	Goodness of fit			Estimated model parameters				
			CFI	RMSEA	p_{RMSEA}	Var(Y)	Var(e)	Var(SR)	Var(T)	Cov(T_z)
Frontopolar	31.92	.471	1.00	.00	.677	0.484	0.025	0.332	0.127	0.033
SE							0.003	0.052	0.052	0.045
Lateral frontal	44.70	.067	.86	.08	.181	0.485	0.021	0.212	0.252	0.248
SE							0.002	0.037	0.067	0.064
Midfrontal	37.18	.243	.94	.05	.444	0.527	0.023	0.170	0.334	0.266
SE							0.003	0.031	0.072	0.070
Anterior temporal	23.37	.866	1.00	.00	.945	1.624	0.049	0.521	1.054	1.010
SE							0.005	0.081	0.241	0.236
Central	43.02	.092	.86	.08	.228	0.882	0.038	0.291	0.553	0.484
SE							0.004	0.046	0.144	0.154
Posterior temporal	32.45	.445	1.00	.02	.654	2.087	0.036	1.234	0.817	0.864
SE							0.004	0.187	0.304	0.305
Parietal	35.40	.311	.96	.04	.522	1.303	0.047	0.380	0.875	0.934
SE							0.005	0.062	0.234	0.238
Occipital	19.56	.958	1.00	.00	.986	3.252	0.042	0.978	2.232	2.077
SE							0.004	0.140	0.480	0.476

Note. All reported statistics are based on parametric generalized least squares estimates. The same restrictive latent state-trait model was analyzed for the asymmetry measure of each region (equal variances of the measurement error e_{ik} , equal variances of the state residuals SR_k , equal variances of the traits T_z , equal effects β_{ik} of states S_k , equal effects γ_k of traits T_z). The covariance matrix of the latent trait variables was not positive definite for the posterior temporal and the parietal regions, which was due to a greater covariance than variance in both cases. All asymmetry measures are based on current source density derivations. $N = 59$. CFI = comparative fit index; RMSEA = root-mean-square error of approximation; p_{RMSEA} = probability of the observed RMSEA under the null hypothesis $RMSEA \leq .05$.

the second interval and $p = .676$ and $\omega^2 = .00$ for the third interval).

The same analysis was repeated for participants who were selected to show either a stable or an unstable parietal asymmetry, and this yielded a comparable pattern of results. There was a significant and large between-groups effect for the first, $F(2, 56) = 75.18, p = .000, \omega^2 = .72$, and a significant but smaller effect for the second measurement interval, $F(2, 56) = 3.80, p = .028, \omega^2 = .09$. Again, the stable group showed

the smallest and the unstable group showed the greatest mean stability index. However, no group differences could be observed for the last interval ($p = .000, \omega^2 = .00$). Supplementing these findings, we conducted a retest correlation analysis of stability indices for the whole sample. There was a correlation of .03 for the frontal stability index of the first and last measurement intervals, and the corresponding retest correlation for the parietal stability index was .13. The sum of these findings suggests that this type of selection procedure did not result in

Table 4
Reliability, Occasion Specificity, and Trait Specificity of Parallel and Total Asymmetry Measures for Eight Scalp Regions

Region	LST parameters for parallel measure			LST parameters for total measure		
	Rel(Y)	OccSpe(Y)	TraSpe(Y)	Rel(Y)	OccSpe(Y)	TraSpe(Y)
Frontopolar	.95	.79	.15 ^a	.97	.81	.15 ^a
Lateral frontal	.96	.44	.51	.98	.45	.52
Midfrontal	.95	.38	.57	.97	.39	.58
Anterior temporal	.97	.34	.63	.98	.35	.64
Central	.96	.36	.60	.98	.37	.61
Posterior temporal	.98	.58	.40	.99	.59	.40
Parietal	.96	.26	.70	.98	.27	.71
Occipital	.99	.32	.66	.99	.32	.67

Note. The coefficients of reliability, Rel(Y), occasion specificity, OccSpe(Y), and trait specificity, TraSpe(Y), of the asymmetry measures are based on the estimated model parameters of the restrictive latent state-trait model (equal variances of the measurement error e_{ik} , equal variances of the state residuals SR_k , equal effects β_{ik} of states S_k , equal effects γ_k of trait T). All asymmetry measures are based on current source density derivations. $N = 59$.

^a This parameter was derived from a latent trait variance that failed to be significantly nonzero (see Table 2).

Table 5
Retest Correlations of Latent Asymmetry Traits and Manifest Total or Aggregated Asymmetry Measures for Eight Scalp Regions

Region	Estimate of latent trait retest correlation Cor(T_1, T_2)	Retest correlation of aggregated measures	Retest correlations of total measures between four single occasions	
			lower bound	upper bound
Frontopolar	.26 ^a	.19	-.04	.39
Lateral frontal	.99	.65	.37	.63
Midfrontal	.80	.63	.41	.57
Anterior temporal	.96	.75	.50	.69
Central	.88	.70	.55	.70
Posterior temporal	1.00 ^b	.72	.22	.66
Parietal	1.00 ^b	.88	.70	.82
Occipital	.93	.76	.61	.75

Note. The estimates of the latent trait retest correlation are based on the estimated model parameters of the restrictive latent state-trait change model (equal variances of the measurement error e_{ik} , equal variances of the state residuals SR_k , equal variances of the traits T_z , equal effects β_{ik} of states S_k , equal effects γ_k of traits T_z). The retest correlation of aggregated measures is based on total asymmetry measures that were averaged across the first or last two occasions of measurement. All asymmetry measures are based on current source density derivations. $N = 59$. $Cor(T_1, T_2)$ = correlation between the latent trait variables T_1 and T_2 .

^a This correlation was derived from a covariance that failed to be significantly nonzero (see Table 3). ^b This correlation was fixed to 1.00, and the resulting model was accepted.

person groups that may be distinguished by a consistent and stable attribute of asymmetry stability.

Aggregation Across Occasions

The coefficients of occasion specificity and trait specificity were recomputed for the total asymmetry measures for the case that these variables are aggregated across two, three, or four occasions of measurement. The resulting coefficients are presented in Table 7. Excluding the frontopolar asymmetry, after an aggregation

across two occasions, the expected occasion specificity was in the range of 0.15 to 0.42 ($Mdn = 0.23$) across all regions of the scalp, after an aggregation across three occasions it was in the range of 0.11 to 0.32 ($Mdn = 0.16$), and after an aggregation across four occasions it was in the range of 0.08 to 0.26 ($Mdn = 0.13$). After an aggregation across two, three, and four occasions, however, the trait specificity was in the range of 0.58 to 0.83 ($Mdn = 0.76$), 0.67 to 0.88 ($Mdn = 0.83$), and 0.73 to 0.91 ($Mdn = 0.86$), respectively. Note that the decrease of occasion specificity but increase of trait

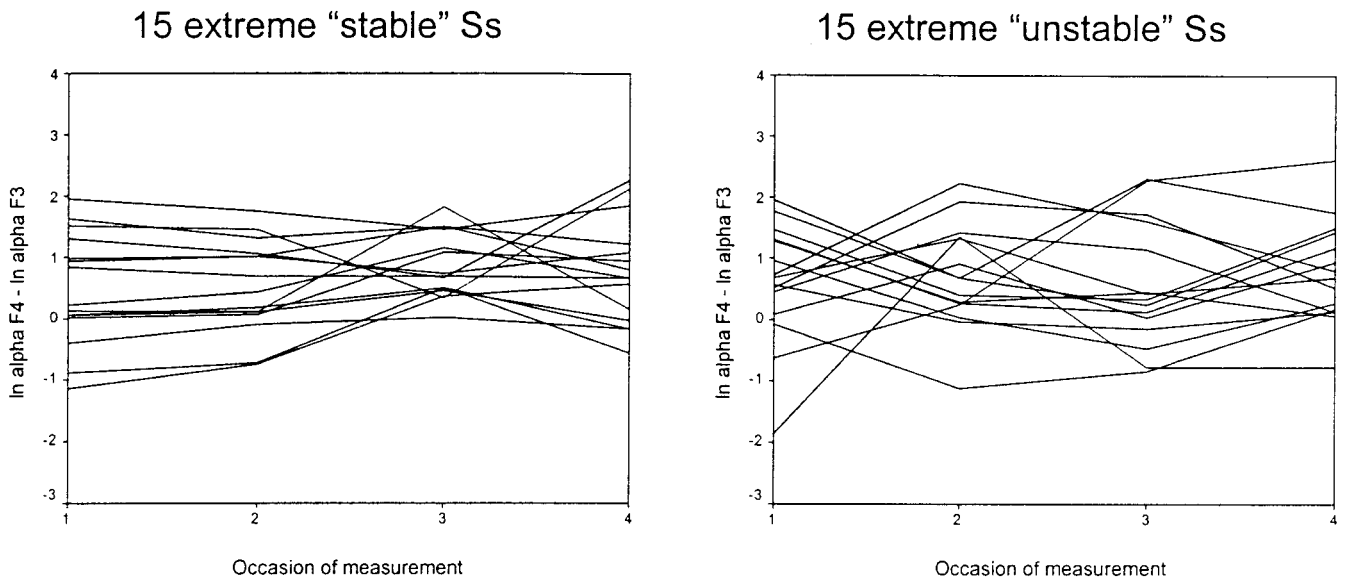


Figure 5. Total midfrontal asymmetry measures (ln alpha power F4 – ln alpha power F3; $\mu V^2/Hz$) on four occasions of measurement of each of 15 participants classified as stable or unstable persons. Ss = subjects.

Table 6
Mean Stability Index, Standard Error, and Test Statistics
for Three Midfrontal Stability Groups

Stability group and statistic	Measurement interval		
	Occasion 1–2	Occasion 2–3	Occasion 3–4
Stable (<i>n</i> = 15)	0.135	0.788	0.816
SE	0.019	0.162	0.181
Middle (<i>n</i> = 29)	0.557	0.638	0.713
SE	0.033	0.099	0.116
Unstable (<i>n</i> = 15)	1.511	1.019	0.879
SE	0.195	0.214	0.123
<i>F</i> (2, 56), <i>p</i>	47.69, .000	1.74, .184	0.39, .676
ω^2	.61	.02	.00

Note. This table presents means and standard errors of a stability index (absolute value of the difference of *z* standardized asymmetry measures of subsequent occasions) separately for three different measurement intervals and for three participant groups who were selected to show minimal (stable group), mediocre (middle group), or maximal (unstable group) stability indices for the first measurement interval. These descriptions are supplemented with the test statistics of a one-way analysis of variance that was performed separately for each measurement interval on the stability indices of the three groups, and the resulting effect sizes, ω^2 . The stability indices were computed from midfrontal asymmetry measures that are based on current source density derivations. *N* = 59.

specificity as a consequence of aggregation is not an empirical finding here but a necessary consequence of the formulae provided by Steyer and Schmitt (1990) for this computation.

Novelty Effect

To evaluate the novelty effect in the present sample, we respecified the restrictive LST model such that it would allow for a different effect of the state residual and the trait in the first as compared with all following occasions (equal variances of the

measurement errors [e_{ik}]; equal variances of the state residuals [SR_k], except SR_1 , which was unrestricted; equal effects [β_{ik}] of states [S_k]; equal effects [γ_k] of trait [T], except γ_1 , which was unrestricted). This model showed an acceptable fit to the data for all eight scalp regions, all $\chi^2(31, N = 59) \leq 43.35$, all *ps* $\geq .069$, all CFIs $\geq .84$, all RMSEAs $\leq .08$, all $p_{RMSEA} \geq .183$. However, the effect of the frontopolar latent trait on the latent state of the first measurement occasion was not significant ($\gamma_1 = .116$, *SE* = 0.431), and the frontopolar region was excluded from further analyses. For the remaining regions, only the model for the parietal scalp sites showed a significantly better fit than the corresponding restrictive LST model, $\Delta\chi^2(2, N = 59) = 9.11, p < .025$; for other regions, $\Delta\chi^2(2, N = 59) \leq 5.30, p > .05$. At this particular region, the occasion specificity was .27 for the first occasion of measurement and .22 for the remaining three occasions, and the respective values for the trait specificity were .71 for the first and .75 for the remaining occasions. In total, these findings suggest that there was no major novelty effect at the first measurement occasion.

Bootstrap Results of LST Models

The same restrictive and liberal latent trait models, LST models, and LST change models that were analyzed with the parametric procedure reported above were subjected to a simulation study. Overall model fit was examined with the bootstrap procedure for testing the hypothesis that the model is correct (Bollen & Stine, 1992). The single model parameters (variances or covariances and their standard errors) were analyzed with the classical bootstrap procedure (Efron, 1982). To facilitate a direct comparison of models and parameters, we drew the identical set of bootstrap samples for each model (Arbuckle, 1997). All analyses were run with 2,000 bootstrap replications (Efron, 1987). All 2,000 bootstrap samples could be used for all models under study.

Neither the restrictive nor the liberal latent trait model could be accepted for any of the asymmetry measures (all *ps* $\leq .012$ for the

Table 7
Expected Occasion Specificity and Trait Specificity of Total Asymmetry Measures After an
Aggregation of the Data Across Two, Three, or Four Measurement Occasions

Region	LST parameters after aggregation across					
	Two occasions of measurement		Three occasions of measurement		Four occasions of measurement	
	OccSpe(Y)	TraSpe(Y)	OccSpe(Y)	TraSpe(Y)	OccSpe(Y)	TraSpe(Y)
Frontopolar ^a	.71	.27	.64	.35	.56	.42
Lateral frontal	.30	.69	.22	.77	.18	.81
Midfrontal	.25	.74	.18	.81	.14	.84
Anterior temporal	.21	.78	.15	.84	.12	.88
Central	.23	.76	.16	.83	.13	.86
Posterior temporal	.42	.58	.32	.67	.26	.73
Parietal	.15	.83	.11	.88	.08	.91
Occipital	.19	.80	.13	.86	.11	.89

Note. The coefficients of occasion specificity, OccSpe(Y), and trait specificity, TraSpe(Y), of the aggregated asymmetry measures are based on the estimated model parameters of the restrictive latent state–trait model for total asymmetry measures (see Table 4) and were computed following Steyer and Schmitt (1990). All asymmetry measures are based on current source density derivations. *N* = 59.

^aThe parameters for this region were derived from a latent trait variance that failed to be significantly nonzero (see Table 2).

restrictive model, and all $ps \leq .003$ for the liberal model). The restrictive LST model showed an acceptable fit to the data for all eight scalp regions (all mean $\Delta GLSs \leq 44.18$, all $ps \geq .228$), and so did the restrictive LST change model (all mean $\Delta GLSs \leq 41.86$, all $ps \geq .227$). For both models, the latent trait variances of the frontopolar region failed to show a significant divergence from zero (C.R. ≤ 1.94). For the remaining regions, the mean variances of the latent error variables, state residuals, and latent traits (and the latent trait covariances) were considerably larger than their empirical standard errors (all C.R.s ≥ 2.09 for the LST model, and all C.R.s ≥ 2.07 for the LST change model). For the LST change model of these seven regions, there was no significant difference in size between the latent trait variances and their respective covariance if related to their standard errors.

Pairwise comparisons of the LST model and the LST change model revealed that the latter models showed a smaller mean GLS discrepancy than did the former model for each of the eight regions (the eight differences of mean ΔGLS were in a range between 1.16 and 2.32 for all eight scalp regions). Although this difference was small in absolute size, it was still large compared with the standard errors of the respective mean GLS discrepancies (the standard errors of all 16 mean $\Delta GLSs$ were below 0.39), indicating a small but significantly better fit of the LST change models to the data.

In sum, these bootstrap findings suggest that the asymmetry measure not only consists of one latent trait plus latent measurement errors but that there are also substantial occasion-specific effects. Although the better fit of the LST change model implies that the latent trait does not have perfect temporal stability, a similar magnitude of latent trait variance and latent trait covariance indicated that the latent trait stability might be close to perfect.

Results for the A1 + A2 Reference

All SEM analyses were repeated with asymmetry measures that were based on an A1 + A2 reference. The restrictive latent trait model could not be accepted for any of the asymmetry measures of all scalp regions (all $ps = .000$), and the liberal version of this model did not fit the data adequately either (all $ps = .000$). However, the restrictive LST model showed an acceptable fit to the data for all but the frontopolar and anterior temporal scalp regions, all $\chi^2s(33, N = 59) \leq 40.73$, all $ps \geq .167$, all CFIs $\geq .90$, all RMSEAs $\leq .06$, all $p_{RMSEA} \geq .348$. A liberalization of this model (relaxation of the restriction that the variances of the measurement errors had to be equal) yielded an acceptable fit for the frontopolar, $\chi^2(30, N = 59) = 38.22$, $p = .144$, CFI = .91, RMSEA = .07, $p_{RMSEA} = .306$, and anterior temporal, $\chi^2(28, N = 59) = 35.76$, $p = .149$, CFI = .91, RMSEA = .07, $p_{RMSEA} = .306$, regions. In addition, the restrictive LST change model also showed an acceptable fit to the data for all eight scalp regions, all $\chi^2s(32, N = 59) \leq 46.00$, all $ps \geq .052$, all CFIs $\geq .84$, all RMSEAs $\leq .09$, all $p_{RMSEA} \geq .151$. For all regions, the variances of the latent state residuals and latent traits contributed significantly to the variance of the manifest asymmetry measures for both models, and the latent trait covariances were positive and showed a significant divergence from zero. Furthermore, a pairwise comparison of the accepted LST model and the respective LST change models revealed no significant differences between the models for all regions (all $ps > .10$) except the frontopolar region, $\Delta\chi^2(2, N = 59) = 7.78$, $p < .025$, where the liberal LST model (RMSEA =

.07) showed a better fit than did the restrictive LST change model (RMSEA = .09).

In the LST change model, the latent trait covariances of the midfrontal, anterior temporal, central, posterior temporal, and parietal regions exceeded their respective latent trait variances, and a perfect trait stability ($Cor[T_1, T_2] = 1$) was assumed for these regions. The respective retest correlations for the anterior temporal, lateral frontal, and occipital regions were .72, .97, and .94, respectively. On the basis of the parameter estimates of the LST models and after an aggregation across both parallel asymmetry measures within one occasion, the median reliability was 0.96 or 0.97 across all eight scalp regions, the median occasion specificity was 0.38 or 0.49, and the median trait specificity was 0.47 or 0.56. In summary, the resting EEG asymmetry measures that were based on an A1 + A2 reference also consisted of a stable and consistent latent trait that was superimposed on occasion-specific effects, which parallels the findings for the CSD data. However, the occasion specificity of the A1 + A2 reference data appeared to be somewhat higher and the trait specificity somewhat lower than for the CSD data, but the trait specificity still exceeded the occasion specificity on average. Most interesting, the frontopolar asymmetry measure of the A1 + A2 reference showed an LST structure similar to that of the measures of all other scalp regions, which marks the only striking difference compared with the CSD results.

Supplementing these LST findings, a one-way analysis of variance of the stability indices of the first measurement interval (Occasion 1–2) with group (stable, middle, unstable) as the independent variable indicated a significant main effect for the midfrontal, $F(2, 56) = 126.34$, $p = .000$, $\omega^2 = .81$, and the parietal, $F(2, 56) = 91.32$, $p = .000$, $\omega^2 = .75$, regions. As expected, the stable group showed the smallest and the unstable group showed the greatest mean stability index, with the middle group falling in between. However, there was no respective between-groups effect for the stability indices of the last measurement interval (Occasion 3–4; midfrontal: $p = .459$ and $\omega^2 = .00$; parietal: $p = .133$ and $\omega^2 = .04$). Paralleling the findings for the CSD data, these results suggest that this type of selection procedure did not result in person groups that may be distinguished by a consistent and stable attribute of asymmetry stability for the A1 + A2 reference.

Discussion

In the majority of studies on tonic activation asymmetry and affective style, the implicit assumption was made that the manifest resting EEG asymmetry is a sufficient proxy for a stable trait and that situation or person-situation interactions would not affect the resting asymmetry in a considerable way. Consequently, asymmetry was measured on only one occasion (e.g., Gotlib, Ranganath, & Rosenfeld, 1998; Hagemann et al., 1999; Harmon-Jones & Allen, 1998; Nitschke, Heller, Palmieri, & Miller, 1999; see Table 1). A latent trait model of resting EEG asymmetry might represent the assumption that underlies this research. In contrast, some researchers were worried by rather low retest correlations of the asymmetry measures and explicitly proposed that the manifest resting asymmetry might reflect the joint contributions of stable individual differences and statelike factors (Davidson, 1992a, 1993; Tomarken, Davidson, Wheeler, & Kinney, 1992; see also Rosenfeld, 2000). Accordingly, these researchers measured asymmetry on two occasions of measurement, and different protocols for the

control of the presumed occasion-specific effects were applied (Sutton & Davidson, 1997; Tomarken & Davidson, 1994; Tomarken, Davidson, Wheeler, & Doss, 1992; Wheeler et al., 1993). An LST model of asymmetry may represent this explicit assumption. However, the low retest correlations of the measures—and the considerable inconsistency of findings on asymmetry and affective dispositions—might alternatively be explained by the assumption of a temporal instability of the trait asymmetry, which in turn may be represented by an LST change model of asymmetry. The present study examines these assumptions.

The LST Structure of Resting Asymmetry Measures

A latent trait model had to be rejected for all areas of the scalp, but an LST model and an LST change model could be accepted for all regions. There was no significant difference between these two models for any area. Omitting the frontopolar region, the median of the estimated retest correlations of the latent traits was .96 across all regions. These findings indicate that an LST model, which assumes perfect trait stability, might adequately represent the asymmetry measures of the current sample.

At the frontopolar region, only state residuals and measurement errors contributed significantly to the variance of the asymmetry measure; in contrast, there was no substantial stable trait component. For the remaining areas of the scalp, about 40% of the variance of the resting EEG asymmetry was due to effects of the situation or the person–situation interaction. Conversely, approximately 60% of the variance of the asymmetry measure was determined by temporally stable and transsituationally consistent individual differences. However, measurement errors contributed in a rather negligible size to the variance of resting EEG asymmetry. The reliability estimates were somewhat higher than reported in previous research (Hagemann et al., 1998; Reid et al., 1998; Sutton & Davidson, 1997; Tomarken, Davidson, Wheeler, & Kinney, 1992), which may be due to the use of a 12-min EEG database in the present study rather than the usual 8 min of resting EEG recordings. The sum of these findings gives strong support for the hypothesis that the manifest asymmetry reflects both a temporally stable latent trait and situational effects or person–situation interactions and thus confirms the respective proposition of Davidson and colleagues (Davidson, 1992a, 1993; Tomarken, Davidson, Wheeler, & Kinney, 1992; Wheeler et al., 1993). Conversely, the implicit assumption that the manifest resting asymmetry is hardly affected by occasion-specific factors had to be rejected. Because the trait-specificity coefficients were larger than the occasion-specificity coefficients, it appears that the resting asymmetry measure is more a trait variable than an occasion-specific state variable.

Procedures for the Control of Occasion-Specific Fluctuations

To account for the presumed occasion specificity of resting EEG asymmetry, some researchers have measured asymmetry on two occasions of measurement and selected only those participants who showed a similar asymmetry measure on both occasions (e.g., Tomarken, Davidson, Wheeler, & Doss, 1992; Wheeler et al., 1993) or aggregated the data across both occasions (e.g., Sutton & Davidson, 1997; Tomarken & Davidson, 1994). However, the

former approach implicitly assumes that the shift of resting EEG asymmetry between two occasions is a stable property of the person, and the latter procedure presumes that an aggregation across only two occasions is sufficient for a substantial decrease of occasion specificity.

To examine the first of these assumptions, we computed stability indices for each person as a difference of midfrontal asymmetry between the first two, the middle two, and the last two occasions of measurement. On the basis of the stability index of the first measurement interval, participants were assigned to subgroups of stable or unstable persons or to a middle group. There was a significant and large group difference of stability scores for the first measurement interval, which reflected the definition of the three groups. However, there was no significant between-groups difference of the stability index for the second or third measurement intervals. A similar finding was obtained after a classification of participants based on parietal stability indices of asymmetry. In addition, the retest correlation of the frontal stability index for the first and last measurement intervals was .03, and the corresponding retest correlation for the parietal region was .13. These findings suggest that this type of selection procedure did not result in person groups that may be distinguished by a consistent and stable property of asymmetry stability.

The effects of aggregation across various occasions of measurements on reliability and occasion specificity are well known (Epstein, 1979, 1980; Schmitt & Steyer, 1990), and therefore aggregation formulas could be used to estimate these effects on a theoretical basis (Steyer & Schmitt, 1990). Omitting the frontopolar asymmetry, the respective calculations suggested that an aggregation of the 12-min EEG across two occasions may be expected to decrease the occasion specificity to a median of 0.23 across all regions and to increase the trait specificity to a median of 0.76. However, the aggregation across three occasions may alter the occasion and trait specificity to values of 0.16 and 0.83, respectively, and the corresponding specificity coefficients after an aggregation across four occasions might be expected to be 0.13 and 0.86. Similar to the effects of aggregation on reliability, the aggregation across measurement occasions may reduce the occasion specificity—and thus increase the trait specificity—but it cannot eliminate it (Steyer & Schmitt, 1990).

Limitations

Before we discuss the consequences of these findings, some potential limitations of the research procedures and results of the current study should be noted. First, the rejection rate of EEG epochs that were contaminated with artifacts was somewhat higher in the present work than the rejection rates of previous studies. For example, Henriques and Davidson (1991) deleted 37% of epochs in a sample of normal control participants, whereas the rejection rates of the present study were in a range between 43% and 49% for different occasions. This higher rejection rate was due to a rather rigid rejection criterion, because recent findings from our lab suggested that even minor blinks and eye movements could result in substantial distortions of frontopolar alpha activity. In particular, Hagemann and Naumann (2001) investigated the effects of ocular artifacts on EEG broadband spectral parameters for different scalp regions. In the alpha band, the correlation between frontopolar asymmetry measures that were based on 10% rejection

(all nonocular artifacts excluded, but all ocular artifacts still included) and 46% rejection (all artifacts excluded) was only .58. In contrast, the same correlation for midfrontal alpha asymmetry was .95. This finding suggests that frontopolar alpha asymmetry is very susceptible to ocular artifacts; therefore, a conservative control procedure was used in the present study. However, this also suggests that, psychometrically, the present findings for sites posterior to the frontopolar region would be highly comparable to those of studies that use a less conservative rejection criterion than in the present work.

Second, the CSD derivations that were used for the main analysis of the present sample have the advantage of being reference free—that is, they do not rely on the assumption that a liberally chosen reference site (e.g., vertex) or a set of those sites (e.g., linked ears, average reference) is electrically inactive (see Hjorth, 1975, 1980; Katznelson, 1981; Lehmann, 1987; Nunez, 1981). However, the CSD approach also has limitations. For example, the CSD transform acts as a spatial high-pass filter and thus attenuates broadly distributed scalp activity, the magnitude of the CSD estimates depends on the spacing of the electrodes, and the CSD estimates are less accurate for sites on the edge line of the electrode system (for a detailed discussion of these issues, see Hagemann, Naumann, & Thayer, 2001). To facilitate a numerical comparison (Katznelson, 1981; Lehmann, 1987) with previous work (see Table 1), we repeated all main analyses with a computational linked ears reference (A1 + A2), and this yielded highly similar conclusions, with one remarkable exception. The frontopolar asymmetry measure for the A1 + A2 reference showed an LST structure similar to that of the measures of all other scalp regions, including a stable latent trait component, which is in contrast to the absence of a significant latent trait component for the frontopolar CSD data. This result might tentatively be explained by the enhanced sensitivity of the CSD method for the measurement of local electrical activity on the scalp (Nunez, 1981), which would suggest a greater weight for the CSD relative to the A1 + A2 finding. Nonetheless, the enhanced spatial selectivity of CSD derivations in conjunction with the proximity of frontopolar sites to the eyes might yield a magnified susceptibility of frontopolar CSD derivations to even minor ocular artifacts. As the present data do not allow a cogent conclusion on this issue and as frontopolar resting EEG asymmetry has become the target of recent research (Papousek & Schuster, 1998; Sutton & Davidson, 1997), this problem might be followed up in future studies.

Third, the initial occasion of measurement differs from all following occasions with respect to the novelty of the situation which may result in different trait and occasion specificities for the first occasion as compared with all further occasions. To account for this problem, we refitted the data to a liberal version of the state-trait model that explicitly allowed a novelty effect. This model showed no significantly better fit or resulted in LST parameter estimates similar to those of the more parsimonious restrictive state-trait model, which suggests that there was no major novelty effect in the present study. Thus, the reported LST parameters may be considered to be representative for the first and the subsequent measurement occasions.

Fourth, the parameter estimates and the inferential statistics used to evaluate the models and parameters of the present study were based on normal theory assumptions and a sufficient sample size. However, the normal theory assumptions are often violated in

practice (West et al., 1995), and no general recommendations concerning sample size can be made (Arbuckle, 1997). To account for this problem, we reanalyzed all models with Monte Carlo studies of the bootstrap type (Bollen & Stine, 1992; Efron & Tibshirani, 1993; Yung & Bentler, 1996). These simulations yielded conclusions identical to those of the normal theory-based analyses and give some confidence that the reported results were not artifacts due to violations of the normal theory assumption or due to an insufficient sample size.

Finally, all participants were assessed in a highly standardized laboratory situation. Hence, the state-residual variance of the asymmetry measures may be reduced compared with the variability of real-life resting situations. Consequently, the trait specificity that was reported for the present study appears to be an upper bound of the trait specificity that may be expected in more natural situations, whereas the estimated occasion specificity appears to be a lower bound. Furthermore, the manipulation of the situation at each measurement occasion (e.g., by emotion induction procedures) might decrease the trait specificity of the asymmetry measures as well. It is important to note that this is not a shortcoming of the LST theory. The coefficients of reliability, situation specificity, and trait specificity are not properties of behavior or the construct (i.e., tonic asymmetry of cortical activation) but are properties of the measured random variable (i.e., resting EEG asymmetry measure; see Steyer & Schmitt, 1990). Of course, the definition of the situation is part of the random experiment that generated the random variables and has to be considered in interpretations of the LST coefficients.

Retrospective Consequences

In total, the main findings of the current study were independent of the particular statistical estimation procedure and do not appear to be statistical artifacts due to an insufficient sample size. In addition, the main findings were not dependent on the particular EEG derivation type or the experimental setup of the EEG recording situation. Thus, the present evidence for occasion-specific effects on the resting EEG asymmetry may be considered with confidence, and it implies some consequences for this field of research.

Assuming that the latent trait but not the state residuals of resting EEG asymmetry is associated with affective traits, the correlations between the manifest asymmetry measure and the affective trait indicators might underestimate the true magnitude of this association (similar to the problem of correlation suppression by unreliability). Although no affective variables were addressed in the current analyses, this problem may be depicted with the different estimates of trait stability. In the present sample (and excluding the frontopolar asymmetry), the retest correlations of the total asymmetry measures between the single occasions were of a median size as low as .60, which replicates the results of Debener et al. (2000), Papousek and Schuster (1998), Sutton and Davidson (1997), and Tomarken, Davidson, Wheeler, and Kinney (1992). However, an aggregation across the first two and last two occasions reduced the occasion specificity, and the respective retest correlations of the manifest asymmetry measures were of a median size of .72. Furthermore, the LST change model allowed the estimation of the latent trait retest correlations with the suppressing effects of occasion specificity being eliminated. The median of the

estimated correlations was .96. Thus, the findings of the present study suggest not only that the temporal stability of resting trait asymmetry might have been underestimated in previous research but also that the association between trait asymmetry and affective dispositions may have been underrated.

Unfortunately, the lack of control of occasion specificity in previous studies was not only likely to reduce the expected inter-trait correlations but also introduced noise to the data, which might explain a substantial portion of the inconsistency of findings. The high occasion specificity of resting EEG asymmetry in the one-occasion paradigm (and the weakness of the selection procedure in the two-occasion paradigm) suggests that the validity of previously used research strategies might have been overestimated. In light of the present study, the bulk of evidence (see Table 1) appears to be less conclusive than was previously appreciated. Future studies on trait asymmetry might benefit from considerations of the problem of occasion-specific fluctuations and use appropriate methods for their control.

Prospective Consequences

In the present sample, about 40% of the variance of the resting EEG asymmetry was due to occasion-specific effects. It appears that resting asymmetry measures from one single assessment are not an adequate proxy for a trait and that further investigations of tonic brain asymmetry and affective dispositions that rely on the single-occasion paradigm might add to the inconsistency rather than reduce it. Alternatively, a multioccasion paradigm may be used to reduce the occasion-specific effects of resting EEG asymmetry, and the present findings emphasize the warning that was put forward by Davidson (1998b): "It is imperative that in future research using these methods at least two sessions (and ideally more than two) of baseline EEG data collection be included" (p. 613). In addition to an assessment of resting EEG asymmetry on several occasions, it is also critical that an appropriate analysis procedure be used.

The present findings suggest that the selection of persons with similar asymmetry measures on two occasions led to a nonrepliable classification of participants. Hence, this selection procedure seems not to be tenable for a reduction of the occasion specificity of resting EEG asymmetry. Two alternative strategies may be efficient. First, the aggregation of data across occasions may reduce the occasion specificity and enhance the trait specificity of the measure. Given that 76% of the variance of the asymmetry measure might be due to a true trait after an aggregation across as few as two occasions, this particular procedure may be considered sufficient for the control of occasion-specific fluctuations if only the trait component is of substantial interest. As an alternative, separate LST models within one simultaneous analysis may represent asymmetry and affectivity, and the correlation of the latent traits or the latent state residuals may be assessed directly (see Eid, Notz, Steyer, & Schwenkmezger, 1994, and Steyer, Schwenkmezger, & Auer, 1990, for applications).

In this context, it is important to note that the classical approach of aggregation across measurement situations aims for a reduction of occasion-specific variance and thus treats the effects of the situation and the situation-person interactions as a part of the measurement error (see Epstein, 1990). In contrast, the LST framework allows for a separation of unsystematic measurement errors

and the systematic effects within a measurement situation (Schmitt & Steyer, 1990; Steyer & Schmitt, 1990). In the case of activation asymmetry, these occasion-specific effects may be meaningful variables rather than random noise. For instance, many findings of the state paradigm of asymmetry research have suggested that the induction of different affective or motivational states results in a systematic statelike shift of asymmetry (for reviews, see Davidson, 1995; Hagemann et al., 1998). Moreover, previous findings have also demonstrated associations between activation asymmetry and mental states such as covert cognitive activity (e.g., Ehrlichman & Wiener, 1980) or physiological states that result from an intake of drugs such as nicotine (e.g., Zinser, Fiore, Davidson, & Baker, 1999). It appears that both the trait and the statelike components of cerebral activation asymmetry are systematically associated with further biological and psychological constructs. Thus, an analysis of multiconstruct associations of resting asymmetry may benefit from the statistical separation of measurement error, occasion-specific effects, and trait-specific effects. Unfortunately, these simultaneous LST models are complex and require rather large sample sizes for reliable estimates.

Finally, it may be noted that the LST approach could also be of interest for an analysis of other physiological variables that are thought to measure traits but may be masked by statelike factors. As was mentioned in the introduction of this article, the arousal theory of extraversion (Eysenck, 1967) stimulated extensive research with alpha activation of the resting EEG serving as the proxy for the physiological trait construct. However, the problem of using the state-dependent spontaneous EEG as a marker for consistent and stable individual differences ultimately led to a decline of this research paradigm (Bartussek, 1984; Stelmack, 1990). Conversely, the LST theory might serve as a framework to readdress this issue in empirical research.

Other psychophysiological variables that intend to capture traits such as heart rate variability (Friedman & Thayer, 1998a, 1998b) or tonic regional cerebral blood flow (rCBF; Fredrikson, Wik, & Fischer, 1999; Holcomb et al., 1993; Schmidt et al., 1996) may also profit from an LST analysis for similar reasons. For example, rCBF is of great interest for individual-differences research (see Davidson, 1998a; Heller, Nitschke, Etienne, & Miller, 1997; Reid et al., 1998), although the rCBF assessment in unstimulated resting states may also show an increased variability/reduced retest stability (see Fredrikson et al., 1999; Holcomb et al., 1993; Schmidt et al., 1996, and the literature cited there). This variability/instability was interpreted as increased error variance/unreliability, and procedures such as neutral visual stimulation during resting rCBF scans, simple discrimination tasks during the rCBF scans, averaging across multiple resting scans, normalization of regional brain activation indices to global activation indices, or the use of tracers with prolonged uptake were recommended to account for this problem (Davidson, 1998a; Fredrikson et al., 1999; Holcomb et al., 1993; Schmidt et al., 1996). However, this error variance might represent physiologically meaningful statelike fluctuations (Holcomb et al., 1993; Schmidt et al., 1996), and the separation of these fluctuations from latent traits might be of great interest. No matter which physiological construct is the target of research and what protocol is used for its assessment, an explication and investigation of each measurement strategy's underlying assumptions may be crucial for the development of cogent paradigms for the study of the biological basis of individual differences.

References

- Allen, J. J., Iacono, W. G., Depue, R. A., & Arbisi, P. (1993). Regional electroencephalographic asymmetries in bipolar seasonal affective disorder before and after exposure to bright light. *Biological Psychiatry*, *33*, 642–646.
- Arbuckle, J. L. (1997). *Amos user's guide* (Version 3.6). Chicago: Small-Waters.
- Barlow, J. S. (1986). Artifact processing (rejection and minimization) in EEG data processing. In F. H. Lopes da Silva, W. Storm van Leeuwen, & A. Rémond (Eds.), *Handbook of electroencephalography and clinical neurophysiology. Clinical applications of computer analysis of EEG and other neurophysiological signals* (Rev. series, Vol. 2, pp. 15–62). Amsterdam: Elsevier.
- Bartussek, D. (1984). Extraversion und EEG: Ein Forschungsparadigma in der Sackgasse? [Extraversion and EEG: A research paradigm in a blind alley?] In M. Amelang & H.-J. Ahrens (Eds.), *Brennpunkte der Persönlichkeitsforschung* (Vol. 1, pp. 157–189). Göttingen, Germany: Hogrefe.
- Basar, E., & Haken, H. (Eds.). (1998). *Brain function and oscillations: Brain oscillations* (Vol. 1). New York: Springer-Verlag.
- Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. *Archives of General Psychiatry*, *4*, 561–571.
- Bell, I. R., Schwartz, G. E., Hardin, E. E., Baldwin, C. M., & Kline, J. P. (1998). Differential resting quantitative electroencephalographic alpha patterns in women with environmental chemical intolerance, depressives, and normals. *Biological Psychiatry*, *43*, 376–388.
- Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin*, *107*, 238–246.
- Berger, H. (1929). Über das Elektrenkephalogramm des Menschen [On the human electroencephalogram]. *Archiv für Psychiatrie*, *87*, 527–570.
- Blom, J. B., & Anneveldt, M. (1982). An electrocap tested. *Electroencephalography and Clinical Neurophysiology*, *54*, 591–594.
- Bollen, K. A. (1989). *Structural equations with latent variables*. New York: Wiley.
- Bollen, K. A., & Stine, R. A. (1992). Bootstrapping goodness-of-fit measures in structural equation modeling. *Sociological Methods and Research*, *21*, 205–229.
- Browne, M. W., & Cudeck, R. (1993). Alternative ways of assessing model fit. In K. A. Bollen & J. S. Long (Eds.), *Testing structural equation models* (pp. 136–162). Newbury Park, CA: Sage.
- Bruder, G. E., Fong, R., Tenke, C. E., Leite, P., Towey, J. P., Stewart, J. E., et al. (1997). Regional brain asymmetries in major depression with or without anxiety disorder: A quantitative electroencephalographic study. *Biological Psychiatry*, *41*, 937–948.
- Buss, A. H., & Perry, M. (1992). The aggression questionnaire. *Journal of Personality and Social Psychology*, *63*, 452–459.
- Calkins, S. D., Fox, N. A., & Marshall, T. R. (1996). Behavioral and physiological antecedents of inhibited and uninhibited behavior. *Child Development*, *67*, 523–540.
- Carver, C. L., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. *Journal of Personality and Social Psychology*, *67*, 319–333.
- Chatrian, G. E., Lettich, E., & Nelson, P. L. (1985). Ten percent electrode system for topographic studies of spontaneous and evoked EEG activities. *American Journal of EEG Technology*, *25*, 83–92.
- Costa, P. T., Jr., & McCrae, R. R. (1992). Trait psychology comes of age. In T.B. Sonderegger (Ed.), *Nebraska Symposium on Motivation: Psychology and aging* (pp. 169–204). Lincoln: University of Nebraska Press.
- Creutzfeldt, O. D. (1995). *Cortex cerebri. Performance, structural and functional organization of the cortex*. Oxford, England: Oxford University Press.
- Curran, T., Tucker, D. M., Kutas, M., & Posner, M. I. (1993). Topography of the N400: Brain electrical activity reflecting semantic expectancy. *Electroencephalography and Clinical Neurophysiology*, *88*, 188–209.
- Davidson, R. J. (1984). Hemispheric asymmetry and emotion. In K. Scherer & P. Ekman (Eds.), *Approaches to emotion* (pp. 39–57). Hillsdale, NJ: Erlbaum.
- Davidson, R. J. (1988). EEG measures of cerebral asymmetry: Conceptual and methodological issues. *International Journal of Neuroscience*, *39*, 71–89.
- Davidson, R. J. (1992a). Anterior cerebral asymmetry and the nature of emotion. *Brain and Cognition*, *20*, 125–151.
- Davidson, R. J. (1992b). Emotion and affective style: Hemispheric substrates. *Psychological Science*, *3*, 39–43.
- Davidson, R. J. (1993). Cerebral asymmetry and emotion: Conceptual and methodological conundrums. *Cognition and Emotion*, *7*, 115–138.
- Davidson, R. J. (1995). Cerebral asymmetry, emotion, and affective style. In R. J. Davidson & K. Hugdahl (Eds.), *Brain asymmetry* (pp. 361–389). Cambridge, MA: MIT Press.
- Davidson, R. J. (1998a). Affective style and affective disorders: Perspectives from affective neuroscience. *Cognition and Emotion*, *12*, 307–330.
- Davidson, R. J. (1998b). Anterior electrophysiological asymmetries, emotion, and depression: Conceptual and methodological conundrums. *Psychophysiology*, *35*, 607–614.
- Davidson, R. J., Ekman, P., Saron, C. D., Senulis, J. A., & Friesen, W. V. (1990). Approach/withdrawal and cerebral asymmetry: Emotional expression and brain physiology, I. *Journal of Personality and Social Psychology*, *58*, 330–341.
- Davidson, R. J., & Fox, N. A. (1989). Frontal brain asymmetry predicts infants' response to maternal separation. *Journal of Abnormal Psychology*, *98*, 127–131.
- Davidson, R. J., & Tomarken, A. J. (1989). Laterality and emotion: An electrophysiological approach. In F. Boller & J. Grafman (Eds.), *Handbook of neuropsychology* (Vol. 3, pp. 419–441). Amsterdam: Elsevier.
- Debener, S., Beauducel, A., Nessler, D., Brocke, B., Heilemann, H., & Kayser, J. (2000). Is resting anterior EEG alpha asymmetry a trait marker for depression? *Neuropsychobiology*, *41*, 31–37.
- Derogatis, L. R., Lipman, R. S., & Covi, L. (1973). The SCL-90: An outpatient psychiatric rating scale. *Psychopharmacology Bulletin*, *9*, 13–28.
- Dummermuth, G., & Molinari, L. (1987). Spectral analysis of EEG background activity. In A. S. Gevins & A. Rémond (Eds.), *Handbook of electroencephalography and clinical neurophysiology. Methods of analysis of brain electrical signals and magnetic signals* (Rev. series, Vol. 1, pp. 85–130). Amsterdam: Elsevier.
- Efron, B. (1982). *The jackknife, the bootstrap and other resampling plans*. Philadelphia: Society for Industrial and Applied Mathematics.
- Efron, B. (1987). Better bootstrap confidence intervals. *Journal of the American Statistical Association*, *82*, 171–185.
- Efron, B., & Tibshirani, R. J. (1993). *An introduction to the bootstrap*. New York: Chapman & Hall.
- Ehrlichman, H., & Wiener, M. S. (1980). EEG asymmetry during covered mental activity. *Psychophysiology*, *17*, 228–235.
- Eid, M. (2000). A multitrait-multimethod model with minimal assumptions. *Psychometrika*, *65*, 241–261.
- Eid, M., & Hoffmann, L. (1998). Measuring variability and change with an item response model for polytomous variables. *Journal of Educational and Behavioral Statistics*, *23*, 193–215.
- Eid, M., Notz, P., Steyer, R., & Schwenkmezger, P. (1994). Validating scales for the assessment of mood level and variability by latent state-trait analysis. *Personality and Individual Differences*, *16*, 63–76.
- Epstein, S. (1979). The stability of behavior: I. On predicting most of the people much of the time. *Journal of Personality and Social Psychology*, *37*, 1097–1126.
- Epstein, S. (1980). The stability of behavior: II. Implications for psychological research. *American Psychologist*, *35*, 790–806.

- Epstein, S. (1983). Aggregation and beyond: Some basic issues on the prediction of behavior. *Journal of Personality*, *51*, 360–392.
- Epstein, S. (1990). Comments on the effects of aggregation across and within occasions on consistency, specificity, and reliability. *Methodika*, *4*, 95–100.
- Eysenck, H. J. (1967). *The biological basis of personality*. Springfield, IL: Charles C Thomas.
- Flor-Henry, P., & Koles, Z. J. (1984). Statistical quantitative EEG studies of depression, mania, schizophrenia and normals. *Biological Psychology*, *19*, 257–279.
- Fox, N. A., Bell, M. A., & Jones, N. A. (1992). Individual differences in response to stress and cerebral asymmetry. *Developmental Neurophysiology*, *8*, 161–184.
- Fox, N. A., & Davidson, R. J. (1988). Patterns of brain electrical activity during facial signs of emotion in 10-month-old infants. *Developmental Psychology*, *24*, 230–236.
- Fredrikson, M., Wik, G., & Fischer, H. (1999). Higher hypothalamic and hippocampal neural activity in Type A than Type B women. *Personality and Individual Differences*, *26*, 265–270.
- Friedman, B. H., & Thayer, J. F. (1998a). Anxiety and autonomic flexibility: A cardiovascular approach. *Biological Psychology*, *47*, 243–263.
- Friedman, B. H., & Thayer, J. F. (1998b). Autonomic balance revisited: Panic anxiety and heart rate variability. *Journal of Psychosomatic Research*, *44*, 133–151.
- Gainotti, G. (1972). Emotional behavior and hemispheric side of lesion. *Cortex*, *8*, 230–236.
- Gale, A. (1973). The psychophysiology of individual differences: Studies of extraversion and the EEG. In P. Kline (Ed.), *New approaches in psychological measurement* (pp. 211–256). New York: Wiley.
- Gale, A. (1983). Electroencephalographic studies of extraversion–introversion: A case study in the psychophysiology of individual differences. *Personality and Individual Differences*, *4*, 371–380.
- Gasser, T., Bächer, P., & Möcks, J. (1982). Transformations towards the normal distribution of broad band spectral parameters of the EEG. *Electroencephalography and Clinical Neurophysiology*, *53*, 119–124.
- Gotlib, I. H., Ranganath, C., & Rosenfeld, J. P. (1998). Frontal EEG alpha asymmetry, depression, and cognitive functioning. *Cognition and Emotion*, *12*, 449–478.
- Hagemann, D., Hewig, J., Seifert, J., Naumann, E., & Bartussek, D. (2002). *No association between EEG trait asymmetry and affective style*. Poster submitted for presentation.
- Hagemann, D., & Naumann, E. (2001). The effects of ocular artifacts on (lateralized) broadband power in the EEG. *Clinical Neurophysiology*, *112*, 215–231.
- Hagemann, D., Naumann, E., Becker, G., Maier, S., & Bartussek, D. (1998). Frontal brain asymmetry and affective style: A conceptual replication. *Psychophysiology*, *35*, 372–388.
- Hagemann, D., Naumann, E., Lürken, A., Becker, G., Maier, S., & Bartussek, D. (1999). EEG asymmetry, dispositional mood and personality. *Personality and Individual Differences*, *27*, 541–568.
- Hagemann, D., Naumann, E., & Thayer, J. F. (2001). The quest for the EEG reference revisited: A glance from brain asymmetry research. *Psychophysiology*, *38*, 847–857.
- Harmon-Jones, E., & Allen, J. J. B. (1997). Behavioral activation sensitivity and resting frontal EEG asymmetry: Covariation of putative indicators related to risk of mood disorders. *Journal of Abnormal Psychology*, *106*, 159–163.
- Harmon-Jones, E., & Allen, J. J. B. (1998). Anger and frontal brain asymmetry: EEG asymmetry consistent with approach motivation despite negative affective valence. *Journal of Personality and Social Psychology*, *74*, 1310–1316.
- Hays, W. L. (1973). *Statistics for the social sciences* (2nd ed.). New York: Holt, Rinehart and Winston.
- Heller, W., & Nitschke, J. B. (1998). The puzzle of regional brain activity in depression and anxiety: The importance of subtypes and comorbidity. *Cognition and Emotion*, *12*, 421–447.
- Heller, W., Nitschke, J. B., Etienne, M. A., & Miller, G. A. (1997). Patterns of regional brain activity differentiate types of anxiety. *Journal of Abnormal Psychology*, *106*, 376–385.
- Henriques, J. B., & Davidson, R. J. (1990). Regional brain electrical asymmetries discriminate between previously depressed and healthy control subjects. *Journal of Abnormal Psychology*, *99*, 22–31.
- Henriques, J. B., & Davidson, R. J. (1991). Left frontal hypoactivation in depression. *Journal of Abnormal Psychology*, *100*, 535–545.
- Hjorth, B. (1975). An on-line transformation of EEG scalp potentials into orthogonal source derivations. *Electroencephalography and Clinical Neurophysiology*, *39*, 526–530.
- Hjorth, B. (1980). Source derivation simplifies topographical EEG interpretation. *American Journal of EEG Technology*, *20*, 121–132.
- Holcomb, H. H., Cascella, N. G., Medoff, D. R., Gastineau, E. A., Loats, H., Thaker, G. K., et al. (1993). PET–FDG test–retest reliability during a visual discrimination task in schizophrenia. *Journal of Computer Assisted Tomography*, *17*, 704–709.
- Hu, L.-T., & Bentler, P. M. (1995). Evaluating model fit. In R. H. Hoyle (Ed.), *Structural equation modeling. Concepts, issues, and applications* (pp. 76–99). Thousand Oaks, CA: Sage.
- Jacobs, G. D., & Snyder, D. (1996). Frontal brain asymmetry predicts affective style in men. *Behavioral Neuroscience*, *110*, 3–6.
- Jöreskog, K. G., & Sörbom, D. (1989). *LISREL 7. A guide to the program and applications*. Chicago: SPSS.
- Kang, D.-H., Davidson, R. J., Coe, C. L., Wheeler, R. E., Tomarken, A. J., & Ershler, W. B. (1991). Frontal brain asymmetry and immune function. *Behavioral Neuroscience*, *105*, 860–869.
- Katznelson, R. D. (1981). EEG recording, electrode placement, and aspects of generator localization. In P. L. Nunez (Ed.), *Electric fields of the brain* (pp. 176–213). New York: Oxford University Press.
- Kemali, D., Vacca, L., Marciano, F., Nolfé, G., & Iorio, G. (1981). CEEG findings in schizophrenics, depressives, obsessives, heroin addicts and normals. *Advances in Biological Psychiatry*, *6*, 17–28.
- Lehmann, D. (1987). Principles of spatial analysis. In A. S. Gevins & A. Rémond (Eds.), *Handbook of electroencephalography and clinical neurophysiology. Methods of analysis of brain electrical signals and magnetic signals* (Rev. series, Vol. 1, pp. 309–354). Amsterdam: Elsevier.
- MacCallum, R. C. (1995). Model specification: Procedures, strategies, and related issues. In R. H. Hoyle (Ed.), *Structural equation modeling. Concepts, issues, and applications* (pp. 16–36). Thousand Oaks, CA: Sage.
- Meyer, T. J., Miller, M. L., Metzger, R. L., & Borkovec, T. D. (1990). Development and validation of the Penn State Worry Questionnaire. *Behaviour Research and Therapy*, *28*, 487–495.
- Miller, G. A., Lutzenberger, W., & Elbert, T. (1991). The linked-reference issue in EEG and ERP recording. *Journal of Psychophysiology*, *5*, 273–276.
- Neurosoft. (1993). *Scan manual II* (Version 3.0). Sterling, VA: Author.
- Nitschke, J. B., Heller, W., Palmieri, P. A., & Miller, G. A. (1999). Contrasting patterns of brain activity in anxious apprehension and anxious arousal. *Psychophysiology*, *36*, 628–637.
- Nunez, P. L. (1981). *Electric fields of the brain. The neurophysics of EEG*. New York: Oxford University Press.
- Nunez, P. L. (Ed.). (1995). *Neocortical dynamics and human EEG rhythm*. New York: Oxford University Press.
- Oldfield, R. C. (1971). The assessment and analysis of handedness: The Edinburgh Inventory. *Neuropsychologia*, *9*, 97–113.
- Papousek, I., & Schuller, G. (1998). Different temporal stability and partial independence of EEG asymmetries from different locations: Implications for laterality research. *International Journal of Neuroscience*, *93*, 87–100.

- Pauli, P., Wiedermann, G., & Nickola, M. (1999). Pain sensitivity, cerebral laterality, and negative affect. *Pain, 80*, 359–364.
- Perris, C., von Knorring, L., Cumberbach, J., & Marciano, F. (1981). Further studies of depressed patients by means of computerized EEG. *Advances in Biological Psychiatry, 6*, 41–49.
- Petruzzello, S. J., & Landers, D. M. (1994). State anxiety reduction and exercise: Does hemispheric activation reflect such changes? *Medicine and Science in Sports and Exercise, 26*, 1028–1035.
- Petruzzello, S. J., & Tate, A. K. (1997). Brain activation, affect, and aerobic exercise: An examination of both state-independent and state-dependent relationships. *Psychophysiology, 34*, 527–533.
- Polich, J., & Lawson, D. (1985). Event-related potential paradigms using tin electrodes. *American Journal of EEG Technology, 25*, 187–192.
- Pollock, V. E., & Schneider, L. S. (1990). Topographic quantitative EEG in elderly subjects with major depression. *Psychophysiology, 27*, 438–444.
- Reid, S. A., Duke, L. M., & Allen, J. J. B. (1998). Resting frontal electroencephalographic asymmetry in depression: Inconsistencies suggest the need to identify mediating factors. *Psychophysiology, 35*, 389–404.
- Robinson, R. G., Kubos, K. L., Starr, L. B., Rao, K., & Price, T. R. (1984). Mood disorders in stroke patients: Importance of lesion location. *Brain, 107*, 81–93.
- Rosenfeld, J. P. (2000). An EEG biofeedback protocol for affective disorders. *Clinical Electroencephalography, 31*, 7–12.
- Rosenfeld, J. P., Baehr, E., Baehr, R., Gotlib, I. H., & Ranganath, C. (1996). Preliminary evidence that daily changes in frontal alpha asymmetry correlates with changes in affect in therapy sessions. *International Journal of Psychophysiology, 23*, 137–141.
- Saletu, B., Brandstätter, N., Metka, M., Stamenkovic, M., Anderer, P., Semlitsch, H. V., et al. (1996). Hormonal, syndromal and EEG mapping studies in menopausal syndrome patients with and without depression as compared with controls. *Maturitas, 23*, 91–105.
- Schaffer, C. E., Davidson, R. J., & Saron, C. (1983). Frontal and parietal electroencephalogram asymmetry in depressed and nondepressed subjects. *Biological Psychiatry, 18*, 753–762.
- Schmidt, M. E., Ernst, M., Matochik, J. A., Maisog, J. M., Pan, B.-S., Zametkin, A. J., & Potter, W. Z. (1996). Cerebral glucose metabolism during pharmacologic studies: Test–retest under placebo conditions. *Journal of Nuclear Medicine, 37*, 1142–1149.
- Schmitt, M., & Steyer, R. (1990). Beyond intuition and classical test theory: A reply to Epstein. *Methodika, 4*, 101–107.
- Schmitt, M., & Steyer, R. (1993). A latent state-trait model (not only) for social desirability. *Personality and Individual Differences, 14*, 519–529.
- Spielberger, C. D., Gorsuch, R. L., & Lushene, R. E. (1970). *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists Press.
- SPSS. (1997). *SPSS 7.5 statistical algorithms*. Chicago: Author.
- Stelmack, R. M. (1990). Biological bases of extraversion: Psychophysiological evidence. *Journal of Personality, 58*, 293–311.
- Stemmler, G., & Meinhard, E. (1990). Personality, situation and physiological arousability. *Personality and Individual Differences, 11*, 293–308.
- Steyer, R. (1987). Konsistenz und Spezifität: Definition zweier zentraler Begriffe der Differentiellen Psychologie und ein einfaches Modell zu ihrer Identifikation [Consistency and specificity: Definition of two pivotal concepts of differential psychology and a simple model for their identification] *Zeitschrift für Differentielle und Diagnostische Psychologie, 8*, 245–258.
- Steyer, R. (1988). *Experiment, Regression und Kausalität: Die logische Struktur kausaler Regressionsmodelle* [Experiment, regression, and causality: The logical structure of causal regression models]. Unpublished thesis, Universität Trier, Trier, Germany.
- Steyer, R. (1998). Eigenschafts- und Zustandskomponenten im moralischen Urteil und Verhalten. [Trait and state components in moral judgment and behavior]. In B. Reichle & M. Schmitt (Eds.), *Verantwortung, Gerechtigkeit und Moral: zum psychologischen Verständnis ethischer Aspekte im menschlichen Verhalten* (pp. 191–200). Weinheim, Germany: Juventa-Verlag.
- Steyer, R., Ferring, D., & Schmitt, M. (1992). States and traits in psychological assessment. *European Journal of Psychological Assessment, 8*, 79–98.
- Steyer, R., & Schmitt, M. (1990). The effects of aggregation across and within occasions on consistency, specificity and reliability. *Methodika, 4*, 58–94.
- Steyer, R., Schmitt, M., & Eid, M. (1999). Latent state-trait theory and research in personality and individual differences. *European Journal of Personality, 13*, 389–408.
- Steyer, R., Schwenkmezger, P., & Auer, A. (1990). The emotional and cognitive components of trait anxiety: A latent state-trait model. *Personality and Individual Differences, 11*, 125–134.
- Sutton, S. K., & Davidson, R. J. (1997). Prefrontal brain asymmetry: A biological substrate of the behavioral approach and inhibition systems. *Psychological Science, 8*, 204–210.
- Thayer, R. E. (1986). Activation–deactivation adjective check list: Current overview and structural analysis. *Psychological Reports, 58*, 607–614.
- Tomarken, A. J., & Davidson, R. J. (1994). Frontal brain activation in repressors and nonrepressors. *Journal of Abnormal Psychology, 103*, 339–349.
- Tomarken, A. J., Davidson, R. J., & Henriques, J. B. (1990). Resting frontal brain asymmetry predicts affective responses to films. *Journal of Personality and Social Psychology, 59*, 791–801.
- Tomarken, A. J., Davidson, R. J., Wheeler, R. E., & Doss, R. C. (1992). Individual differences in anterior brain asymmetry and fundamental dimensions of emotion. *Journal of Personality and Social Psychology, 62*, 676–687.
- Tomarken, A. J., Davidson, R. J., Wheeler, R. E., & Kinney, L. (1992). Psychometric properties of resting anterior EEG asymmetry: Temporal stability and internal consistency. *Psychophysiology, 29*, 576–592.
- Tucker, D. M., Stenslie, C. E., Roth, R. S., & Shearer, S. L. (1981). Right frontal lobe activation and right hemisphere performance: Decrement during a depressed mood. *Archives of General Psychiatry, 38*, 169–174.
- Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*, 1063–1070.
- Watson, D., Weber, K., Assenheimer, J. S., Clark, L. A., Strauss, M. E., & McCormick, R. A. (1995). Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology, 104*, 3–14.
- West, S. G., Finch, J. F., & Curran, P. J. (1995). Structural equation models with nonnormal variables. In R. H. Hoyle (Ed.), *Structural equation modeling. Concepts, issues, and applications* (pp. 56–75). Thousand Oaks, CA: Sage.
- Wheeler, R. E., Davidson, R. J., & Tomarken, A. J. (1993). Frontal brain asymmetry and emotional reactivity: A biological substrate of affective style. *Psychophysiology, 30*, 82–89.
- Wiedermann, G., Pauli, P., Dengler, W., Lutzenberger, W., Birbaumer, N., & Buchkremer, G. (1999). Frontal brain asymmetry as a biological substrate of emotions in patients with panic disorders. *Archives of General Psychiatry, 56*, 78–84.
- Yung, Y.-F., & Bentler, P. M. (1996). Bootstrapping techniques in analysis of mean and covariance structures. In G. A. Marcoulides & R. E. Schumacker (Eds.), *Advanced structural equation modeling: Issues and techniques* (pp. 195–226). Mahwah, NJ: Erlbaum.
- Zinser, M. C., Fiore, M. C., Davidson, R. J., & Baker, T. B. (1999). Manipulating smoking motivation: Impact on an electrophysiological index of approach motivation. *Journal of Abnormal Psychology, 108*, 240–254.

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