

11-12-2007

Prefrontal Cortex Asymmetry and the Regulation of Communication: A Meta-Analytic Study

Michelle Elaine Pence

University of Missouri-St. Louis, mep26a@umsl.edu

Follow this and additional works at: <http://irl.umsl.edu/thesis>

Recommended Citation

Pence, Michelle Elaine, "Prefrontal Cortex Asymmetry and the Regulation of Communication: A Meta-Analytic Study" (2007). *Theses*. 61.

<http://irl.umsl.edu/thesis/61>

This Thesis is brought to you for free and open access by the Graduate Works at IRL @ UMSL. It has been accepted for inclusion in Theses by an authorized administrator of IRL @ UMSL. For more information, please contact marvinh@umsl.edu.

Prefrontal Cortex Asymmetry and the Regulation of Communication:

A Meta-Analytic Study

by

Michelle E. Pence

B.A., Communication Studies, University of Missouri- Kansas City, 2005

A Thesis

Submitted to the Graduate School of the

University of Missouri- St. Louis
in partial fulfillment of the requirements for the degree

Master of Arts

Communication

November 2007

Advisory Committee

Alan D. Heisel, Ed.D.
Chairperson

Yan Tian, Ph.D.

Amber Reinhart, Ph.D.

Table of Contents

CHAPTER 1

Introduction and Review of Literature.....	p 6
Review of Literature.....	p 9
Neurobiological Foundations of the Communibiological Model.....	p 9
Prefrontal Cortex Asymmetry and Self-Regulation.....	p 18
The Present Study.....	p 20

CHAPTER 2

Method.....	p 22
Identification of Studies.....	p 22
Selection of Studies.....	p 23
Analytical Model.....	p 24

CHAPTER 3

Results.....	p 32
Correcting for Artifacts.....	p 36
Overall Analysis.....	p 40
Moderator Search.....	p 41

CHAPTER 4

Discussion.....	p 44
Overview.....	p 44
Theoretical Implications.....	p 46
Implications for Research.....	p 49
Conclusion.....	p 52

APPENDIX.....p 72

Tables

Table 1.....	32
Table 2.....	37
Table 3.....	43

Abstract

A rapidly growing body of research indicates that individual differences in the ability to regulate emotions depends on symmetric functioning in the prefrontal cortex. When prefrontal functioning is asymmetrical- especially when the right side is more active than the left- a lack of emotional control is more likely. This lack of emotional regulation has consequences for affective social interaction and, therefore, communication. Although a great deal of theoretical speculation abounds regarding the research literature, conclusions have been based on narrative literature reviews. In the present study, a psychometric meta-analysis of studies that examined the relationship between prefrontal asymmetry and variables related to temperament, personality, and communication was conducted. Results indicated that the mean correlation between prefrontal asymmetry and communication-related constructs was .410 and that all of the variance in the cluster of studies could be attributed to sampling error. The results for the temperament/personality cluster indicated a mean correlation of .33 and that all of the variance in the correlations was due to sampling error once a few anomalous effects were removed. Overall, the findings have implications for communication theory, especially with respect to neurobiological functioning and self-regulation.

CHAPTER 1

Introduction and Review of Literature

Nearly a decade ago, Beatty and McCroskey (1998) proposed the communibiological paradigm in which individual differences in communication functioning were conceptualized as manifestations of individual differences in the neurobiological systems. Communication functioning referred to a broad range of constructs including behaviors, traits, and mental processes. Beatty and McCroskey's emphasis on neurobiological underpinnings of communication functioning led them to conclude that a "neurobiology" of communication was necessary (p. 46). Although early communibiological research relied on self-report measures as proxy indicators of the sensitivity of neurobiological systems (e.g., neuroticism was used as an index of behavioral inhibition system sensitivity and extraversion was employed as an index of behavioral activation system sensitivity in Beatty, McCroskey, & Heisel, 1998), the neurobiological systems thought to underlie communication functioning were described in great anatomical detail (For examples, see Beatty & McCroskey, 1997; Beatty, McCroskey, & Heisel, 1998). In recent years, however, researchers have begun to study brain activity with respect to

communication functioning more directly (e.g., Beatty & Heisel, 2007; Heisel & Beatty, 2006).

Beatty and McCroskey (1998) initially posited, among other things, that (1) all mental and motor processes involved in communication depend on brain activity, and (2) brain activity precedes psychological experience. Beatty and his colleagues (Beatty, McCroskey, & Valencic, 2001) reaffirmed these propositions in later work on the subject. These propositions led Beatty and McCroskey to a “decidedly reductionistic” position regarding the brain-mind problem (p. 48). With respect to the brain-mind problem, physical reductionism holds that all brain processes are initiated and regulated by other brain processes rather than by a meta-physical force (e.g., consciousness or will).

Prominent scholars interested in brain activity and mental functioning (e.g., Clark, 2005; Davidson, 2000; Pinker, 1997; Tooby & Cosmides, 2000; Wilson, 1994) recognize that one of the challenges that accompanies physical reductionism is to identify the brain processes which regulate other brain processes. In part, meeting this challenge includes the identification of brain regions involved in regulation of other brain activities. A considerable amount of neuroscience research points toward the prefrontal cortex (PFC) as a likely region in which regulatory functions are executed (e.g., Bachara et al., 1996; Beatty & Heisel, 2007; Beatty & Lewis, in press; D’Esposito et al., 1995; Damasio, 1995; Heisel & Beatty, 2006; Stuss & Benson, 1986; Tanji & Hoshi, 2001). However, it is also important to identify activity patterns within regions that correspond to different behavioral outcomes when regions such as the PFC perform multiple functions.

One of the most persistent questions for communibiology stemming from the reductionist foundation concerns the degree to which individuals can regulate their

emotions and exert control over their behavior in social situations (Beatty, McCroskey, & Valencic, 2001). On the basis of a twins study, Beatty, Marshall, and Rudd (2001) concluded that the ability to adapt to situational demands is to a large extent inherited. Based on Beatty and McCroskey's (1998) observation that neurobiological systems, not behaviors, are inherited, it makes sense to suspect that individual differences in adaptability or the ability to regulate and exert behavioral control must be traceable to individual differences in the brain functioning.

In recent years, a huge body of research literature has accumulated regarding asymmetrical activity, especially in the PFC, and individual differences in emotional regulation and behavioral control (Representative summaries include Clark, 2005; Davidson, 2000). Much of this research bears directly on constructs studied in the communication discipline. Examples include shyness (Schmidt, 1999; Schmidt & Fox, 1994), public speaking anxiety (Davidson, Marshall, Tomarken, & Henriques, 2000), facial expressions (Coan & Allen, 2001), perspective-taking (Sabbagh & Flynn, 2006), and social competence (Henderson, Marshall, Fox, & Rubin, 2004). In addition, some studies examined the relationship between prefrontal asymmetry and constructs such as behavioral inhibition/behavioral activation (Hewig et al., 2006; Sutton & Davidson, 1997) and neuroticism (Minnix & Kline, 2004); which are central to communication models of communication apprehension (Beatty, McCroskey, & Heisel, 1998) and trait verbal aggressiveness (Beatty & McCroskey, 1997).

Although the PFC asymmetry literature regarding clinical variables such as depression and chronic anxiety have been meta-analyzed (Thibodeau, Jorgensen, & Kim, 2006), the roughly 25 studies focused on PFC asymmetry and variables of interest to

communication scholars, such as verbal aggressiveness, shyness, and sociability, has not been summarized. Therefore, a meta-analytic review (Hunter & Schmidt, 2004) of those studies is proposed to provide an estimate of the average strength of association between PFC asymmetry and the variables of interest and to provide an assessment of the robustness of the association. In this chapter, a rationale for such a study is developed by reviewing the theoretical and research literatures pertaining to (1) the neurobiological foundation of the communibiological model, and (2) the role of PFC asymmetry in self-regulation.

Review of Literature

Neurobiological Foundations of the Communibiological Model

Overview and background. In 1991, Cappella argued that “the biological origins are as important to understanding aspects of human communication as are its social origins...because we are, after all, biological organisms” (p. 5). Seven years later, Beatty and McCroskey (1998) pushed the issue further, arguing that the biological origins are not merely as important, but far more important than social origins of social interaction patterns. In fact, Beatty and McCroskey (1998) referred to the influence of environment or situation as “negligible” (p. 50) or having only a “minimal direct effect on communication functioning” (Beatty, McCroskey, & Valencic, 2001, p. 78).

Beatty and McCroskey’s thinking was heavily influenced by the results of twins studies conducted by behavioral geneticists, studies of infants and children conducted under the rubric of temperament, and work done in the emerging field of cognitive neuroscience (Beatty, McCroskey, & Valencic, 2001). Informed by this work, Beatty and McCroskey proposed a model of communication, known as the *communibiological*

paradigm, in which communicative functioning was seen as a manifestation of neurobiological activation and that individual differences in communication traits, behaviors, or processes such as subjective interpretation of messages represented individual differences in neurobiological functioning- either thresholds for activating systems or intensity once activated. The proposition that situation or environment had little direct main effect on communication functioning was based on the assumption that all input from the environment is mediated through individuals' neurobiological systems (Beatty, McCroskey, & Valencic, 2001) and since individuals' neurobiology systems vary, a constant input would result in varied interpretation.

Neurobiological foundation of early work. The initial work under the rubric of communibiology focused on communicator traits. Beatty, McCroskey, and Heisel (1998) reconceptualized the communication apprehension construct to reflect the research literatures in behavioral genetics, temperament, and cognitive neuroscience which had accumulated since the construct's initial introduction to the discipline (McCroskey, 1978). Working from Gray's (1991) three-component neurobiological model of temperament, Beatty, McCroskey, and Heisel theoretically mapped the neurobiology of communication apprehension.

According to Gray (1991) three interconnected neurobiological systems account for temperament, the behavioral activation system (BAS), the behavioral inhibition system, (BIS), and the fight or flight system (FFS). The BAS energizes goal-directed behavior. Anatomically, the BAS consists of "the basic nuclei, the neocortical regions connected to it, the dopaminergic fibers that extend from the midbrain, and the thalamic nuclei" (Beatty & McCroskey, 1998, p. 52). The BIS responds to the perception of novel stimuli

or those associated with anticipated punishment or cessation of rewards, producing “increased arousal as a function of its connection with the limbic system, increased attentional focus on threatening stimuli due to anterior attention network involvement, and cessation of contemporaneous behavior” (Beatty & McCroskey, 1998, p. 52).

According to Beatty and McCroskey (1998), the BIS system consists of the following:

...hippocampus, the subiculum, and the limbic system, which consists of the medial wall of the limbic lobe, the olfactory cortex, the cingulate and subcallosal gyri, and the subcortical areas of the amygdala, hypothalamus, epithalamus, anterior thalamic nuclei and part of the basal nuclei (p. 52).

The FFS moderates the initiation of aggression or active withdrawal. The anatomical circuitry “interconnects the basolateral and centromedial nuclei of the amygdale, the ventromedial nucleus of the hypothalamus, the central gray region of the midbrain, and the somatic and motor nuclei of the lower brain stem” (Beatty & McCroskey, 1998, p. 52). Beatty and McCroskey (1998) pointed out that “although humans share in common the anatomic features of the three systems described by Gray, individual differences in the reactivity of these structures produce individual differences in behavior” (p. 52).

Strelau (1994) observed that individual differences in neurobiological systems can take the form of:

...sensitivity to neurons postsynaptic receptors or sensitivity in their synaptic transmission, the amount of neurotransmitters being released, the activity of the neural structures (including structures) to different kinds of stimuli, all taking part in the determination of individual differences in traits (p. 135).

Beatty, McCroskey, and Heisel (1998) described communication apprehension as a function of BIS and BAS sensitivity. Specifically, the more reactive or sensitive the BIS and the less sensitive the BAS, the higher the apprehension. Although the model was consistent with an immense body of research (see Beatty et al. 1998 for a review), it was difficult to test. Ultimately, the model was tested using Eysenck's self-report measures of neuroticism and extraversion as proxy indicators of BIS and BAS reactivity. The rationale for these particular proxy measures was based on Gray's (1991) contention that Eysenck's three dimensions of personality (neuroticism, extraversion, and psychoticism) parallel the BIS, BAS, and FFS. As Gray put it, "temperament reflects parameter values" and "the major dimensions of personality...are created by individual differences in such parameter values" (p. 23). The results of Beatty, McCroskey, and Heisel's (1998) test employing proxy measures of the BIS and BAS were consistent with theoretical expectations.

Beatty and McCroskey (1997) conceptualized trait verbal aggressiveness as a manifestation of reactive FFS and BAS relative to BIS functioning. A later study using self-report proxy measures provided considerable support for the model (Valencic et al., 1998).

More recent theory and research. Communibiological research focused exclusively on BIS, BAS, and FFS neurobiological circuitry principally because Gray (1991) proposed them as neurological substrates of temperament and Beatty and his colleagues reconceptualized communicator traits in terms of temperament. A temperament-based paradigm worked reasonably well for traits with a strong emotional base because the overwhelming majority of BIS, BAS, FFS circuitry is comprised of subcortical brain

regions, which are more associated with affective than cognitive functioning. For instance, as recently as five years ago researchers successfully accounted for appreciable portions of variance in observable verbal aggressiveness and interpersonal affiliation using extraversion and neuroticism as proxy measures for the BAS and BIS neurobiological circuits (Heisel, La France, & Beatty, 2003). In the past two years, however, communication researchers have turned their attention to brain scanning techniques. Moreover, focus has been shifted to cortical activity because it can provide insight into cognitive processes that occur during social interaction. Because many of the cognitive functions implemented by specific sites in the cortex have been documented, it is possible to validate cognitive theories of social interaction against brain activity (Beatty & Heisel, 2007).

In recent studies, researchers have begun to identify the specific sites activated during particular types of communication functioning. Heisel and Beatty (2006) monitored cortical activity while experimental participants either thought about why a friend might refuse a request to borrow a CD or merely thought about the friend. Heisel and Beatty found significantly greater electrical activity in orbitofrontal and dorsolateral prefrontal cortices for participants thinking about the request refusal than for participants merely thinking about the friend. These findings were theoretically important because studies have shown that thoughts about the mental states of others are primarily processed in the orbitofrontal cortex (Baron-Cohen et al., 1994) and activity in the dorsolateral cortex has been associated with cognitive activity in response to novel challenges (D'Esposito et al., 1995).

In a second study, Beatty and Heisel (2007) measured electrical activity continuously while experimental participants planned a message designed to retrieve a loan from a friend. In one condition, experimental participants were given feedback that their influence attempt had failed, and revised their strategies. In the other condition, experimental participants were given positive feedback about their messages and were simply asked to respond to the same scenario multiple times. Consistent with theoretical expectations, Beatty and Heisel found that the level of electrical activity in the dorsolateral prefrontal cortex while planning messages when previous efforts had failed was significantly higher than during the initial influence attempt but that electrical activity decreased when experimental participants were allowed to use verbal plans that required no modification. These findings were especially significant because the decreasing activation in the dorsolateral prefrontal cortex, which is activated when dealing with novel tasks, documents the development of a communication routine (See Beatty & Heisel, 2007 for a discussion of routines).

Physical reductionism and regulatory processes. The commitment to *physical reductionism* has particular implications for theory development under a communibiological paradigm. Physical reductionism is one approach to what scholars have termed the “mind-brain” problem (Popper & Eccles, 1997), a derivative of the duality versus monism debate regarding the “mind-body” problem. Beatty and his colleagues (Beatty, McCroskey, & Valencic, 2001) were crystal clear about their philosophical stance:

Our view is decidedly reductionist, holding that introspective experience is a reflection of brain activity. The former is merely delayed in time.

What we perceive as cognition, which includes goals, intentions, and so forth, are to brain activity what the images on a screen while viewing a motion picture are to a reel of film. We might become emotionally involved in the images, we might form expectations and hopes concerning the outcome, but those experiences cannot alter the events unfolding on the screen (p. 73).

Physical reductionism is not merely stated as a collateral philosophical stance: It is embedded in the basic propositions underlying communibiology. For instance, the proposition that “communication functioning depends on brain activity” (Beatty, McCroskey, & Valencic, 2001, p.72), by its very nature, excludes meta-physical influences and limits communication to a prosaic source. Moreover, in earlier writing, Beatty and McCroskey (1998) elaborate on this point, simply stating that “theoretical speculation about thinking, feeling, and behaving during human interactions must be consistent with knowledge regarding the brain and brain related functioning” (p. 46), leaving no room for hypothetical constructs that are not traceable to neurobiological signatures. In a more explicit statement that excludes meta-physical explanatory states of consciousness, Beatty, McCroskey, and Pence (in press) revised the propositions regarding the dependence of communication functioning on brain activity to read “all mental processes involved in social interaction and reducible to brain activity.”

Beatty and his colleagues (Beatty, McCroskey, & Valencic, 2001) argue that a physical reductionist position, while widely held by theorists working in the area of brain behavior and social behavior (e.g., Clark, 2005; Davidson, 2000; Pinker, 1997; Tooby & Cosmides, 2000; Wilson, 1998), is not universally accepted by many in the behavioral

and social sciences. Indeed, within the “mind-brain” dialogue, two alternatives, *mentalism* and *interactionism* are implicit in the work of many humanistic oriented scholars (Beatty, McCroskey, & Valencic, 2001). Beatty, McCroskey, and Valencic (2001) describe those two standpoints:

The *mentalist approach* adopts philosophical dualism, positing that the conscious mind exists outside the brain (Popper & Eccles, 1977). This was the stance argued by Descartes. Accordingly, the mind gives directions to the brain. The *interactionist position*, also founded in dualism, holds that the mind and brain cooperate in the production of activity. Both alternatives to our position on the matter require that an outside agency or entity be posited. So, an initial question concerns the evidence for such an entity. Also, where does the entity reside and of what is it made? If the conscious mind and the brain are separate, through what mechanism do they interact? (p. 73)

Pinker (2002) makes the point that advocates of the mentalist and interactionist positions are identifiable by arguments that “we” can decide not to act in certain ways in spite of our genetic or evolutionary inheritance. In a manner similar to Beatty and McCroskey’s position regarding meta-physical forces, Pinker (2002) was unclear about what the “we” referred to if not the production of a neurological circuit that gives rise to self-awareness.

In spite of the empirical conundrums underlying mentalist and interactionist perspectives, their attraction is that they provide a sense of understanding- an often cited criterion of good theory (Reynolds, 1971)- regarding regulatory processes, as well as

possible ideological appeal (e.g., empowering human beings). Both mentalism and interactionism posit a force that intervenes and can redirect brain activity and can be marshaled as an explanation for human conduct that is not yet explained by scientific data.

Physical reductionism requires that not only are mental processes involved in social interaction reducible to brain activity but that the brain activity underlying mental process can be explained in terms of other brain activity. At the abstract level, Beatty and colleagues (2001) argued that:

...an individual's neurobiological program consisting of selective monitoring of the environment, thresholds for initiating molar behavioral responses, and reflection on the consequences of actions, are principally inborn. The "agent" of action consists of neurobiological manifestations of evolutionary imperatives, fitted to the individual's temperament (p. 74).

The prominent sociobiological theorist, Edward O. Wilson (1998) made a similar proposal, but both Beatty et al.'s position and Wilson's are highly abstract.

Several theorists have argued that the task for reductionist approaches to social behavior is to isolate the specific neurobiological processes that implement regulatory pressures as well as those responsible for impulses (Beatty & Lewis, in press; Clark, 2005; Davidson, 2000; Wilson, 1998; Tooby and Cosmides, 2000). Although a great deal is known about where mental processes are implemented once systems are activated (For a review of mental processes related to social interaction and communicator traits see Beatty & Heisel, 2007; Beatty & Lewis, in press; Beatty, McCroskey, & Heisel, 1998; Beatty, McCroskey, & Valencic, 2001; Heisel & Beatty, 2006), regulatory

processes that influence whether systems are activated or suppressed are comparatively understudied in communication research. However, a huge and growing body of research and theory examining the neurobiological regulation of emotional expression and behavior has accumulated across a variety of fields.

Prefrontal Cortex Asymmetry and Self-Regulation

A considerable amount of research indicates that executive functions take place in the prefrontal cortex (D'Esposito et al., 1995; Fuster, 1999; Tanji & Hoshi, 2001). Indeed, Tanji and Hoshi (2001) pointed out that “generating purposeful action is the cardinal aspect of the cognitive functions of the prefrontal cortex (PFC)” (p. 164). Moreover, neuroscientists are beginning to identify sites in the PFC that implement specific sets of cognitive functions. For instance, conscious reflection on the possible consequences of a course of action have been linked to prefrontal orbital and ventromedial PFC activation (Bachara, Tranel, & Damasio, 1996), understanding how a plan of action might proceed is also associated with the ventromedial PFC (Damasio, 1995) and downloading episodic memory is associated with the right anterior cortex (Buckner, 2000). However, a sizeable corpus of research (for reviews of this research, see Cacioppo, 2004; Coan & Allen, 2004; Davidson, 1995, 2000; Fox, 1994) has been published which indicates that an individual's ability to regulate emotional responses to social stimuli and to control behavioral impulses depends in part on the degree of symmetry in basal level activation of the left and right frontal (anterior) PFC: The greater the symmetry, the greater the ability to self-regulate. The overwhelming majority of studies measure electrical activity in the alpha range using electroencephalographs. Asymmetry is most often operationally defined as the differences between activity levels in the left and right anterior PFC. In

general, deficits in self-regulation are more likely when right anterior activation is greater than left anterior activation because right anterior PFC is associated with emotional reactivity (Davidson, 2000).

A literature search (which is discussed in detail in Chapter 2) turned up over 300 published articles and book chapters dedicated to PFC asymmetry and various outcome variables. Of those published research pieces, a huge number focused on asymmetry and self-regulatory problems that can be classified as mental or behavioral disorders. Within this subgroup of articles and chapters, researchers studied asymmetry and panic disorders (Wiedemann et al., 1999), autism (Stroganova et al., 2007), mood and general affect (Harmon-Jones & Allen, 1997; Papousek & Shulter, 2002; Rosenfeld et al., 1996; Urry et al., 2004), and clinical depression (Allen, Urry, Hitt, & Coan, 2004; Baehr, Rosenfeld, Baehr, & Earnest, 1998; Blackhart, Minnix, & Kline, 2006; Bruder et al., 2001; Davidson, Schaffer, & Saron, 1985; Dawson et al., 1992, 1997, 1999; Dawson, Panagiotides, Klinger, & Spieker, 1997; Debenor et al., 2000; Field, Fox, Pickens, & Nawrocki, 1998; Field et al., 2000; Forbes et al., 2006; Gilbert et al., 1999; Gilbert, Meliska, Welser, & Estes, 1994; Gotlib, Ranganath, & Rosenfield, 1998; Harmon-Jones et al., 2002; Henriques & Davidson, 1990, 1991; Jones, Field, Davalos, & Pickens, 1997; Jones et al., 1997, 1998; Kentgen et al., 2000; Lewis, Weeks, & Wang, 2007; Minnix et al., 2004; Miller et al., 2002; Posthuma, Boomsma, & De Geus, 2007). A handful of other asymmetry studies focused on outcomes such as creative thinking (Molle et al., 1999), meditation states (Davidson, 2004; Travis & Arenander, 2004), motor responses (Miller & Tomarken, 2001), and infant reactions to maternal separation (Davidson & Fox, 1989; Fox & Davidson, 1987).

The Present Study

Over forty studies focused on traits, behaviors, and processes that are of interest to communication researchers, especially those taking a biological perspective on social interaction (The criteria for selecting these studies are described in Chapter 2). Furthermore, the specific studies along with relevant data are presented in Chapter 3. However, these studies have not been summarized. A quantitative review summary in the form of meta-analysis would provide an estimate of the average magnitude of association between PFC asymmetry and the outcome variables and an estimate of the robustness of the observed effect (Hunter & Schmidt, 2004). In light of the overlap between the constructs contained in the study sample and those examined by communication researchers, the results of a meta-analysis would provide insight about the potential of PFC asymmetry as a neurobiological substrate of self-regulation in social contexts. This would constitute valuable guidance to theorists and researchers prior to designing studies and investing considerable resources into conducting them.

The rationale for the present study was framed within a communibiology paradigm. Developments in the communibiological paradigm have particular conceptual implications for analytic discussions regarding data analysis. Psychologists interested in cerebral asymmetry as a substrate for mental processes have argued that research and theory would profit by moving from the simple analysis or correlational relationship to positing causal processes (see, for example, Cacioppo, 2004; Coan & Allen, 2006). Importantly, a clearly articulated, albeit evolving, causal model depicting the temporal order of constructs has appeared in the communibiological literature for nearly a decade. The most recent model depicts one causal chain in which the relationship between

neurobiological functioning (e.g., PFC asymmetry) and communicator traits and behavior (e.g., shyness or verbal aggression) is mediated by dimensions of temperament or personality (Beatty, 2005). For example, the model suggests that neurobiological functioning leads to temperamental characteristics (such as negative affectivity or neuroticism) which in turn leads to an associated trait such as shyness or verbal aggressiveness.

Among the empirical implications of a mediated model is that the correlation between neurobiological functioning and communication functioning should be equal to the product of the neurobiological functioning/temperament correlation and the temperament/communication functioning correlation (Beatty, 2005). In terms of the present study, the relative effect sizes for studies focused on temperament or personality variables and those for communication-related studies might be especially illuminating with respect to the mediated model advanced in the communibiological literature. If the mediated chain is correct, the average effect size for PFC asymmetry/temperament studies should be larger than the average effect for the asymmetry/communication studies. Therefore, in the face of possible evidence of moderator variables, a theoretically-driven moderator search would strongly suggest a temperament/personality versus communication-related variables focus.

CHAPTER 2

Method

Conducting meta-analysis involves (1) identification of a sample of studies, (2) selection of studies for analysis based on a set of conceptually driven criteria, and (3) specification of an analytic model (Hunter & Schmidt, 2004). In general, the meta-analytic procedures in the present study were patterned after previous meta-analyses of research domains consisting of traits, social behaviors, and cognitive and affective process (Beatty et al., 2002; La France, Heisel, & Beatty, 2004).

Identification of Studies

The literature search consisted of three steps. First, an electronic search of PsychINFO and EBSCOhost databases was conducted. In order to complete an exhaustive search of the published work involving asymmetry and variables of interest, (1) a broad search base on keywords such as “EEG,” “electroencephalograph,” “prefrontal cortex,” “anterior cortex,” “personality,” and “behavior,” and (2) a narrow search using “neuroticism,” “extraversion,” “facial expression,” “shyness,” “anxiety,” “prefrontal asymmetry” and “anterior prefrontal cortex asymmetry” were executed. Second, reference lists from literature reviews and recently published asymmetry studies

were reviewed and a list of published studies not appearing in electronic searches was compiled. Third, a list of journals that have published studies of asymmetry was compiled and the journals were searched for asymmetry studies not detected through either the electronic or reference list searches.

Selection of Studies

Overall, the search uncovered over 300 citations. Although the search was highly rigorous, yielding many duplications, many of the studies were not relevant to the present study. Even after duplications were eliminated, many studies remained irrelevant to the present study (although they are included in the reference list). The pool of articles and chapters were pruned in the following five ways. First, review essays, methodological discussions, editorials, and other essays that did not contain original data, or contained insufficient results for calculating effect sizes (e.g., merely reported “ns” for non-significant findings or merely reported $p < .05$ for significant findings without accompanying test statistics or means and standard deviations) were eliminated. Second, articles that focused on mental or behavioral disorders or employed samples classified as suffering from personality or behavioral disorders were removed because these samples often are medicated and/or classified as displaying abnormal brain functioning. Third, studies in which asymmetry in areas of the brain other than the anterior area in the PFC (e.g., parietal) were eliminated. Fourth, because the purpose of this study was to examine resting asymmetry as an explanatory variable, studies in which experimental stimuli were used in an attempt to induce asymmetry rather than examine resting asymmetry as an independent variable were deleted. Fifth, the remaining studies were examined for relevance to the goals of the present study. Specifically, studies kept examined the

empirical relationship between asymmetric PFC functioning and (1) variables included in studies published in communication journals, (2) variables included in review pieces published in major reference texts in communication (e.g., *Handbook of Interpersonal Communication*) and (3) variables included in communibiological models of traits, social behavior, and receiver processes. All others were discarded. The results of this procedure yielded 21 studies for analysis.

Analytical Model

The meta-analysis was conducted following the procedures described by Hunter and Schmidt (2004). Hunter and Schmidt's method assumes a random effects, rather than a fixed effect, statistical model. A random effects model was most appropriate for the present study because the assumptions of a fixed effects model would be inconsistent with the theoretical assumptions of the present study. For example, a fixed effects model assumes homogeneity of effects (i.e., r 's) across studies (Hedges & Vevea, 1998). However, the rationale for the present study makes it clear that moderators are likely. In addition, Hedges and Vevea (1998) argued that "the analyst may wish to make a different kind of inference, on that embodies an explicit generalization beyond the observed studies" (p. 487). In such cases, "Random-effects analysis procedures are designed to facilitate unconditional inferences" (p. 487). Ultimately, the object is to make inferences about the general relationship between left and right anterior power and communication or personality variables that would apply to studies not specifically included in this study. For both reasons, therefore, a random effects model seemed most appropriate.

Research indicates that Hunter & Schmidt's approach to estimating the sources of variance across a body of studies is the most accurate of the major approaches to meta-

analysis, especially when the number of studies is less than 70 (as it is in the present study) and the sample sizes per study are less than 100 (as they are in the present study) (Sackett, Harris, & Orr, 1986). The theoretical importance of communication variables versus temperament/personality as a potential moderator of asymmetry effects resides in the fact that “the Schmidt-Hunter procedure has a much higher likelihood of detecting moderator variables” (Sackett et al., p. 310).

A second major reason for choosing Hunter and Schmidt’s approach as the analytical model is that Hunter and Schmidt proposed the “most elaborate set of adjustments” (Rosenthal, 1991, p. 24) for correcting artifacts. A scan of the studies meta-analyzed in the present study showed that adjustments pertaining to measurement error and dichotomization of continuous measures would be required to put the effect sizes on a common metric (the details of these adjustments will be presented below). While one use of meta-analysis is to describe the state of the literature, some methodologists have argued that the best use of meta-analysis is to estimate the degree and nature of relationships not merely among measures, but of the underlying theoretical constructs as well. As Hunter & Schmidt put it, “the purpose is to estimate as accurately as possible the construct-level relationships in the population” (p. 462). Hunter and Schmidt attempt to uncover construct-level relationships by estimating “what research findings would have been had it been possible to conduct studies without methodological flaws” (p. 464). This is an ideal, of course, since it is not possible to make adjustments for every methodological flaw in a group of studies, especially when reports do not contain enough information to discover a flaw. However, Hunter and Schmidt argue that making the

corrections that can be made produces a cleaner picture of the true relationships than making no adjustments at all.

Unit of analysis. Following Hunter and Schmidt's (2004) recommendation, Pearson's r was employed as the basic unit of analysis. In addition to the preference for correlation coefficients as the units of analysis expressed by Hunter and Schmidt (2004), Rosenthal (1991) for that matter, the correlation coefficient is especially appropriate in the present study given its theoretical framework. The present study was anchored in the communibiological paradigm and was undertaken to assess the predictive power of prefrontal asymmetry with respect to temperamental and communication-related variables. In laying out the foundation for the communibiological paradigm, Beatty and McCroskey (1998, p. 44) explicitly established the correlation coefficient as the preferred measure of effect for assessing predictive power. Furthermore, Beatty and McCroskey adopted Guilford's (1956) scheme for interpreting correlations (i.e., almost negligible $<.20$; low, $.20-.40$; moderate, $.40-.70$; high, $.70-.90$; very high $>.90$). Indeed, this interpretive framework can be seen in Beatty's (2005) mediated effects model, mentioned earlier, meta-analyses conducted within the communibiological literature (e.g., Beatty et al., 2002) and Beatty's (2002) general scholarship regarding appropriate measures of effect. Therefore, the convention of using and interpreting the correlation coefficient as a measure of effect, which has been established in the communibiological literature, was followed in the present study.

Effect sizes other than r , were converted to r and correlation coefficients were calculated for studies in which effect sizes were not reported. In the present data set, studies not reporting correlations, reported either t or F tests statistics. These were

converted to correlation coefficients employing the following formula (Rosenthal, 1991, p. 19): r equals the square root of t -squared/ t -squared plus N minus 2, where F equals t -squared.

Correcting effects for measurement error. Measurement error reduces the observed correlation between two variables in a systematic way: The correlation between two variables cannot exceed the square root of the products of the reliability coefficients for those variables (Hunter and Schmidt, 2004). Therefore, even if the true correlation at the construct level is 1.00 (perfect), in a study in which the reliabilities of the two measures were .60 each, the observed correlation between the variables could not exceed .60. Hunter and Schmidt apply the correction for attenuation, which consists of dividing the observed correlation by the square root of the reliability coefficients. In addition to estimating the degree of relationship between the two constructs, absent measurement error, correcting for attenuation removes the variance among study results, due to different levels of measurement error among studies. Unless variance among study results, such as correlations, due to measurement error is removed, it can be mistaken as variance due to moderator variables.

In the present study, correlations between PFC asymmetry and dependent variables were corrected for attenuation. A review of the selected studies for analysis in the present study indicated that reliability coefficients were not reported in nine of the studies (the specific studies are identified in Chapter 3). Based on the proposition that a correction for measurement error based on an approximation of the missing reliability coefficient is preferable to no correction, reliability coefficients were estimated for studies with missing variables by searching the references cited in the research report as

the source for the measure or procedure. For example, Schmidt (1998) reported no reliability coefficients for the measures of shyness and sociability but did cite Cheek and Buss (1981) as the source of the measures. Thus, the reliability coefficients cited by Cheek and Buss were employed in the correction of attenuation procedures for Schmidt's results.

Hunter and Schmidt recommend correcting observed effect sizes for attenuation due to both variables. Unfortunately, however, reliability coefficients for PFC asymmetry scores were reported in only three studies (Blackhart & Kline, 2005; Fox et al., 1995; Wheeler et al., 1993). The decision not to correct asymmetry scores for attenuation was made because the available data indicate that to do so would likely obscure the relationship between PFC asymmetry and other variables for the following reasons. First, while the reliability estimates reported were quite low (i.e., the alpha reliability coefficients ranged from .43 to .57 in Blackhart & Kline, was .70 in Fox et al. and the test-retest reliability coefficient was .66 in Wheeler et al., 1993), other researchers describing the psychometric characteristics of PFC asymmetry scores in general have reported that "coefficient alphas were quite high, with all values exceeding .85, indicating that the electrophysical measures of asymmetric activation showed excellent internal consistency" (Davidson, 2000, p. 1153). If, in fact, coefficient alpha is greater than .85 in all studies, corrections based on the square root reliability coefficients greater than .85 would have little effect on correlations. However, Blackhart and Kline's report stands in contrast to Davidson's findings with a range of .43 to .85, it is unclear as to what estimate should be employed as a proxy for the actual reliability of the study data. Blackhart and Kline's estimate would greatly inflate the estimate of the true relationship between

asymmetry and other variables. On the other hand, estimates such as those reported by Davidson would effect little change. Furthermore, while applying a single estimate of reliability to the set of studies would increase the magnitude of the correlations, variance among study results that is actually due to variance in measurement error from one study to another would not be eliminated because such a procedure treats measurement error as uniform across studies.

Correcting for effects due to dichotomization. Sorting participants into “high” and “low” groups on the basis of their scores on a measure (i.e., dichotomizing) and statistically treating the groups as conditions or treatments results in different effect sizes than using the actual score on a measure as the variable. Cohen (1990) referred to dichotomizing continuous measures as “mutilating the variable” (p. 1306). In spite of the distortion of effects produced by dichotomization, it is still widely used among researchers (MaCallum, Zhang, Preacher, & Rucker, 2002).

Dichotomization can either attenuate or inflate the effect size, depending on whether the researchers use all of the data in their analysis. If researchers perform a median-split, assigning participants above the median to the “high” category and participants below the median to the “low” category, the effect size will be attenuated. If, on the other hand, the researchers assigned the upper ten percent to the “high” category and the bottom ten percent to the “low” category and omitted everyone in between from the analysis, the effect size will be artificially inflated (Hunter & Schmidt, 2004, pp. 36-39). In the set of studies to be meta-analyzed, dichotomization of continuous variables occurred in five studies, three performed median splits and two discarded midrange scorers (specific

studies are identified in Chapter 3). Hunter and Schmidt's (2004, pp. 36-39) procedures for correcting the effects of both types of dichotomization were followed.

Examining multiple effects. Nine studies reported two effects that were relevant to the present study. These studies are identified in Chapter 3 (Table 1 and 2). There are four general approaches to handling multiple effects (Hunter and Schmidt, 2004). First, if appropriate, the mean of the effects can be used. However, in the present data set, the mean correlation between variables such as behavioral inhibition system sensitivity and behavioral activation sensitivity would make little sense, especially if PFC asymmetry affects one but not the other. Second, if variables are intercorrelated, and the correlations are available, a composite variable can be constructed and treated as a single effect. However, the necessary data were not available in most studies. Third, if there are sufficient studies reporting effects for the same variables examined in studies reporting multiple effects, meta-analysis can be conducted separately for batches of variables including only one variable per meta-analyses from multiple effect studies. In the present study, however, in some cases the only reported effect for a variable is from a multiple effects report and the number of studies with common variables was quite low (see Table 1, Chapter 3). Finally, each effect can be treated statistically as a separate study. Certainly, this is the simplest approach and has been employed in our discipline (e.g., Beatty et al., 2002; La France et al., 2004). This approach violates the independence assumption if significance levels are employed as the unit of analysis (Rosenthal, 1984). However, Rosenthal (1984) notes critics "have confused the effect of nonindependence on significance testing with its effect on effect size estimation" (p. 27). Although, in general, treating multiple effects as independent is not Rosenthal's preferred approach,

statistically speaking, “there is certainly nothing wrong with doing so” (p. 27). In the present study, each effect or correlation was treated independently. As a consequence, the 21 studies yielded 30 effects.

Data analysis. Conducting the meta-analysis consists of the following three steps: (1) The average r weighted for sample size is computed, (2) the confidence interval for the average sample weighted r is calculated and (3) the variance in r expected due to sampling error is calculated. If the variance in r exceeds that expected due to sampling error, a moderator search is conducted. Moderators are factors responsible for differences or variance in the average r 's for clusters of studies. For example, if communication studies versus temperament/personality studies were a moderator as suggested in Chapter 1, meta-analyzing each set separately should result in variances in the correlations that are solely due to sample error, unless a second moderator exists within one of the cluster of studies (Hunter & Schmidt, 2004). The fewer the moderators, the more robust or generalizable the average r for the sample.

Because asymmetric functioning of the PFC is associated with what might be described as personally and socially problematic outcomes such as anxiety and aggression but symmetry tends to be associated with more positive and prosocial outcomes such as social competence and sociability, the direction of the correlations depend on the direction the communication and temperament variables are scored. Therefore, both positive and negative correlations can be consistent with the hypothesis depending on the variables predicted by asymmetry. Although negative correlations presented in tables include signs, the absolute value of correlations that were consistent with hypotheses were used for the purpose of meta-analysis.

CHAPTER 3

Results

The studies analyzed the variable associated with PFC asymmetry in each study, the effects expressed as correlation coefficients, sample size, gender and age composition of each sample, and reliability coefficients for each criterion variable are reported in Table 1.

Table 1

Studies, variables, uncorrected r's, sample size and composition, and reliability coefficients organized by moderator cluster.

Studies	Variable	<i>r</i>	N	Sample	Reliability
<i>Communication Studies</i>					
Blackhart & Kline, 2005	Defensiveness	.26	64	Female Adults	.85
Fox et al.,	Social	-.47	48	Male/Female	.85*

1995	Competence			Children	
Harmon-	Verbal	.28	26	Male/Female	.76
Jones &	Aggression			Children	
Allen, 1998					
Harmon-	Aggressive	.60	42	Male Adults	.91
Jones &	Response to				
Siegelman,	Insult				
2001					
Henderson	Wariness	.40	97	Male/Female	.90*
et al., 2001				Children	
Kline et al.,	Defensiveness	-.40	25	Male Adults	.85*
1998					
	Defensiveness	.30	60	Female	.85*
				Adults	
Kline et al.,	Defensiveness	.22	42	Female	.85*
2001				Adults	
Sabbagh &	Perspective-	-.49	23	-	.85*
Flynn, 2006	Taking				
Schmidt,	Sociability	-.52	40	Female	.70*
1999				Adults	
	Shyness	.59	40	Female	.79*
				Adults	
Schmidt &	Sociability	-.48	20	Female	.70*

Fox, 1994

Adults

Temperament/Personality

Coan &	BAS	-.44	32	Male/Female	.83
Allen, 2003				Adults	
	BIS	.17	32	Male/Female	.75
				Adults	
Tomarken et al., 1992	Positive Affect	-.20	84	Female	.88
	Negative Affect	-.14	84	Female	.87
				Adults	
Fink, 2005	Extraversion	-.26	66	Male/Female	.82*
				Adults	
Hagemann et al., 2005	Positive Affect	-.23	61	Male/Female	.74
	Negative Affect	-.06	61	Male/Female	.88
				Adults	
Hagemann et al., 1998	Positive Affect	.19	37	Male/Female	.88
	Negative Affect	.03	37	Male/Female	.87
				Adults	
Hewig et al., 2006	BAS	-.25	59	Male/Female	.85
				Adults	

	BIS	-.01	59	Male/Female	.88
				Adults	
Minnix &	Neuroticism	.23	72	Male/Female	.85
Kline, 2004				Adults	
Sobatka et	Reward	-.68	15	Male/Female	.85*
al., 1992				Adults	
Sutton &	BAS	-.40	34	Male/Female	.85
Davidson,				Adults	
1997					
	BIS	.41	34	Male/Female	.75
				Adults	
Tomarken &	Anxiety	.26	87	Female	.89
Davidson,				Adults	
1994					
Wheeler et	Positive	-.45	81	Female	.88*
al., 1993	Affect			Adults	
	Negative	.49	81	Female	.87*
	Affect			Adults	

*proxy reliability estimates

Corrections for Artifacts

Dichotomization of categorical variables. In three studies, continuous measures of defensiveness (Kline et al., 2001), extraversion (Fink, 2005) and neuroticism (Minnix & Kline, 2004) were split at the medium and all participants were assigned to either high or low groups and the data were analyzed via Analysis of Variance or *t*-test. The effects for these studies were corrected for bias due to dichotomization of a continuous variable were increased from .22 to .25 for defensiveness, -.26 to -.33 for extraversion, and from .23 to .29 for neuroticism. Two studies selected participants for high and low groups on the bases of extreme scores, excluding midrange scorers from the tests of hypotheses. Specifically, L. Schmidt and Fox (1994) compared the upper and lower 10% of scores on sociability in their analysis. As discussed in Chapter 2, selecting only extreme scores for difference tests artificially inflates the observed effect size (Hunter & Schmidt, 2004, p. 38). Correcting the effect for this treatment reduced the reported correlation from -.48 to -.33. In a later study, L. Schmidt (1999) repeated the error, which affected two effects, one for sociability ($r = -.52$) and another for shyness ($r = .59$). Correcting the effects for this artifact yielded correlations of -.37 for sociability and .43 for shyness.

Correction for measurement error. The mean reliability coefficient for the 30 effects was .835, making the average correction (i.e., .913) quite modest. The mean reliability coefficient for the studies in which reliabilities were reported was .84 compared to .82 for the proxy reliabilities estimated on the bases of other research. Because the correction for attenuation is based on the square root of the reliability coefficient, there was little difference in the amount of correction for studies in which reliabilities were reported (mean root of reliability = .91) and the proxy reliability estimates (mean root of

reliability= .92). Therefore, the corrections based on the proxy estimates were quite similar to the corrections derived from calculated reliabilities. Table 2 presents the same information and data as Table 1, except correlations corrected for dichotomization and measurement error are reported, instead of raw correlation coefficients.

Table 2

Studies, variables, corrected r, N organized by moderator variables.

Study	Variable	Rc	N	Sample
<i>Communication Studies</i>				
Blackhart & Kline, 2005	Defensiveness	.28	64	Female Adults
Fox et al., 1995	Social Competence	-.51	48	Male/Female Children
Harmon-Jones & Allen, 1998	Verbal Aggression	.24	26	Male/Female Children
Harmon-Jones & Sigelman, 2001	Aggressive Response to Insult	.63	42	Male Adults
Henderson et al., 2001	Wariness	.44	97	Male/Female Children
Kline et al.,	Defensiveness	-.43	25	Male Adults

1998

	Defensiveness	.33	60	Female Adults
--	---------------	-----	----	---------------

Kline et al.,	Defensiveness	.27	42	Female Adults
---------------	---------------	-----	----	---------------

2001

Sabbagh & Flynn, 2006	Perspective-Taking	-.53	23	-
-----------------------	--------------------	------	----	---

Schmidt, 1999	Sociability	-.44	40	Female Adults
---------------	-------------	------	----	---------------

	Shyness	.48	40	Female Adults
--	---------	-----	----	---------------

Schmidt & Fox,	Sociability	-.39	20	Female Adults
----------------	-------------	------	----	---------------

1994

Temperament/Personality

Coan & Allen,	BAS	-.48	32	Male/Female Adults
---------------	-----	------	----	-----------------------

2003

	BIS	.20	32	Male/Female Adults
--	-----	-----	----	-----------------------

Tomarken et al., 1992	Positive Affect	-.21	84	Female Adults
-----------------------	-----------------	------	----	---------------

	Negative Affect	-.14	84	Female Adults
--	-----------------	------	----	---------------

Fink, 2005	Extraversion	-.36	66	Male/Female Adults
------------	--------------	------	----	-----------------------

Hagemann et al., 2005	Positive Affect	-.36	61	Male/Female Adults
-----------------------	-----------------	------	----	-----------------------

		Negative Affect	-.06	61	Male/Female Adults
Hagemann et al., 1998		Positive Affect	.20	37	Male/Female Adults
		Negative Affect	.03	37	Male/Female Adults
Hewig et al., 2006		BAS	-.27	59	Male/Female Adults
		BIS	-.01	59	Male/Female Adults
Minnix & Kline, 2004		Neuroticism	.32	72	Male/Female Adults
Sobatka et al., 1992		Reward	-.74	15	Male/Female Adults
Sutton & Davidson, 1997		BAS	-.43	34	Male/Female Adults
		BIS	.47	34	Male/Female Adults
Tomarken & Davidson, 1994		Anxiety	.27	87	Female Adults
Wheeler et al., 1993		Positive Affect	-.48	81	Female Adults
		Negative Affect	.50	81	Female Adults

Overall Analysis

The results of psychometric meta-analysis, based on a random effects model (Hunter & Schmidt, 2004) conducted on sample weighted correlation coefficients that had been corrected for dichotomization and attenuation due to measurement error revealed a mean r of .327 ($N= 1543$, $K= 30$, 95% CI= .277-.377), where N equals the total sample size for the meta-analysis, K equals the number of effects, and CI represents the 95% confidence interval. Although 69.56% of the variance in r ($sr^2= .023$) was attributed to sampling error ($Se^2= .016$) alone, 30.44% of the variance was unexplained. Hunter and Schmidt (2004) recommended that moderator searches should be conducted whenever the variance unexplained after controlling for artifacts is greater than 25%. Hunter and Schmidt (2004) refer to this recommendation as the “75% rule.” As they put it:

If 75% or more of the variance is due to artifacts, we conclude that all of it is, on the grounds that the remaining 25% is likely to be due to artifacts for which no corrections has been made (p. 401).

Hunter and Schmidt admit that “the 75% rule is not a statistical test, but rather a simple ‘rule of thumb’ decision rule” (p. 401). However, Sackett, Harris, and Orr (1986) found in a simulation study that the 75% rule resulted in outcomes that were superior to the other methods in detecting variation in r , especially when the number of studies was not large (i.e., fewer than 64) and the average sample size was relatively small (i.e., 50 to 100). Both the number of studies (21) and the sample sizes of those studies (15 to 97) were in the range for which the 75% rule was superior to other approaches.

Moderator Search

In part, the rationale for the present study was based on the conceptual distinction between dimensions of temperament/personality and variables specifically aligned with social interaction. As the brief overview of Beatty's (2005) mediated-effects model illustrated, the average effect for the asymmetry-temperament relationship should be larger than the asymmetry-communication variable relationship because, according to the model, the effect of PFC asymmetry is mediated by temperament. Within the context of meta-analysis, whether the variable is related to temperament or communication should constitute a moderator.

The ten studies sorted into the communication sample included the following variables: shyness, verbal aggressiveness, responses to insults, sociability, defensiveness, social competence and perspective-taking in response to facial expressions. Two studies (Kline et al., 1998; Schmidt, 1999) reported two effects. Therefore, $K= 12$ for this cluster. Eleven studies were classified as temperament/personality in focus. Variables associated with PFC asymmetry in these studies were indices of the behavioral inhibition and behavioral activation system sensitivity (specifically emphasized in communibiology), neuroticism, extraversion, response to reward, and anxiety (also emphasized by Beatty, McCroskey, & Valencic, 2001), and two temperament dimensions, positive and negative affective style. Seven of those studies reported two effects (Coan & Allen, 2003; Hagemann et al., 1998, 2005; Hewig et al., 2006; Sutton & Davidson, 1997; Tomarken et al., 1992; Wheeler et al., 1993).

A meta-analysis conducted on only the communication cluster indicated that sampling error accounted for all of the variance in r . The mean correlation coefficient for the

communication cluster was .410 (N= 527, K= 12, 95% CI = .32 to .50). Sampling error variance ($Se^2 = .016$) accounted for 100% of the variance in r ($Sr^2 = .012$). In an attempt to discover whether the unexplained variance observed in r during the overall meta-analysis was attributable to the moderator, a meta-analysis was also conducted on the temperament/personality cluster. The mean r for this cluster was .284 (N= 1016, K= 18, 95% CI= .224-.344). Sampling error ($Se^2 = .015$) accounted for 57.69% in variance in r ($Sr^2 = .026$), falling short of the 75% benchmark.

An inspection of Table 2 indicated that the effects that most obviously are inconsistent with the pattern of correlations were those pertaining to BIS indices and negative affectivity then those seven effects were removed from the temperament/personality cluster, all of the variance in r ($Sr^2 = .013$) was attributable to sampling error ($Se^2 = .014$). Mean r was .33 (N= 628, K=11, 95% CI= .25-.41). However, the meta-analysis performed on the BIS/negative affective style cluster, left 54.05% of the variance unexplained (mean $r = .21$, N= 388, K=7, 95% CI= .11-.30; $Sr^2 = .037$, $Se^2 = .017$). Unfortunately, further exploration into the possible source of variation in the BIS/negative affectivity subgroup was beyond the scope of the present data because the identifiable moderators (sample composition and variable) were distributed across the effects contributing to the variation.

A summary of the overall meta-analysis and the moderator searches is presented in Table 3.

Table 3

Summary of overall meta-analysis

Analysis	r	Sr ²	Se ²	N	K	Variation Explained
Overall meta-analysis	.327	.023	.016	1543	30	69.56%
Communication Only	.410	.012	.016	527	12	100%
Temperament/personality	.284	.026	.015	1016	18	57.69%
Temperament/personality (BIS, Negative Affectivity removed)	.330	.013	.014	628	11	100%
BIS, Negative Affectivity Only	.210	.037	.017	388	7	45.95%

CHAPTER 4

Discussion

Overview

The present study was undertaken to provide a quantitative summary of the literature pertaining to the relationship between PFC asymmetry and two general sets of variables, one related to communication and the other related to temperament and personality. In addition to synthesizing the studies, a psychometric meta-analysis (Hunter & Schmidt, 2004) was conducted to glimpse the relationships between the underlying constructs. Specifically, correlations that had been corrected for attenuation, and in some cases for dichotomization, were employed as the unit of analysis. In the meta-analysis, the mean correlation (weighted for sample size), its 95% confidence interval, variance, and variance expected due to sampling error alone were computed.

A meta-analysis conducted on the entire sample provided a mean correlation of .33. The calculation of the variance in the correlations and the sampling error variance indicated the possible presence of at least one moderator variable. Guided by the mediated effects model posited first by Beatty (2005) and again by Beatty, McCroskey, and Pence (in press), the category of variable (communication versus

temperament/personality) was probed as a potential moderator of asymmetric PFC functioning. According to the model, the degree of symmetry/asymmetry in PFC functioning should lead to particular temperamental traits, which in turn, should lead to particular communication patterns. Not only should category of variable function as a moderator variable, but the specific pattern of results is dictated by the theoretical model: The mean correlation for temperament/personality construct should be larger than the mean correlation for communication-related variables.

When the cluster of studies focused on the communication-related constructs (shyness, verbal aggressiveness, etc.) was meta-analyzed, a somewhat larger mean correlation ($r = .410$) emerged. Moreover, inspection of the variance calculations revealed that after the artifactual influences of measurement error, dichotomization, and sample size were removed, all of the variance among the studies was attributable to sampling error. Therefore, the relationship between asymmetric PFC functioning and communication-related outcome variables is uniform across the studies of different constructs and samples when methodological artifacts and biases are controlled.

The results of the meta-analysis performed on the temperamental personality constructs were a lot more complicated. First, examination of the variance estimates indicated the likely presence of a moderator variable within the cluster. Second, and contrary to expectations, the mean correlation for the temperament/personality cluster was lower ($r = .284$) than the mean correlation in the communication cluster. An inspection of the correlations (see Table 2) within the cluster revealed correlations for some studies of negative affectivity and self-reported BIS sensitivity that deviated considerably from the mean correlation for the cluster. When all of the effects for those

constructs were removed, the mean correlation for the temperament/personality cluster improved slightly ($r = .33$), but remained smaller than the correlation for the communication constructs. However, when the negative affectivity and BIS effects were omitted, all of the remaining variance was attributable to sampling error.

The meta-analysis conducted on the negative affectivity and BIS cluster removed from the temperament/personality cluster indicated that sampling error also did not explain the variance in the correlations among the studies. It was not possible, however, to identify the moderator or moderator variables. There were moderate correlations and very small correlations (almost .00) reported across studies for both negative affectivity and BIS scales. Furthermore, there were significant correlations for BAS variables or positive affectivity reported within studies reported very low correlations for negative affectivity or BIS scales. Sample age was constant across the cluster and the sample composition with respect to gender was distributed across the effects. It is likely that the variance in this cluster is due to a variable or set of variables that escaped description in the research reports, and is, therefore, beyond the scope of the present study. Overall, however, the results of the present study have implications for theory and research.

Theoretical Implications

On one hand, the findings of the present study indicate considerable promise for symmetry of PFC functioning as a neurobiological mechanism that contributes to the regulation of social interaction behavior, a finding that addresses an initial impetus for the present study. On the other hand, the pattern of findings ran counter to the theoretical expectations based on Beatty's (2005) mediated effects model.

First, with respect to symmetry and regulation, the average correlation for the communication constructs was not large but it was respectable and the correlations within the cluster were in the theoretically expected direction. Recall, also, that the correlations were not corrected for the attenuating effects of measurement error associated with asymmetry scores. Although an insufficient number of studies reported reliability data for asymmetry, two studies within the cluster did supply alpha reliability coefficients based on multiple within-session samples of electrical activity (Blackhart & Kline, 2005; Fox et al., 1995). Completing the correction for attenuation for the correlations associated with these studies shows that the relationship between interpersonal defensiveness and asymmetry is improved from .28 to .40 (Blackhart & Kline) and the correlation between asymmetry and social competence improves from -.51 to -.61. It is uncertain what the mean correlation for the communication-related constructs and asymmetry might be if all the effects could have been corrected for measurement error associated with asymmetry. The problem underscores the need for complete reporting of information, such as reliability estimates of all variables, so that the relationships among constructs, not merely measures, can be explored.

It is noteworthy that accounting for the variance in correlations for the communication and temperament clusters did not required correcting the correlations for attenuation due to measurement error in asymmetry. Although the estimate of the mean correlation for each cluster was lower than it would have been had the corrections been made, the variance among the studies was solely due to sampling error. While it is clear from the reliabilities reported that there is variation in the internal consistency of asymmetry

measurement, the variation is not large enough to result in statistically significant variation across studies.

Second, the type of function (communication versus temperament/personality) emerged as a moderator, but in the opposite direction predicted by Beatty (2005). The mean correlation for communication constructs is too large relative to the correlation for temperament/personality cluster. This difference could not be due to correcting the correlations for attenuation because the uncorrected correlation for communication ($r = .371$) is also larger than the uncorrected correlation for the personality/temperament cluster ($r = .304$). In fact, the degree of difference between the two correlations is almost identical to the difference between the corrected correlations. Certainly, no single study- even a meta-analysis- is definitive. However, the results of the present study are at odds with the mediated effects model. The degree to which the findings of the present study can be taken as disconfirming evidence regarding Beatty's mediated effects model must be discussed within both theoretical and methodological contexts. At the theoretical level, Beatty arranged the model in accordance with ever-evolving propositions about communication but it is critical to note that Beatty presented no empirical evidence for the temporal ordering of the variables. However, the results of the present study are data-based. Moreover, the average effect sizes for the personality/temperament and communication clusters were based on multiple studies. Therefore, the empirical evidence appears to clash with theoretical speculation.

In general, when empirical evidence conflicts with a speculative model, the model is modified to reflect the empirical evidence. From a methodological standpoint, however, it is possible that the results of the present meta-analysis reflect systematic differences in

the reliability of asymmetry scores. Given that the reliabilities for asymmetry were not reported in the vast number of studies examined and that the range of reliability coefficients was quite wide for the few reported reliability coefficients, it is always possible that the personality cluster and the communication cluster differed as a function of the reliability of asymmetry. If, for some reason, the average reliability for asymmetry was at the low end for the personality/temperament cluster but at the high end for the communication cluster corrected average correlations might be in the correct direction. Unfortunately, the researchers did not report reliabilities, nor did they report the essential data needed for estimating reliability. Reliability, in the form of internal consistency, is a function of the number data points (e.g., the number of items on a self-report questionnaire or the number of measurements of brain activity made at baseline when studying asymmetry) and the correlation among those data points. The higher the average correlation among data points and the greater the number of data points, the higher the reliability. Not only did the researchers generally fail to report reliability coefficients, but they also failed to report the number of data points upon which asymmetry was based. Therefore, it is impossible to determine the degree to which, if any, the average correlations for the clusters of studies reflect systematic measurement error in the measurement of asymmetry. This has implications for future research, which will be discussed below.

Implications for Research

The results have implications for research and research methodology. First, the previous discussion regarding failure to report adequate information about the reliability of asymmetry scores raises the need for future research. Although theoretical models can

be evaluated by piecing together results from meta-analyses (Hunter & Schmidt, 2004, pp. 22-23), doing so requires adequate understanding of the important factors that influence effects in the set of studies examined. It seems obvious from the discussion regarding reliability of asymmetry that a better route to testing Beatty's (2005) mediated effects model would be to collect asymmetry data, personality/temperament data, and communication data on one sample. Then, correlations among the variables can be calculated and corrected for attenuation. Because personality/temperament is multi-dimensional (e.g., extraversion, neuroticism, psychoticism) and a large set of potential communication variables exist (e.g., communication apprehension, verbal aggressiveness, communication competence, assertiveness, etc.), there is potential for a great number of studies that should be conducted.

A second line of research that might be worthwhile concerns the determinates of asymmetry in the anterior cortex. If the degree of symmetry in the anterior cortices strongly contributes to the ability to self-regulate emotions and behaviors, the question arises as to the origin of asymmetry. Specifically, to what degree is asymmetry hard-wired? Some interesting research indicates that prenatal hormonal exposure influences the organization of the brain, especially lateralization (Grimshaw, Bryden, Raggatt, 1995; Witelson & Nowakowski 1991). The correlations between particular hormonal exposure and right versus left side dominance are in the moderate range. Importantly, these correlations have not been corrected for attenuation. Beatty, McCroskey, and Pence (in press) have suggested that prenatal hormone exposure is an important variable in the development of personality traits. It may be, however, that asymmetry mediates the relationship between prenatal hormonal exposure and subsequent communicator traits. A

line of research designed to integrate prenatal experience, various neurobiological functions (such as PFC asymmetry), temperament factors and communication variables would provide a greater sense of understanding than afforded by current biological models of communication.

Some methodological issues became apparent while reviewing studies. First, a great number of studies had to be deleted because of insufficient statistical reporting. The meta-analysis conducted in the present study would have included twice the data had adequate reporting been done. Moreover, the ways in which the findings might have been different had reliabilities been reported for all variables have already been discussed. Hunter and Schmidt (2004) emphasized the relation between meta-analysis work and theory buildings. They draw attention to how correlations derived from meta-analysis can be inserted as coefficients in causal models. However, this type of application required estimates of measurement error and a large body of research. Had the researchers conducting asymmetry work whose studies were exclude been guided by a unifying theoretical model, they might have reported reliabilities for all measures and others would have computed asymmetry indices rather than merely correlating variables with either the left or right cortex. Had these procedures been followed, the present meta-analysis could have been conducted on the type of sample size needed to build theoretical models. However, as mentioned, the most appropriate course of action, given the reporting and treatment of the data in the literature in general, it is necessary to conduct several studies to adequately test the mediated effects model proposed by Beatty (2005).

Second, the effects in some studies were artificially inflated by poor methodological choices. For instance, at first glance, the association of shyness and asymmetry appears stronger than it truly is (Schmidt, 1999; Schmidt & Fox, 1994) due to the dichotomization of shyness. Such practices exaggerate the potential of an explanatory variable.

Researchers should be encouraged to treat the data appropriately when analyzing it.

Otherwise, the results will be misleading, requiring others to perform corrections before interpreting them.

Conclusion

The results of the present study clearly point to the role of symmetry in the PFC as at least part of the neurobiological mechanism that permits regulation of behavior.

According to the communibiological paradigm, individual differences in behavior and traits are traceable to individual differences in neurobiological functioning. The meta-analysis reported in the present study seems to indicate a fairly robust association between individual differences in asymmetrical activity in the prefrontal cortex.

Although these findings disconfirm elements of the mediated effects model derived from communibiological assumptions, more well-designed studies are needed to test the model. It is hoped that the present investigation contributes in some way to that effort.

References

* denotes a study included in the meta-analysis

- Allen, J. B., Coan, J. A., & Nazarian, M. (2004). Issues and assumptions on the road from raw signals to metrics of frontal EEG asymmetry in emotion. *Biological Psychology*, *67*, 183-218.
- Allen, J. B., & Kline, J. P. (2004). Frontal EEG asymmetry, emotion, and psychopathology: the first, and the next 25 years. *Biological Psychology*, *67*, 1-5.
- Allen, J. B., Urry, H. L., Hitt, S. K., & Coan, J. A. (2004). The stability of resting frontal electroencephalographic asymmetry in depression. *Psychophysiology*, *41*, 269-280.
- Amodio, D. M., Shah, J. Y., Sigelman, J., Brazy, P. C., & Harmon-Jones, E. (2004). Implicit regulatory focus associated with asymmetrical frontal cortical activity. *Journal of Experimental Social Psychology*, *40*, 225-232.
- Anokhin, A. P., Heath, A. C., & Myers, E. (2006). Genetic and environmental influences on frontal EEG asymmetry: A twin study. *Biological Psychology*, *71*, 289-295.
- Bachara, A., Tranel, D., Damasio, H., & Damasio, A.R. (1996). Failure to respond automatically to anticipated future outcomes following damage to prefrontal cortex. *Cognition*, *50*, 7-15.
- Baehr, E., Rosenfeld, J. P., Baehr, R., & Earnest, C. (1998). Comparison of two EEG asymmetry indices in depressed patients vs. normal controls. *International Journal of Psychophysiology*, *31*, 89-92.
- Baron-Cohen, S. (2000). The cognitive neuroscience of autism: Evolutionary approaches. In M. S. Gazzaniga (Ed.), *The new cognitive neurosciences* (2nd ed., pp. 845-866). Cambridge, MA: MIT Press.

- Beatty, M.J. (2002). Do we know a vector from a scalar?: When r (not r square) is the appropriate measure of effect. *Human Communication Research*, 28, 605-611.
- Beatty, M. J., (2005). Fallacies in the textual analyses of the communibiological literature. *Communication Theory*, 15, 456-467.
- Beatty, M. J., & Heisel, A.D. (2007). Spectrum analysis of cortical activity during verbal planning: Physical evidence for the formation of social interaction routines. *Human Communication Research*. 33, 48-63.
- Beatty, M.J., & Heisel, A.D., Hall, A.E., Levine, T.R., & La France, B.H. (2002). What can we learn from the study of twins about genetic and environmental influences on interpersonal affiliation, aggressiveness, and social anxiety?: A meta-analytic study. *Communication Monographs*, 69, 1-18.
- Beatty, M. J., & Lewis, R. J. (in press) The cognitive neuroscience perspective. In M. J. Beatty, J. C. McCroskey, & K. Floyd (Eds.), *Biological dimensions of communication*. Cresskill, NJ: Hampton Press.
- Beatty, M.J., & Marshall, L.A., & Rudd, J. E. (2001). A twins study of communicative adaptability: Heritability of individual differences. *Quarterly Journal of Speech*, 87, 366-377.
- Beatty, M. J., & McCroskey, J. C. (1997). It's in our nature: Verbal aggressiveness as temperamental expression. *Communication Quarterly*, 45, 445-460.
- Beatty, M. J., & McCroskey, J. C. (1998). Interpersonal Communication as Temperamental Expression: A Communibiological Paradigm. In J. C. McCroskey, J. A. Daly, M. M. Martin, & M. J. Beatty (Eds.), *Communication and*

Personality: Trait Perspectives (p. 41-67). Cresskill, New Jersey: Hampton Press, Inc.

Beatty, M. J., & McCroskey, J. C. (1998). Interpersonal Communication as Temperamental Expression: A Communibiological Paradigm. In J. C. McCroskey, J. A. Daly, M. M. Martin, & M. J. Beatty (Eds.), *Communication and Personality: Trait Perspectives* (p. 41-67). Cresskill, New Jersey: Hampton Press, Inc.

Beatty, M. J., McCroskey, J. C., & Heisel, A. D. (1998). Communication apprehension as temperamental expression: A communibiological paradigm. *Communication Monographs*, 65, 197-219.

Beatty, M. J., McCroskey, J. C., & Pence, M. E. (in press). Communibiological paradigm. In M. J. Beatty, J. C. McCroskey, & K. Floyd (Eds.), *Biological dimensions of communication*. Cresskill, NJ: Hampton Press.

Beatty, M. J., McCroskey, J. C., & Valencic, K. M. (2001). *The Biology of Communication*. Cresskill, New Jersey: Hampton Press, Inc.

*Blackhart, G. C., & Kline, J. P. (2005). Individual differences in anterior EEG asymmetry between high and low defensive individuals during a rumination/distraction task. *Personality and Individual Differences*, 39, 427-437.

Blackhart, G. C., Minnix, J. A., & Kline, J. P. (2006). Can EEG asymmetry patterns predict future development of anxiety and depression? A preliminary study. *Biological Psychology*, 72, 46-50.

- Bruch, M. A., Gorsky, J. M., Collins, T. M., & Berger, P. A. (1989). Shyness and sociability reexamined: A multicomponent analysis. *Journal of Personality and Social Psychology, 57*, 904-915.
- Bruder, G. E., Stewart, J. W., Tenke, C. E., McGrath, P. J., Leite, P., Bhattacharya, N., & Quitkin, F. M. (2001). Electroencephalographic and perceptual asymmetry differences between responders and nonresponders to an SSRI antidepressant. *Society of Biological Psychiatry, 49*, 416-425.
- Buckner, R. L. (2000). Neuroimaging of memory. In M. Gazzaniga (Ed.), *The new cognitive neurosciences* (2nd ed., pp. 817-828). Cambridge, MA: MIT Press.
- Buss, K. A., Malmstadt Schumacher, J. R., Dolski, I., Kalin, N. H., Goldsmith, H. H., & Davidson, R. J. (2003). Right frontal brain activity, cortisol, and withdrawal behavior in 6-month-old infants. *Behavioral Neuroscience, 117*, 11-20.
- Cacioppo, J. T. (2004). Feelings and emotions: Roles for electrophysiological markers. *Biological Psychology, 67*, 235-243.
- Cappella, J. N. (1991). The biological origins of automated patterns of human interaction. *Communication Theory, 1*, 4-35.
- Carver, C. S., & 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.
- Cheek, J. M., & Buss, A. H. (1981). Shyness and sociability. *Journal of Personality and Social Psychology, 41*, 330-339.
- *Coan, J. A., & Allen, J. A. (2003). Frontal EEG asymmetry and the behavioral activation and inhibition systems. *Psychophysiology, 40*, 106-114.

- Coan, J. A., & Allen, J. B. (2004). Frontal EEG asymmetry as a moderator and mediator of emotion. *Biological Psychology*, *67*, 7-49.
- Coan, J. A., Allen, J. B., & Harmon-Jones, E. (2001). Voluntary facial expression and hemispheric asymmetry over the frontal cortex. *Psychophysiology*, *38*, 912-925.
- Coan, J. A., Allen, J. B., & McKnight, P. E. (2006). A capability model of individual differences in frontal EEG asymmetry. *Biological Psychology*, *72*, 198-207.
- Cohen, J. (1990). Things I learned (so far). *American Psychologist*, *45*, 1304-1312.
- Collet, L., & Duclaux, R. (1987). Hemispheric lateralization of emotions: Absence of electrophysiological arguments. *Physiology and Behavior*, *40*, 215-220.
- D'Esposito, M., Detre, J. A., Alsop, D. C., Shin, R. K., Atlas, S., & Grossman, M. (1995). The neural basis of the central executive system of working memory. *Nature*, *378*, 279-281.
- Damasio, A. R. (1995). Toward a neurobiology of emotion and feeling: Operational concepts and hypotheses. *Neuroscientist*, *1*, 19-25.
- Davidson, R. J., & Fox, N. A. (1982). Asymmetrical brain activity discriminates between positive and negative affective stimuli in human infants. *Science*, *218*, 1235-1237.
- 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. (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. 361-387). Cambridge, MA: MIT Press.

- Davidson, R.J. (2000). The neuroscience of affective style. In M. S. Gazzaniga. (Ed.), *The new cognitive neurosciences* (2nd ed., pp. 1149-1159). Cambridge, MA: MIT Press.
- Davidson, R. J. (2004). EEG asymmetry and mindfulness meditation: Response. *Psychosomatic Medicine*, *66*, 147-152.
- Davidson, R. J. (2004). What does the prefrontal cortex "do" in affect: perspectives on frontal EEG asymmetry research. *Biological Psychology*, *67*, 219-233.
- Davidson, R. J., Chapman, J. P., Chapman, L. J., & Henriques, J. B. (1990). Asymmetrical brain electrical activity discriminates between psychometrically-matched verbal and spatial cognitive tasks. *Psychophysiology*, *27*, 528-543.
- 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., Marshall, J. R., Tomarken, A. J., & Henriques, J. B. (2000). While a phobic waits: Regional brain electrical and autonomic activity and social phobics during anticipation of public speaking. *Biological Psychiatry*, *47*, 85-95.
- Davidson, R. J., Schaffer, C. E., & Saron, C. (1985). Effects of lateralized presentations of faces on self-reports of emotion and EEG asymmetry in depressed and non-depressed subjects. *Psychophysiology*, *22*, 353-364.
- Dawson, G., Frey, K., Panagiotides, H., Osterling, J., & Hessel, D. (1997). Infants of depressed mothers exhibit atypical frontal brain activity: A replication and extension of previous findings. *Journal of Child Psychology and Psychiatry*, *38*, 179-186.

Dawson, G., Frey, K., Panagiotides, H., Yamada, E., Hessler, D., & Osterling, J. (1999).

Infants of depressed mothers exhibit atypical frontal electrical brain activity during interactions with mother and with a familiar, nondepressed adult. *Child Development, 70*, 1058-1066.

Dawson, G., Frey, K., Self, J., Panagiotides, H., Hessler, D., Yamada, E., & Rinaldi, J.

(1999). Frontal brain electrical activity in infants of depressed and nondepressed mothers: Relation to variations in infant behavior. *Development and Psychopathology, 11*, 589-605.

Dawson, G., Klinger, L. G., Panagiotides, H., Hill, D., & Spieker, S. (1992). Frontal lobe

activity and affective behavior of infants of mothers with depressive symptoms. *Child Development, 63*, 725-737.

Dawson, G., Panagiotides, H., Klinger, L. G., & Spieker, S. (1997). Infants of depressed

and nondepressed mothers exhibit differences in frontal brain electrical activity during the expression of negative emotions. *Developmental Psychology, 33*, 650-656.

Debener, S., Beauducel, A., Nessler, D., Brocke, B., Heilemann, H., & Kayser, J. (2000).

Is resting anterior EEG alpha asymmetry a trait marker for depression? Findings for healthy adults and clinically depressed patients. *Neuropsychobiology, 41*, 31-37.

Ekman, P., Davidson, R. J., & Friesen, W. V. (1990). The Duchenne Smile: Emotional

expression and brain physiology II. *Journal of Personality and Social Psychology, 58*, 342-353.

- Eysenck, S. G., Eysenck, H. J., & Barrett, P. (1985). A revised version of the psychoticism scale. *Personality and Individual Differences*, 6, 21-29.
- Field, T., Fox, N. A., Pickens, J., & Nawrocki, T. (1995). Relative right frontal EEG activation in 3- to 6- month-old infants of "depressed" mothers. *Developmental Psychology*, 31, 358-363.
- Field, T., Pickens, J., Prodromidis, M., Malphurs, J., Fox, N., Bendell, D., Yando, R., Schanberg, S., & Kuhn, C. (2000). Targeting adolescent mothers with depressive symptoms for early intervention. *Adolescence*, 35, 381-414.
- *Fink, A. (2005). Event-related desynchronization in the EEG during emotional and cognitive information processing: Differential effects of extraversion. *Biological Psychology*, 70, 152-160.
- Forbes, E. E., Fox, N. A., Cohn, J. F., Galles, S. F., & Kovacs, M. (2006). Children's affect regulation during a disappointment: Psychophysiological responses and relation to parent history of depression. *Biological Psychology*, 71, 264-277.
- Fox, N.A. (1994). Dynamic cerebral processes underlying emotion regulation. N.A. Fox (Ed.), *The development of emotion regulation: Behavioral and biological considerations. Monographs of the Society for Research in Child Development*, 59, 152-166.
- Fox, N. A., & Davidson, R. J. (1986). Taste-elicited changes in facial signs of emotion and the asymmetry of brain electrical activity in human newborns. *Neuropsychologia*, 24, 417-422.

- Fox, N. A., & Davidson, R. J. (1987). Electroencephalogram asymmetry in response to the approach of a stranger and maternal separation in 10-month-old infants. *Developmental Psychology, 23*, 233-240.
- 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.
- *Fox, N. A., Coplan, R. J., Rubin, K. H., Porges, S. W., Calkins, S. D., Long, J. M., Marshall, T. R., & Stewart, S. (1995). Frontal activation asymmetry and social competence at four years of age. *Child Development, 66*, 1770-1784.
- Fuster, J. (1997). *The prefrontal cortex: Anatomy, physiology, and neuropsychology of the frontal lobe*. (3rd Ed.). Philadelphia: Lippincott-Raven.
- Gilbert, D. G., McClernon, F. J., Rabinovich, N. E., Dibb, W. D., Plath, L. C., Hiyane, S., Jensen, R. A., Meliska, C. J., Estes, S. L., & Gehlbach, B. A. (1999). EEG, physiology, and task-related mood fail to resolve across 31 days of smoking abstinence: Relations to depressive traits, nicotine exposure, and dependence. *Experimental and Clinical Psychopharmacology, 7*, 427-443.
- Gilbert, D. G., Meliska, C. J., Welser, R., & Estes, S. L. (1994). Depression, personality, and gender influence EEG, cortisol, beta-endorphin, heart rate, and subjective responses to smoking multiple cigarettes. *Personality and Individual Differences, 16*, 247-264.
- Glass, A., & Riding, R. J. (1999). EEG differences and cognitive style. *Biological Psychology, 51*, 23-41.

- Gotlib, I. H., Ranganath, C., & Rosenfeld, J. P. (1998). Frontal EEG alpha asymmetry, depression, and cognitive functioning. *Cognition and Emotion, 12*, 449-478.
- Gray, J. A. (1991). The neuropsychology of temperament. In J. Strelau & A. Angleitner (Eds.), *Explorations in temperament* (pp. 105-128). New York: Plenum.
- Grimshaw, G.M., Bryden, M.P., & Raggatt, P. (1995). Relations between prenatal testosterone and cerebral lateralization in children. *Neuropsychology, 9*, 68-79.
- Guilford, J.P. (1956). *Fundamental statistics in psychology and education*. New York: McGraw-Hill.
- Hagemann, D. (2004). Individual differences in anterior EEG asymmetry: Methodological problems and solutions. *Biological Psychology, 67*, 157-182.
- *Hagemann, D., Hewig, J., Naumann, E., Seifert, J., & Bartussek, D. (2005). Resting brain asymmetry and affective reactivity: Aggregated data support the right-hemisphere hypothesis. *Journal of Individual Differences, 26*, 139-154.
- 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.
- *Hagemann, D., Naumann, E., Becker, G., Maier, S., & Bartussek, D. (1998). Frontal brain asymmetry and affective style: A conceptual replication. *Psychophysiology, 35*, 372-388.
- Harmon-Jones, E., & Allen, J. B. (1997). Behavioral activation sensitivity and resting frontal EEG asymmetry: Covariation of putative indicators related to risk for mood disorders. *Journal of Abnormal Psychology, 106*, 159-163.

- *Harmon-Jones, E., & Allen, J. B. (1998). Anger and frontal brain activity: EEG asymmetry consistent with approach motivation despite negative affective valence. *Journal of Personality and Social Psychology, 74*, 1310-1316.
- *Harmon-Jones, E., & Siegelman, J. (2001). State anger and prefrontal brain activity: Evidence that insult-related relative left-prefrontal activation is associated with experienced anger and aggression. *Journal of Personality and Social Psychology, 80*, 797-803.
- Harmon-Jones, E. (2004). Contributions from research on anger and cognitive dissonance to understanding the motivational functions of asymmetrical frontal brain activity. *Biological Psychology, 67*, 51-76.
- Harmon-Jones, E., Abramson, L. Y., Sigelman, J., Bohlig, A., Hogan, M. E., & Harmon-Jones, C. (2002). Proneness to hypomania/mania symptoms or depression symptoms and asymmetrical frontal cortical responses to an anger-evoking event. *Journal of Personality and Social Psychology, 82*, 610-618.
- Hedges, L.V. & Vevea, J.L. (1998). Fixed- and random-effects models in meta-analysis. *Psychological Methods, 3*, 486-504.
- Heisel, A. D., & Beatty, M. J. (2006). Are cognitive representations of friends' request refusals implemented in the orbitofrontal and dorsolateral prefrontal cortices?: A cognitive neuroscience approach to "theory of mind" in relationships. *Journal of Social and Personal Relationships, 23*, 249-265.
- Heisel, A. D., La France, B. H., & Beatty, M. J. (2003). Self-reported extraversion, neuroticism, and psychoticism as predictors of peer related verbal aggressiveness and affinity-seeking competence. *Communication Monographs, 70*, 1-15.

- *Henderson, H. A., Fox, N. A., & Rubin, K. H. (2001). Temperamental contributions to social behavior: The moderating roles of frontal EEG asymmetry and gender. *Journal of the American Academy of Child Adolescent Psychiatry, 40*, 68-74.
- Henderson, H. A., Marshall, P. J., Fox, N. A., & Rubin, K. H. (2004). Psychophysiological and behavioral evidence for varying forms and functions of nonsocial behavior in preschoolers. *Child Development, 75*, 251-263.
- 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.
- *Hewig, J., Hagemann, D., Seifert, J., Naumann, E., & Bartussek, D. (2006). The relation of cortical activity and BIS/BAS on the trait level. *Biological Psychology, 71*, 42-53.
- Hunter, J. E., & Schmidt, F. L. (2004). *Methods of meta-analysis: Correcting error and bias in research findings* (2nd ed.). Thousand Oaks, CA: Sage.
- Jacobs, G. D., & Snyder, D. (1996). Frontal brain asymmetry predicts affective style in men. *Behavioral Neuroscience, 110*, 3-6.
- Jensen-Campbell, L. A., Knack, J. M., Waldrip, A. M., & Campbell, S. D. (2007). Do Big Five personality traits associated with self-control influence the regulation of anger and aggression? *Journal of Research in Personality, 41*, 403-424.

- Jones, N. A., & Fox, N. A. (1992). Electroencephalogram asymmetry during emotionally evocative films and its relation to positive and negative affectivity. *Brain and Cognition, 20*, 280-299.
- Jones, N. A., Field, T., Davalos, M., & Pickens, J. (1997). EEG stability in infants/children of depressed mothers. *Child Psychiatry and Human Development, 28*, 59-70.
- Jones, N. A., Field, T., Fox, N. A., Davalos, M., Lundy, B., & Hart, S. (1998). Newborns of mothers with depressive symptoms are physiologically less developed. *Infant Behavior and Development, 21*, 537-541.
- Jones, N. A., Field, T., Fox, N. A., Lundy, B., & Davalos, M. (1997). EEG activation in 1-month-old infants of depressed mothers. *Development and Psychopathology, 9*, 491-505.
- Kentgen, L. M., Tenke, C. E., Pine, D. S., Fong, R., Klein, R. G., & Bruder, G. E. (2000). Electroencephalographic asymmetries in adolescents with major depression: Influence of comorbidity with anxiety disorders. *Journal of Abnormal Psychology, 109*, 797-802.
- *Kline, J. P., Allen, J. B., & Schwartz, G. E. (1998). Is left frontal brain activation in defensiveness gender specific? *Journal of Abnormal Psychology, 107*, 149-153.
- *Kline, J. P., Knapp-Kline, K., Schwartz, G. E., & Russek, L. G. (2001). Anterior asymmetry, defensiveness, and perceptions of parental caring. *Personality and Individual Differences, 31*, 1135-1145.

- La France, B. H., Heisel, A. D., & Beatty, M. J. (2004). Is there empirical for a nonverbal profile of extraversion?: A meta-analysis and critique of the literature. *Communication Monographs, 71*, 38-48.
- Lewis, R. S., Weekes, N. Y., & Wang, T. H. (2007). The effect of a naturalistic stressor on frontal EEG asymmetry, stress, and health. *Biological Psychology, 75*, 239-247.
- MaCallum, R. C., Zhang, S., Preacher, K. J., & Roeker, D. D. (2002). One the practice of dichotomization of quantitative variables. *Psychological Methods, 7*, 19-40.
- McCroskey, J. C. (1978). The validity of the PRCA as an index of oral communication apprehension. *Communication Monographs, 45*, 193-203.
- McManis, M. H., Kagan, J., Snidman, N. C., & Woodward, S. A. (2002). EEG asymmetry, power, and temperament in children. *Developmental Psychobiology, 41*, 169-177.
- Miller, A., & Tomarken, A. J. (2001). Task-dependent changes in frontal brain asymmetry: Effects of incentive cues, outcome expectancies, and motor responses. *Psychophysiology, 38*, 500-511.
- Miller, A., Fox, N. A., Cohn, J. F., Forbes, E. E., Sherrill, J. T., & Kovacs, M. (2002). Regional patterns of brain activity in adults with a history of childhood-onset depression: Gender differences and clinical variability. *American Journal of Psychiatry, 159*, 934-940.
- *Minnix, J. A., & Kline, J. P. (2004). Neuroticism predicts resting frontal EEG asymmetry variability. *Personality and Individual Differences, 36*, 823-832.

- Minnix, J. A., Kline, J. P., Blackhart, G. C., Pettit, J. W., Perez, M., & Joiner, T. E. (2004). Relative left-frontal activity is associated with increased depression in high reassurance-seekers. *Biological Psychology, 67*, 145-155.
- Molle, M., Marshall, L., Wolf, B., Fehm, H. L., & Born, J. (1999). EEG complexity and performance measures of creative thinking. *Psychophysiology, 36*, 95-104.
- 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.
- Papousek, I., & Schuster, G. (2001). Associations between EEG asymmetries and electrodermal lability in low vs. high depressive and anxious normal individuals. *International Journal of Psychophysiology, 41*, 105-117.
- Papousek, I., & Schuster, G. (2002). Covariations of EEG asymmetries and emotional states indicate that activity at frontopolar locations is particularly affected by state factors. *Psychophysiology, 39*, 350-360.
- Pinker, S. (1997). *How the mind works*. New York: W. W. Norton.
- Pinker, S. (2002). *The blank slate: The modern denial of human nature*. New York: Viking.
- Pizzagalli, D. A., Sherwood, R. J., Henriques, J. B., & Davidson, R. J. (2005). Frontal brain asymmetry and reward responsiveness: A source-localization study. *Psychological Science, 16*, 805-813.
- Popper, K. R., & Eccles, J. C. (1977). *The self and its brain*. New York: Springer.

- Reid, S. A., Duke, L. M., & Allen, J. B. (1998). Resting frontal electroencephalographic asymmetry in depression: Inconsistencies suggest the need to identify mediating factors. *Psychophysiology*, *35*, 389-404.
- Reiman, E. M., Fusselman, M. J., Fox, P. T., & Raichle, M. E. (1989). Neuroanatomical correlates of anticipatory anxiety. *Science*, *243*, 1071-1074.
- Rosenfeld, J. P., Baehr, E., Baehr, R., Gotlib, I. H., & Ranganath, C. (1996). Preliminary evidence that daily changes in frontal alpha asymmetry correlate with changes in affect in therapy sessions. *International Journal of Psychophysiology*, *23*, 137-141.
- Rosenfeld, R. (1991). *Meta-analytic procedures for social research (rev. ed.)*. Newbury Park: Sage.
- *Sabbagh, M. A., & Flynn, J. (2006). Mid-frontal EEG alpha asymmetries predict individual differences in one aspect of theory of mind: Mental state decoding. *Social Neuroscience*, *1*, 299-308.
- Sackett, H.R., Harris, M.M., & Orr, J.M. (1986). On seeking moderator variables in the meta-analysis of correlational data: A Monte Carlo investigation of statistical power and resistance to Type 1 error. *Journal of Applied Psychology*, *71*, 302-310.
- *Schmidt, L. A., & Fox, N. A. (1994). Patterns of cortical electrophysiology and autonomic activity in adults' shyness and sociability. *Biological Psychology*, *38*, 183-198.
- *Schmidt, L. A. (1999). Frontal brain electrical activity in shyness and sociability. *Psychological Science*, *10*, 316-320.

- Smit, D. A., Posthuma, D., Boomsma, D. I., & De Geus, E. J. (2007). The relation between frontal EEG asymmetry and the risk for anxiety and depression. *Biological Psychology, 74*, 26-33.
- *Sobatka, S. S., Davidson, R. J., & Senulis, J. A. (1992). Anterior brain electrical asymmetries in response to reward and punishment. *Electroencephalography and clinical Neurophysiology, 83*, 236-247.
- Stough, C., Donaldson, C., Scarlata, B., & Ciorciari, J. (2001). Psychophysiological correlates of the NEO PI-R openness, agreeableness, and conscientiousness: Preliminary results. *International Journal of Psychophysiology, 41*, 87-91.
- Stroganova, T. A., Nygren, G., Tsetlin, M. M., Posikera, I. N., Gillberg, C., Elam, M., & Orekhova, E. V. (2007). Abnormal EEG lateralization in boys with autism. *Clinical Neurophysiology, 118*, 1842-1854.
- Strelau, J. (1994). The concept of arousal and arousability as used in temperament studies. In J. E. Bates & T. D. Wachs (Eds.), *Temperament: Individual differences at the interface of biology and behavior* (pp. 117-141). Washington, D.C. American Psychological Association.
- Stuss, D.T. & Benson, D.F. (1986). *The frontal lobes*. New York (Raven Press).
- *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.
- Tanji, J., & Hoshi, E. (2001). Behavioral planning in the prefrontal cortex. *Current Opinion in Neurobiology, 11*, 164-170.

- Thibodeau, R., Jorgensen, R. S., & Kim, S. (2006). Depression, anxiety, and resting frontal EEG asymmetry: A meta-analytic review. *Journal of Abnormal Psychology, 115*, 715-729.
- *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 emotions. *Journal of Personality and Social Psychology, 62*, 676-687.
- Tooby, J., & Cosmides, L. (2000). Toward mapping a functional organization of mind and brain. In M. S. Gazzaniga (Ed.), *The new cognitive neurosciences* (2nd ed., pp. 1167-1178). Cambridge, MA: MIT Press.
- Travis, F., & Arenander, A. (2004). EEG asymmetry and mindfulness meditation. *Psychosomatic Medicine, 66*, 147-152.
- Turkheimer, E., & Waldron, M. (2000). Nonshared environment: A theoretical, methodological, and quantitative review. *Psychological Bulletin, 126*, 78-108.
- Urry, H. L., Nitschke, J. B., Dolski, I., Jackson, D. C., Dalton, K. M., Mueller, C. J., Rosenkranz, M. A., Ryff, C. D., Singer, B. H., & Davidson, R. J. (2004). Making a life worth living: Neural correlates of well-being. *Psychological Science, 15*, 367-372.

- Valencic, K. M., Beatty, M. J., Rudd, J. E., Dobos, J. A., & Heisel, A. D. (1998). An empirical test of a communibiological model of trait verbal aggressiveness. *Communication Quarterly, 46*, 327-341.
- Waldstein, S. R., Kop, W. J., Schmidt, L. A., Haufler, A. J., Krantz, D. S., & Fox, N. A. (2000). Frontal electrocortical and cardiovascular reactivity during happiness and anger. *Biological Psychology, 55*, 3-23.
- *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.
- Wiedemann, 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.
- Wilson, E. O. (1998). *Consilience: The unity of knowledge*. New York: Vintage.
- Witelson, S.F., & Nowakowski, R.S. (1991). Left out axons make men right: A hypothesis for the origin of handedness and functional asymmetry. *Neuropsychologia, 28*, 327-333.

APPENDIX

Table 4

*Studies Excluded from Meta-Analysis by Criterion**Studies that contained no data or inadequate reporting*

Coan, Allen, & Harmon-Jones (2001) (Reported significance but no test statistics)

Cole et al. (1996)

Coan, Allen, & McKnight (2006)

Studies of disordered samples

Allen, Urry, Hitt, & Coan (2004)

Baehr, Rosenfeld, Baehr, & Earnest (1998)

Blackhart, Minnix, & Kline (2006)

Bruder et al. (2001)

Davidson, Schaffer, & Saron (1985)

Dawson et al. (1992)

Dawson et al. (1997)

Dawson et al. (1999)

Dawson, Pangagiotides, Klinger, & Spieker (1997)

Debenor et al. (2000)

Field, Fox, Pickens, & Nawrocki (1998)

Field et al. (2000)

Forbes et al. (2006)

Gilbert et al. (1999)

Gilbert, Meliska, Welser, & Estes (1994)
Gotlib, Ranganath, & Rosenfeld (1998)
Harmon-Jones et al. (2002)
Harmon-Jones & Allen (1997)
Henriques & Davidson (1990)
Henriques & Davidson (1991)
Jones, Field, Davalos, & Pickens (1997)
Jones et al. (1997)
Jones et al. (1998)
Kentgen et al. (2000)
Lewis, Weeks, & Wang (2007)
Minnix et al. (2004)
Miller et al. (2002)
Nitschke et al. (1999)
Papousek & Shulter (2002)
Posthuma, Boomsma, & De Geus (2007)
Rosenfeld et al. (1996)
Stroganova et al. (2007)
Urry et al. (2004)
Wiedemann et al. (1999)

Studies that did not measure anterior PFC

Bruch et al. (1989)

Davidson, Chapman, Chapman, & Henriques (1990)

Davidson, Marshall, Tomarken, & Henriques (2000)

Pizzagalli et al. (2005)

Studies of induced rather than baseline asymmetry

Davidson (2004)

Davidson & Fox (1989)

Ekman et al. (1990)

Fox & Davidson (1987)

Fox & Davidson (1988)

Henderson, Marshall, Fox, & Rubin (2004)

Miller & Tomarken (2001)

Molle et al. (1999)

Tomarken, Davidson, & Henriques (1990)

Travis & Arenander (2004)

Waldstein et al. (2000)

Studies that did not employ an asymmetry index (i.e., correlated variable with either left or right region).

Amodis et al. (2004)

Buss, Schumacher, Dolski, Kalin, Goldsmith, & Davidson (2003)

Glass & Riding (1999)

Jacobs & Snyder (1996)

Jones & Fox (1992)

McManis, Kagan, Snidman, Woodward (2001)

Stough et al. (2001)