Handbook of Approach and Avoidance Motivation

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Effects of Early Experience on the Development of Cerebral Asymmetry and Approach–Withdrawal

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Cortical Asymmetry

3 Effects of Early Experience on the Development of Cerebral Asymmetry and Approach–Withdrawal

Nathan A. Fox and Bethany C. Reeb

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Over 25 years ago, a study conducted by Davidson and colleagues demonstrated that frontal electroencephalo-
geraphic (EEG) asymmetry was related to different expres-
sions of emotion (Davidson, Taylor, & Saron, 1979). Numerous studies followed in the examination of the role of frontal EEG asymmetry in relation to emotional valence (Coan & Allen, 2004; Harmon-Jones, 2003), motivation (Davidson, 1995, 2004; Davidson, Jackson, & Kalin, 2000a; Harmon-Jones, 2004) and affect disorders, such as depression and anxiety (Gotlib, Ranganath, & Rosenfeld, 1998; Heller & Nitschke, 1998; Thibodeau, Jorgensen, & Kim, 2006; Tomarken & Keener, 1998). Frontal EEG asymmetry has been viewed alternatively as a state-dependent measure of negative or positive emotion (Ahern & Schwartz, 1985; Coan, Allen, & Harmon-Jones, 2001; Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Gotlib et al., 1998; Heller, 1990), as an index of approach or withdrawal motivation (Davidson, 1995; Davidson et al., 2000a; Fox, 1991, 1994), and as a trait measure reflecting the disposition to either express certain types of emotion or respond in a motivationally biased manner (Fox, Henderson, Rubin, Calkins, & Schmidt, 2001; Schmidt & Fox, 1994; Tomarken, Davidson, Wheeler, & Doss, 1992a; Wheeler, Davidson, & Tomarken, 1993).
Compared to other theories of emotional valence, the approach–withdrawal theory of EEG frontal asymmetry as proposed by Davidson has been suggested to best explain results observed in both studies of state and trait emotional valence as well as studies of affective disorders (Coan & Allen, 2004; Davidson, 1998a, 2004; Harmon-Jones, 2003, 2004). According to the approach–withdrawal model, increased activation of the left frontal cortex is associated with increases in appetitive, approach-related behavior that are typically displayed in the context of moving toward a desired goal. The approach system includes such emotions as joy, interest, and even anger. In contrast, increased right frontal activation is related to increases in defensive, withdrawal-related behavior that is displayed in the context of moving away from or avoiding threatening or novel stimuli. The withdrawal system includes such emotions as fear and disgust. This chapter serves two purposes: (1) to review studies that support the approach–withdrawal theory of frontal asymmetric activation and (2) to discuss the role of early experience in the development of motivationally related asymmetry by reviewing both the human and animal literatures.

MEASURES OF FRONTAL CEREBRAL ACTIVATION

Evidence for cerebral lateralization of affective information in the frontal cortex was initially observed in patients with lesion to the right or left frontal cortex (Gainotti, 1972, 1989; Morris, Robinson, Raphael, & Hopwood, 1996; Robinson & Downhill, 1995; Robinson, Kubos, Starr, Rao, & Price, 1984; Sackeim et al., 1982). In these studies, it was demonstrated that left frontal lobe lesions resulted in depressive symptomatology, while in contrast, patients with right frontal damage were more likely to develop manic symptomatology. Additionally, the more frontal (Robinson et al., 1984) and the more focal the lesions (Morris et al., 1996), the greater the symptomatology. These results suggest that the left and right frontal regions are specialized for differential emotional processing, and therefore should lead to asymmetric activation in a normal population when different emotional states are evoked. EEG is an ideal tool to investigate such activation, because it is noninvasive and allows for excellent temporal resolution. Studies investigating asymmetric activation have primarily focused on differences in the alpha frequency band (8–13 Hz) between the left and right frontal sites. Alpha power is used because it has been shown to be inversely related to activation (Shagass, 1972). Therefore, lower alpha power reflects greater activation while greater alpha power reflects less activation.

FRONTAL ASYMMETRY AS A STATE OR TRAIT MEASURE OF APPROACH–WITHDRAWAL

Asymmetric frontal activation has been shown to change as a function of exposure to various stimuli believed to evoke the approach or withdrawal systems. In one study by Davidson and colleagues (1990), EEG was recorded while subjects viewed film clips that evoked either joy or disgust. Greater right frontal activation was found only during the periods in which subjects expressed the clear emotional expression of disgust rather than during the entire disgust-evoking film clip. In addition, greater left frontal activation was observed during segments in which subjects displayed a Duchenne’s smile (Ekman, Davidson, & Friesen, 1990), a smile typically associated with genuine happiness (Ekman & Friesen, 1982). Such asymmetries were not observed when alpha power was averaged over the entire film clip suggesting that actual “feeling” or presence of the subject’s affective state, as expressed by his or her facial expressions, is important in uncovering differences in frontal asymmetric activation. Similar frontal asymmetries were obtained in a study where subjects were asked to produce various facial expressions that reflect either approach or withdrawal emotional states (Coan & Allen, 2003b; Coan et al., 2001).

In another study, differences in frontal asymmetric activation were observed in the absence of facial expression during a task that manipulated reward and punishment (Sobotka, Davidson, & Senulis, 1992). During reward trials, subjects had to respond quickly to a particular target stimulus to receive a reward (monetary increase); the subject would not receive a reward if his or her response was not fast enough. During punishment trials, subjects would lose money if they were not able to respond quickly to the target stimulus or would not lose money if the response was fast enough. As predicted, greater right frontal activation was observed during punishment trials when compared to reward trials. Therefore, it was concluded that right-sided frontal activation is reflective of withdrawal-related emotions associated with aversion to punishment.

In the studies described above, increased left frontal activation associated with approach-related stimuli appears to be associated with positive emotional valence. However, this is not always the case, because studies conducted by Harmon-Jones have shown that anger, an emotion associated with approach but with a negative valence, is related to increased levels of left frontal asymmetry (for reviews see Harmon-Jones, 2003, 2004). To demonstrate that state measures of anger are related to left frontal activation, subjects were randomly assigned...
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INDIVIDUAL DIFFERENCES IN FRONTAL ACTIVATION

The research reviewed above compares approach and withdrawal-oriented emotional states by evoking various emotional states within the subjects. However, when the individual comes into the laboratory to have his or her EEG measured, he or she already varies in his or her own dispositional mood which, subsequently, should also influence baseline resting EEG measures. Therefore, frontal EEG asymmetry additionally has been investigated as a trait measure or tendency for individuals to respond in a motivationally biased manner (Hagemann, Naumann, Thayer, & Bartussek, 2002; Jackson et al., 2003; Sutton & Davidson, 1997; Tomarken, Davidson, & Henriquez, 1990; Tomarken et al., 1992a; Tomarken, Davidson, Wheeler, & Kinney, 1992b; Wheeler et al., 1993). The approach–withdrawal theory of frontal EEG asymmetry suggests that individuals with greater right activation will be more vulnerable to experiencing negative emotional states and stronger withdrawal/inhibitory tendencies, such as those experienced in anxiety disorders. In contrast, individuals with decreased left frontal activation will be more vulnerable to experiencing decreased positive emotional states and weaker approach tendencies (Davidson, 1995; Davidson, 1998a).

In an initial study investigating the relation between resting EEG asymmetry and reported global negative and positive affect during emotion-eliciting film clips, Tomarken and colleagues (1990) found that resting alpha power asymmetry observed in female subjects predicted their reported feelings of negative affect during the clips. The greater the negative affect reported, the greater the relative right frontal activation found during rest. These effects were observed independent of the subjects’ mood state reported during the recording of the baseline measures.

In another study conducted by the same group, it was found that only among subjects with high test–retest reliability in resting frontal EEG measured over a 3-week time period did frontal asymmetric activation predict subjective emotional responses while watching emotionally valenced film clips (Wheeler et al., 1993). Subjects who showed greater left frontal activation stability during rest reported greater positive affect after watching the positive film clip, whereas those who showed greater stability in right frontal activation reported greater negative affect after watching the negative film clip. This pattern of findings remained even when baseline mood was statistically controlled. Other studies have also reported measures of resting EEG asymmetry to be highly stable over time periods ranging from several weeks (Tomarken et al., 1992b; Wheeler et al.) to months (Hagemann et al., 2002). Together, these results suggest that stability in frontal EEG asymmetry reflects possible trait patterns of approach–withdrawal and that this asymmetry measure may be used as an index of an individual’s predispositional response to emotion-evoking stimuli. However, it should be noted that some studies find only moderate stability in EEG asymmetry (Vuga et al., 2006), but these results may be explained by the dual nature of resting asymmetry in that it has the potential to reflect both trait and state measures of the individual (Hagemann et al., 2002).

Additional studies have examined the relations between resting frontal EEG asymmetries and dispositional depressive moods, positive and negative affect, and anger. Using the Beck Depression Inventory (BDI) (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) to measure subjects’ depressive mood, Davidson’s group found that subjects with consistently high BDI scores had significantly less left frontal activation during baseline EEG collection compared to subjects who consistently scored low on the BDI (Schaffer, Davidson, & Saron, 1983). Using the Positive and Negative Affect Scale (PANAS) (Watson, Clark, & Tellegen, 1988) as a measure of dispositional mood, it was found that subjects who displayed greater stability as well as high levels of left frontal activation reported significantly greater dispositional positive affect and less negative affect when compared to those who displayed greater stability in right frontal activation (Tomarken et al., 1992a). In a study assessing trait anger, Harmon-Jones and Allen (1998) measured adolescent trait anger using the Buss and Perry Aggression questionnaire (1992). This trait anger measure was found
to be associated with greater left activation and decreased right activation.

In a number of studies, individual differences in the strength of the behavioral activation system (BAS) and behavioral inhibition system (BIS) and its relations to frontal EEG asymmetry have been investigated. Gray (1975, 1994) hypothesized that the BAS is responsible for guiding and executing behaviors that are involved in obtaining a desirable stimulus while the BIS is responsible for guiding and executing behaviors involved in the removal of or moving away from an undesirable stimulus. Therefore, the BAS may be reflective of a dispositional tendency for motivational approach while the BIS may be reflective of a dispositional tendency for motivational withdrawal. In a study conducted by Sutton and Davidson (1997), frontal EEG asymmetry measured during rest was assessed in its relation to measures of BAS and BIS strength using Carver and White’s (1994) BIS and BAS scales. Results revealed that subjects with greater left frontal activation had higher BAS scores while subjects with greater right frontal activation had higher BIS scores. In addition, subjects that displayed greater left frontal activation also displayed greater differences between BAS and BIS scores. Frontal asymmetry measures in this unselected population were not predictive of positive and negative affect using the PANAS (Sutton & Davidson, 1997) suggesting that measures of frontal asymmetry are more predictive of motivational tendencies as reflected by measures from the BAS/BIS scale rather than measures of positive and negative affect. Replications of these results have been reported for the relation between left frontal activation and the BAS scale (Coan & Allen, 2003a; Harmon-Jones & Allen, 1997; Hewig, Hagemann, Seifert, Naumann, & Bartussek, 2006), but the relation between the BIS scale and right frontal activation has been found to be inconsistent.

**Psychopathology and Frontal EEG Asymmetry**

The literature reviewed thus far has primarily focused on frontal EEG asymmetry and how it relates to either state or trait measures of approach and withdrawal emotion-related affect. There are some studies that suggest that frontal asymmetric activation may also be a useful predictive assessment tool for dispositional biases for affective disorders. In a study conducted by Henriques and Davidson (1991), it was found that, similar to subjects who scored high on the BDI, clinically depressed subjects showed significantly less left frontal activation, referred to as left frontal hypoactivation, compared to control subjects with no history of depression. This direction of activation is predicted by the approach–withdrawal theory because states of depression should be associated with deficits in the approach motivation system, and thus decreased activation in the left frontal cortex should be observed (Davidson, 1998b). In addition, left frontal hypoactivation has been suggested to serve as a state-independent marker for individuals who are at greater risk for developing depressive symptomatology (Henriques & Davidson, 1990). For example, subjects who had a history of depression but were in remission at the time resting EEG was recorded and additionally did not differ in their emotional state from never-depressed controls still displayed left frontal hypoactivation compared to controls (Henriques & Davidson, 1990). Similar decreases in left frontal activity were also observed in subjects who suffered from seasonal affect disorder even after subjects were in remission following light therapy treatment (Allen, Iacono, Depue, & Arbisi, 1993).

If the approach–withdrawal theory predicts that decreased left frontal activation reflects a faulty motivational approach system, then what would be predicted when one has a hyperactive or faulty motivational withdrawal system? It is adaptive to show withdrawal behaviors when faced with a valid threat, but it is not adaptive to display heightened withdrawal when the target stimulus is considered harmless. However, in individuals who suffer from anxiety-related disorders, seemingly harmless situations are subjectively interpreted as threatening. Therefore, it is hypothesized that these anxious individuals would display heightened levels of right frontal activation compared to nonanxious individuals (Davidson, Marshall, Tomarken, & Henriques, 2000b). Resting EEG was recorded from controls and phobics who suffered from a fear of public speaking. Prior to recording EEG, subjects were told that they would have to give a public speech in front of a group of raters. EEG was then recorded while the subjects prepared and anticipated giving their speech. Increased right frontal activation was observed in the phobics compared to controls during the anticipation of giving a speech. Phobics also reported higher levels of anxiety and negative affect prior to giving their speech, suggesting that right frontal activation is increased in anxious individuals during tasks that evoke anxiety (Davidson et al., 2000b). These results and the results from depressed subjects suggest that tendencies to display certain patterns of frontal asymmetric activation may be a marker for affective disorders. However, it should be noted that some studies have found inconsistencies in the results (for review see Gotlib et al., 1998; Heller & Nitschke, 1998; Heller, Nitschke, Etienne, & Miller, 1997; Nitschke, Heller, Palmieri, & Miller, 1999); but see Davidson’s (1998b) comments on some of these issues.
DEVELOPMENTAL ORIGINS OF FRONTAL EEG ASYMMETRY

The research reviewed above was conducted using adult populations. However, there has also been some research examining similar relations between motivational tendencies and frontal EEG asymmetry among infant and child samples. This research has covered ground similar to the adult work examining relations between frontal EEG asymmetry and the expression of different emotions (Fox & Davidson, 1986, 1987, 1988), indexing approach or withdrawal motivation (Davidson & Fox, 1992; Fox, 1991, 1994) or a trait measure reflecting temperament bias or disposition (Calkins, Fox, & Marshall, 1996; Fox et al., 2001; Henderson, Fox, & Rubin, 2001; McManis, Kagan, Snidman, & Woodward, 2002).

Unlike studies with adults where short- and long-term stability of frontal EEG asymmetry has been examined (Allen, Urry, Hitt, & Coan, 2004; Debener et al., 2000; Tomarken et al., 1992b; Vuga et al., 2006), there are few studies of the stability of this metric in infants or young children. The exceptions are a study by Bell and Fox (1994) that examined stability in young infants over a 6-month interval and found modest stability (between age correlations ranging from 0.07 to 0.83) and a study by Jones, Field, Davalos, and Pickens (1997) that found moderate stability from infancy to early childhood (between age correlations ranging from 0.42 to 0.66). Underlying the lack of repeated measurement of frontal EEG asymmetry in infancy and early childhood is the issue of understanding the etiology of this pattern and the possible changes in asymmetry that may be a result of early experience.

We know little about the developmental origins of frontal EEG asymmetry. For example, is it present at birth or does it emerge over the first years of life? What, if any, are the effects of early experiences on the development of this metric? Recent findings in behavioral genetics have shown that heritability of frontal EEG asymmetry is relatively modest (<30% of the variance) between identical twins, suggesting that environment must play a substantial role in the development of frontal asymmetry (Anokhin, Heath, & Myers, 2006). Here, we review studies that will help elucidate possible experiential contributions in the development of frontal EEG asymmetry.

STATE MEASURES OF EEG ASYMMETRY IN INFANT AND CHILD POPULATIONS

An initial formulation of a developmental model for frontal EEG asymmetry was articulated by Fox and Davidson over 20 years ago (Fox & Davidson, 1984). This model was centered on research and theory articulated by the ethologist T. C. Schneirla, who proposed that a fundamental organization of the nervous system across phylogeny involved circuits specialized for approach and withdrawal behaviors (Schneirla, 1959). The tendencies to approach or withdraw could be elicited by stimuli based upon their hedonic value and intensity. Fox and Davidson (1984) proposed that at birth, areas of the cortex were lateralized to support the motor programs and cognitive processes underlying either approach or withdrawal. These areas were thought to be connected to more sub-cortical regions that were activated during the experience of either positive or negative emotions, such that certain emotions would be associated with approach behaviors while other emotions were associated with withdrawal. As infants developed a more complex repertoire of motor and cognitive behaviors in response to contexts eliciting approach or withdrawal, regions of the prefrontal cortex would come to be specialized for these complex behavioral responses.

As evidence of these developmental changes, Fox and Davidson reported on two studies with human infants. In the first, newborn infants were presented with different liquid tastes (sugar water, citric acid, and quinine solutions). Previous work by Steiner (1979) had demonstrated that newborns displayed discrete facial movements suggesting either approach (eye widening and lip pursing) or avoidance (disgust expressions) in response to these tastes. Fox and Davidson (1986) recorded EEG from frontal and parietal scalp locations while presenting these liquids to the newborns and later coded infant facial expressions. They reported finding right EEG asymmetry during facial expressions associated with disgust or avoidance and left EEG asymmetry during facial expressions associated with approach. Of note, there were no differences in the pattern of asymmetry between frontal or parietal sites, suggesting that at birth there is hemispheric specialization but not regional specialization for approach–withdrawal behaviors.

In a second study, Fox and Davidson (1987) observed 10-month-old infants’ facial expression responses to approach of an unfamiliar adult and separation from their mother while recording EEG. The EEG recordings were synchronized to the video, and segments of EEG coinciding with the facial expressions were extracted for analysis. Examination of these data found significant asymmetric effects specific to the frontal leads. Emotions (interest, joy) linked with approach behaviors were associated with left frontal EEG asymmetry while emotions (fear, distress)
linked with withdrawal were associated with right frontal EEG asymmetry. The data suggest that, at least by 10 months of age, there are clear regional differences in EEG asymmetry related to approach and withdrawal behaviors, with specificity for the frontal region.

**EEG ASYMMETRY AS A TRAIT MEASURE IN INFANTS AND CHILDREN**

Although Fox and Davidson (1984) proposed a developmental model linking the emergence of emotions and approach–withdrawal behaviors to the development of frontal EEG asymmetry, they did not directly address the issue of the effects of early experience on its emergence. Rather, Davidson and Fox (1989) proposed that frontal EEG asymmetry may serve as a marker for temperamental disposition. In a study examining relations between baseline frontal EEG asymmetry and 10-month infant response to maternal separation, they reported that infants displaying right frontal EEG asymmetry were more likely to display distress at separation than those displaying left frontal EEG asymmetry (Davidson & Fox, 1989).

Fox and colleagues have since examined the role of frontal EEG asymmetry in infant temperament (Fox et al., 1995; Fox et al., 2001; Fox, Schmidt, Calkins, Rubin, & Coplan, 1996). In a series of studies, relations between infant temperament, social competence and behavior, and frontal EEG asymmetry have been examined. These studies have found that young children exhibiting right frontal EEG asymmetry are more likely to exhibit social withdrawal and behave in a socially maladaptive manner when interacting with unfamiliar peers (Fox et al., 1995; Fox et al., 1996; Fox et al., 2001). This pattern of frontal EEG asymmetry can be identified as early as nine months of age, and it, in combination with measures of infant negative or fearful temperament, predicts social reticence or withdrawal in the preschool and school age period. In addition, children who display stable patterns of behavioral inhibition over time also exhibit stable right frontal asymmetry. Interestingly, children who originally displayed a right frontal bias but changed over development from a more to a less inhibited behavioral profile displayed a change in their pattern from right to left frontal asymmetry (Fox et al., 2001). These results suggest that there are experiential factors that influence the stability of frontal EEG asymmetry across development. For example, in a longitudinal study of behavioral inhibition and frontal EEG asymmetry, inhibited children who were placed in daycare during the first year of life were more likely to change and became less inhibited, suggesting early daycare experience as a possible modulator of EEG asymmetry (Fox et al., 2001). Thus, it would appear that frontal EEG asymmetry may be affected by early experiences involved in patterns of caregiving.

**EARLY EXPERIENCE AND THE DEVELOPMENT OF EEG ASYMMETRY**

One interesting model for examining the effects of early caregiving experience on the development of frontal EEG asymmetry is maternal depression. Maternal depression has been shown to be related to an increased likelihood of the development of negative affect (Durbin, Klein, Hayden, Buckley, & Moerk, 2005; Field, Pickens, Fox, Gonzalez, & Nawrocki, 1998; Zahn-Waxler, Cummings, Iannotti, & Radke-Yarrow, 1984) as well as inhibition or withdrawal (Kochanska, 1991; Pauli-Pott, Mertesacker, & Beckmann, 2004) in infants and children. A variety of data suggest that women who are depressed, presenting either postpartum depression or a clinical history of major depressive disorder, display nonoptimal patterns of caregiving to their infants (Cohn, Matias, Tronick, Connell, & Lyons-Ruth, 1986; Cooper & Murray, 1997; Field, 1984; Field et al., 1988; Field et al., 1985). Specifically, studies have shown that mothers suffering from postpartum depression were more negative toward their 2-month-old infants and their infants were, in turn, less positive in face-to-face social interactions (Cohn et al., 1986; Cohn, Campbell, Matias, & Hopkins, 1990). Other research has found that the type of interaction patterns that infants have with their depressed mothers generalizes to those with nondepressed adults (Field, 1992). Field claims that infants appear to mirror the behavior of their depressed mothers (Malphurs et al., 1996), displaying less activity (Field et al., 1988), less contingent responsivity (Cohn et al., 1990), and more negative facial expressions (Cohn et al., 1990) compared to infants of nondepressed mothers.

Field and colleagues (Field, Diego, Hernandez-Reif, Schanberg, & Kuhn, 2003) have identified a number of individual differences in interactive styles among depressed women with their infants. Among them were those mothers who were “intrusive,” “withdrawn,” and those with typical “good” interaction skills. These different patterns were identified by careful observation and coding of the dyadic mother–infant interaction. “Intrusive” mothers were those who displayed rough physical contact, using what the authors describe as rapid staccato actions and tense or fake facial or vocal expressions (p. 239). In contrast, “withdrawn” mothers displayed
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flat affect, rare instances of vocalization or touching their infant and showed little face-to-face interaction. “Good” mothering was characterized as involving smiling, sensitive touching and contingent, responsive appropriate behaviors. Although infants of “good” mothering still displayed right frontal EEG asymmetry, this group showed the least lateralization compared to either withdrawn or intrusive mothering (Field et al., 2003). These results parallel findings reported by Hane and Fox (2006) in which quality of maternal care within a nondepressed sample influenced development of frontal EEG asymmetry where infants of mothers displaying high quality care were more likely to show a left EEG asymmetry bias. In contrast, infants who received low quality maternal care showed right EEG asymmetry suggesting that quality of maternal care in general may influence the development of EEG asymmetry.

Field and colleagues reported that the two aberrant styles of mothering (withdrawn and intrusive) resulted in different patterns of frontal EEG asymmetry in their infants. Infants of withdrawn mothers were more likely to display right frontal EEG asymmetry while infants of intrusive mothers were more likely to display left frontal EEG asymmetry (Diego et al., 2002; Diego, Field, Jones, & Hernandez-Reif, 2006a). As well, infants of mothers with these differing styles display different EEG patterns to face stimuli. Diego et al. (2002) found that infants of intrusive depressed mothers displayed a shift from left to right frontal EEG asymmetry when presented with facial expressions of surprise and sadness, while infants of withdrawn depressed mothers showed no such change in EEG asymmetry. The authors argue that these differences in infant baseline and reactive EEG reflect the different underlying physiologies of infants of depressed mothers, based upon their experience with one or another style of caregiving.

In another example of the effects of early experience on frontal EEG activity, Jones, McFall, and Diego (2004) reported that depressed mothers who breastfed their infants until the third month of life were less likely to show right frontal EEG asymmetry while infants of depressed mothers who were bottle fed exhibited a bilateral decrease in frontal EEG power. Although the precise mechanisms underlying the differences in frontal EEG activity are left unspecified, the effects of differential early experiences, whether they are behavioral or nutritional, on frontal EEG asymmetry are clear.

One obvious question that remains about the influence of maternal depression on the development of infants’ frontal EEG asymmetry is whether the asymmetry is a result of the prenatal or the postnatal environment. Recent work by a number of labs has begun to examine the influences of depression in pregnant women on the developing fetus and subsequently on the newborn and infant child. This work stems from data from two sources. The first is research on the effects of depression in women during pregnancy on physical parameters and birth outcomes. For example, women suffering from depression have infants with more perinatal complications and these infants are more likely to have low birth weight (Diego et al., 2006b; Field, Diego, & Hernandez-Reif, 2006; Hoffman & Hatch, 2000). A second source is data on the effects of depression on the fetus, itself. For example, Field and colleagues (2001) found that fetuses of depressed women are less active and less responsive to stimulation (see also work by DiPietro, Hawkins, Hilton, Costigan, & Pressman, 2002; DiPietro, Irizarry, Costigan, & Gurewitsch, 2004). The data from Field and colleagues clearly demonstrate physiological effects on the fetus among women with depression. If that is the case, it calls into question the findings on changes in frontal EEG asymmetry of infants with depressed mothers. The effects on fetal development and the effects on neonatal development appear to be tangled and hard to parse apart.

EARLY EXPERIENCE AND DEVELOPMENT OF CEREBRAL ASYMMETRY IN ANIMALS

Because there is little control over the environment in human development studies, it is difficult to determine which environmental factors lead to observed frontal asymmetries. Therefore, examining environmental influences on brain asymmetry using nonhuman animal models may give some insight into how such asymmetry develops in humans. Occurrence of brain lateralization in nonhuman species has been known for several decades (for a review see Rogers & Andrew, 2002). Both nonhuman primates (Kalin, Larson, Shelton, & Davidson, 1998) and rats (Sullivan & Gratton, 1998, 2002) display similar patterns of asymmetry to emotional stimuli within the frontal cortex as that observed in behaviorally inhibited children (Calkins et al., 1996; Fox et al., 2001; McManis et al., 2002). Kalin and colleagues (1998), for example, showed that individual differences in frontal EEG asymmetry in monkeys were related to trait-like fear behaviors and cortisol response, where monkeys with greater right frontal activation had greater fear and higher levels of cortisol. In rats, lesions to the right medial prefrontal cortex (mPFC) led to increased entry into the open arms of an elevated plus maze and decreased avoidance of a quinine solution (Sullivan & Gratton, 2002) as...
well as decreased corticosterone and stress-induced ulcer development (Sullivan & Gratton, 1999). The relations between emotion-related behavior and brain asymmetry has been shown to be dependent on early life experiences (Denenberg, 1981; Lyons, Afarian, Schatzberg, Sawyer-Glover, & Moseley, 2002; Sullivan & Dufresne, 2006; Tang, 2003). Examining this literature within the framework of emotional development may help explain how EEG asymmetry develops in the human infant.

The first study examining early experience-induced asymmetry was conducted 30 years ago by Denenberg (1978). From postnatal days 1–20, litters of rat pups were exposed to a novel cage for 3 min per day while control litters remained undisturbed. This neonatal handling procedure has been shown to have a lifelong impact on the emotional development of the rat by decreasing emotionality in an open field test (Denenberg, 1962; Denenberg, 1964) as well as inducing a better adaptive stress response (Levine, 1957, 1960). This decrease in emotionality observed among the handled animals was shown to be influenced by a stimulation-induced right-shift in brain asymmetry. Handled rats with ablations to the right neocortex showed decreased emotionality while no differences were observed between handled animals with left ablations and those with intact brains (Denenberg, 1978, 1981). In contrast, nonhandled animals showed no cerebral asymmetry.

The observed asymmetry among handled animals parallels the right cerebral lateralization for emotional stimuli that is commonly observed in the human literature (for reviews see Davidson, 2003; Davidson et al., 2000a; Fox, 1991, 1994). Of interest is the finding that nonhandled rats do not display such lateralization, suggesting that some facet of stimulation early in life induces normal development of brain lateralization for emotional regulation (Denenberg, 1981). As well, neonatal stimulation may induce cerebral lateralization by enhancing the growth and development of callosal connections which then gives rise to more specialized brain function (Denenberg, 1981). In a more recent study, it was found that individual rats display differential levels of dopamine content where some rats were more left lateralized and some were more right lateralized (Thiel & Schwarting, 2001). Of these animals, the ones who were more right lateralized also displayed less emotionality. These results suggest that observation of brain asymmetry in relation to emotional stimuli in rats is not uncommon and further supports Denenberg’s original hypothesis (Denenberg, 1981)—early experience actually induces a rightward shift in asymmetric activation at the population level. However, because this study did not control the localization of the lesions made within the neocortex, it is hard to determine if the early stimulation effects are localized to a particular region or from where in the brain the frontal lateralization propagates.

EARLY POSTNATAL STIMULATION AND UNDERLYING NEUROBIOLOGY OF CEREBRAL ASYMMETRY: RAT MODELS

One region of the brain suggested to give rise to cerebral lateralization via early stimulation is the hippocampus (Sullivan & Gratton, 2003; Tang, 2003; Verstynen, Tierney, Urbanksi, & Tang, 2001; Tang, Zou, Reeb, & Connor, 2008). Enhanced negative feedback to a stressor observed among animals that received early life stimulation has been shown to be mediated by the increased number of glucocorticoid receptors (GRs) within the hippocampus (Meaney et al., 1985). Given the connections between the hippocampus and the frontal cortex (Thierry, Gioanni, Degeneaais, & Glowinski, 2000), it is logical to explore the hippocampus as a possible brain region linked to differences in cortical asymmetry in animals exposed to early stimulation (Tang, 2003).

In studies examining the effects of early stimulation on hippocampal function, Verstynen et al. (2001) found that brief exposures to novelty during the first three postnatal weeks induced a right-shift in volumetric asymmetry in the hippocampus. As well, Tang and colleagues reported similar lateralization in hippocampal long-term potentiation (LTP) (Tang et al., 2008) and sensitivity of hippocampal LTP to exogenous corticosterone (Tang, 2003). In another study by Sullivan and Gratton (2003), the effects of neonatal handling on benzodiazepine receptor binding within the hippocampus and dentate gyrus were investigated. Handling was found to induce a rightward shift in benzodiazepine receptor binding (Sullivan & Gratton, 2003).

Increased sensitivity to corticosterone is an indirect measure of the number of active glucocorticoid receptors (GRs) within the hippocampus, suggesting that stimulated animals have a greater number of GRs on the right side of the hippocampus. Right-lateralized hippocampal GRs may lead to a right-shift in negative feedback to emotional stimuli and thus down-regulate activation within the neonatally stimulated animals’ right frontal cortex. Similarly, benzodiazepines induce GABAergic inhibition perhaps leading to greater inhibition of right-sided stress-induced activation among handled animals. These studies offer direct evidence that neonatal stimulation induces a rightward shift in the hippocampus and indirect evidence that early stimulation-induced frontal asymmetry may originate from the hippocampus.

In addition to the study of early stimulation effects on hippocampal lateralization development, there are studies
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examining lateralization of dopamine, a catecholamine that acts as a modulator of prefrontal activation (for review, see Seamans & Yang, 2004; Thierry et al., 2000). Individual differences in the lateralization of postmortem dopamine content in the frontal cortices were related to levels of emotionality where greater elevations in right compared to left dopamine content predicted less emotionality in the rat (Thiel & Schwarting, 2001). Given these results, it was hypothesized that early life stimulation may influence the development of brain lateralization via the dopaminergic system within the prefrontal cortex (PFC). In separate experiments examining the effects of early handling on the development of mesocortical dopaminergic output, Sullivan and Dufresne (2006) found that early stimulation enhanced dopaminergic turnover in the right prefrontal cortex. Parallel to this lateralization in dopamine turnover, early stimulation decreased corticosterone to both repeated and acute stress. These results suggest that increased dopaminergic activation in the right frontal cortex may counteract levels of corticosterone by facilitating the return to basal levels of glucocorticoids as well as help inhibit activation of central amygdalar output and other cortical regions typically involved in stress regulation (Day et al., 2002). In addition, dopamine appears to modulate the excitability of hippocampal activity as it propagates to the prefrontal cortex (for a review, see Thierry et al., 2000). Thus, early experience leads to not only an increased downregulation of right frontal activity via increased right lateralized negative feedback to a stressor but also has additional control over hypothalamic–pituitary–adrenal (HPA) axis activation through increases in dopaminergic metabolism within the right prefrontal cortex.

Early Experience and Cerebral Lateralization: Nonhuman Primate Models

Research in early stimulation and asymmetry has primarily focused on the rat. However, there has been one report of the effects of early life stimulation on the development of asymmetry in nonhuman primates’ prefrontal cortex. Lyons et al. (2002) randomly assigned infant monkeys to one of three groups: low maternal foraging demand, high maternal foraging demand, or intermittent social separations. In the low foraging demand group, the mothers had easy access to an abundance of food, whereas in the high foraging demand group, mothers had to search longer and had to exert greater effort to acquire a similar amount of food. The intermittent separation group was fed regularly. However, the infants were exposed to brief 1-hr separations from their mother on a biweekly basis from postnatal weeks 13–21. Both high foraging demand and intermittent separations have been shown to decrease emotionality as well as enhance cortisol stress response (Lyons, Martel, Levine, Risch, & Schatzberg, 1999; Rosenblum & Andrews, 1994). Four years following this early life stimulation procedure, structural magnetic resonance imaging was performed and white and gray matter volumes of the prefrontal cortex were measured.

Compared to monkeys raised in the low foraging demand group, monkeys who were raised in either the high foraging demand or intermittent separation groups displayed a right-shift in gray matter volume in the dorsolateral prefrontal cortex as well as an overall right-shift in prefrontal white matter volume (Lyons et al., 2002). In addition, only the monkeys who were exposed to intermittent separations early in life displayed greater right gray matter volume in the ventral medial frontal cortex. The authors suggest that this rightward shift in prefrontal volume may reflect a rightward increase in prefrontal GRs which more quickly activates negative feedback of the stress response system, thus down-regulating activation of the right prefrontal cortex. Similar to early stimulation effects and development of asymmetry in rats, early stimulation in nonhuman primates induces a rightward shift in lateralization.

The animal studies reviewed above have shown that early stimulation induces asymmetry in prefrontal cortex volume and dopaminergic turnover, as well as hippocampal response to corticosterone and benzodiazepines. However, it is likely that early stimulation induces asymmetry in several other regions of the brain, especially those which are known to be involved in the processing of emotional information. For example, the amygdala has been suggested to modulate right prefrontal cortex (for reviews see Davidson & Irwin, 1999; Davidson et al., 2000a; Fox, Henderson, Marshall, Nichols, & Ghera, 2005). The amygdala has connections to both the hippocampus and the prefrontal cortex (Sotres-Bayon, Bush, & LeDoux, 2005), suggesting that the neural connections between the amygdala and these brains regions or perhaps the neural activation of the amygdala itself may also be affected by early stimulation in an asymmetric fashion. However, studies have yet to be conducted to examine whether such lateralization occurs.

Integrating Human and Animal Findings

What elements of early stimulation lead to these lateralized effects and how can animal studies inform us about the developmental origins of asymmetry in EEG in humans? It has been suggested that increased maternal
care in the form of licking-grooming and arched-back nursing in rats mediates the observed effects of neonatal stimulation (Francis, Diiorio, Liu, & Meaney, 1999; Liu et al., 1997). Therefore, it may be that the development of asymmetry in neonatally stimulated rats is also mediated by increases in maternal care. This may explain the results found by Hane and Fox (2006) where infants who received high quality maternal care display decreased right frontal activation compared to those who received low quality maternal care.

However, more recent studies in both primates (Parker, Buckmaster, Sundlass, Schatzberg, & Lyons, 2006) and rodents (Tang, Akers, Reeb, Romeo, & McEwen, 2006) show that increased maternal care does not necessarily produce early stimulation effects. One suggested alternative mechanism is stress inoculation, which directly results from early repeated exposures to novelty/stress (Parker et al., 2006). This mechanism may help explain the change in behaviorally inhibited children who were put into day-care at an early age (Fox et al., 2001). It is probable that exposure to the daycare environment provided infants and children exposure to an ever-changing, unpredictable environment, thus increasing the chances that the child will be exposed to new and stressful situations. Subsequently, this exposure would lead to the increased chance to learn how to self-regulate during these stressors, thus ultimately leading to better emotional regulation and stress “inoculation” for stress observed in later childhood. In contrast, those children who were more likely to remain stable in their behavioral inhibition profile were those who remained in the exclusive care of the parent during the first year of life, having little opportunity to interact with new and stressful situations to help “inoculate” them against stress during later life.

Another hypothesis proposed to explain early stimulation effects suggests that these effects are modulated by maternal care in which the early stimulation has a direct effect on the infant’s stress response system and the mother acts to regulate the infant’s stress response immediately after the infant has been exposed to novelty (Reeb, Romeo, McEwen, & Tang, 2006; Tang et al., 2006). If the mother is reliable in her caretaking behavior after the infant is exposed to novel stimuli, then this will act to alleviate the infant’s stress response. After repeated encounters with stressors and reliable responses from the mother, this helps to shape a more adaptive stress response over time. However, if the mother is not reliable in her care toward her infant, a less adaptive stress response will develop. This may be related to what is occurring in infants with depressed mothers. Although reliability in caretaking behaviors has not been examined among depressed and nondepressed mothers, it could be that mothers who are depressed may also be less reliable in maternal care compared to nondepressed mothers. If this were the case, then it would be expected that infant interactions with depressed mothers would lead to the development of right frontal activation while those with nondepressed mothers would lead to more left frontal activation. Differences in the development of EEG asymmetry observed between the infants of depressed mothers who are either intrusive or withdrawn in their caregiving style may also be explained by the reliability of maternal behavior. However, these patterns of reliability within depressed mothers remain to be tested.

SUMMARY AND CONCLUSIONS

In this chapter, we briefly reviewed the relations between approach–withdrawal motivation and frontal EEG asymmetry. There is a corpus of evidence that suggests that left frontal activation is approach related and right frontal activation is withdrawal related. This phenomenon has been described as a reflection of the evoked emotional state as well as a trait of motivational biases. Individual differences in EEG asymmetry are apparent at very early ages and early experience plays a role in influencing the development of such asymmetry. It is not yet clear which components of early experience, such as having a depressed mother, affect the development of EEG asymmetry or whether certain components of the environment increase the likelihood of stability in EEG asymmetry over a long period of time. Because isolation of such components is difficult in a human population, the use of animals in translational research may help illuminate specific environmental components that influence the development of EEG asymmetry. Although some animal research investigates the impact of early experience on development of cerebral asymmetry, further research as well as greater communication between animal and human researchers is needed.

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