Resting frontal EEG asymmetry in children: Meta-analyses of the effects of psychosocial risk factors and associations with internalizing and externalizing behavior

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Abstract

Asymmetry of frontal cortical electroencephalogram (EEG) activity in children is influenced by the social environment and considered a marker of vulnerability to emotional and behavioral problems. To determine the reliability of these associations, we used meta-analysis to test whether variation in resting frontal EEG asymmetry is consistently associated with a) having experienced psychosocial risk (e.g., parental depression or maltreatment) and b) internalizing and externalizing behavior outcomes in children ranging from newborns to adolescents. Three meta-analyses including 38 studies (N = 2,523) and 50 pertinent effect sizes were carried out. The studies included in the analyses reported associations between frontal EEG asymmetry and psychosocial risk (k = 20; predominantly studies with maternal depression as the risk factor) as well as internalizing (k = 20) and externalizing (k = 10) behavior outcomes. Psychosocial risk was significantly associated with greater relative right frontal asymmetry, with an effect size of d = 0.36 (p < .01), the effects being stronger in girls. A non-significant relation was observed between right frontal asymmetry and internalizing symptoms (d = 0.19, p = .08), whereas no association between left frontal asymmetry and externalizing symptoms was observed (d = 0.04, p = .79). Greater relative right frontal asymmetry appears to be a fairly consistent marker of the presence of familial stressors in children but the power of frontal asymmetry to directly predict emotional and behavioral problems is modest.

Keywords: children; electroencephalogram; frontal asymmetry; psychosocial risk; depression; maltreatment; internalizing; externalizing

Introduction

A common goal in the field of developmental psychopathology is to determine biomarkers that show reliable associations with children's vulnerability to emotional or behavioral problems. Among the candidate markers, a considerable amount of attention has been devoted to patterns of hemispheric asymmetry in frontal electroencephalogram (EEG) alpha-band activity. The interest in frontal EEG asymmetry in developmental research is largely due to the fairly consistent pattern of greater relative right-sided frontal EEG asymmetry observed in currently and previously depressed adults (e.g., Henriques & Davidson, 1990; Schaffer, Davidson, & Saron, 1983). In the present study, we used meta-analysis on 38 studies (N = 2,523) to test whether children's frontal EEG asymmetry is consistently associated with a) the presence of psychosocial risk factors such as parental depression or child maltreatment, and b) child internalizing and externalizing behavior.

Frontal EEG alpha asymmetry refers to the difference in the amount of cortical activity in one hemisphere relative to the other. Asymmetry scores are computed from the EEG signal as the difference in ln-transformed EEG power within the alpha frequency band (8-13 Hz in adults, 6-9 Hz in infants and young children; Marshall, Bar-Haim, & Fox, 2002) between left and right frontal electrode sites (i.e., ln-right minus ln-left). The typical experimental setup for EEG asymmetry consists of 1 to 8 minutes of resting/baseline recording during which external stimulation is minimal or kept constant and neutral. As power in the alpha frequency band is inversely related to neural activity in the underlying cortex (i.e., stronger power indicating less activity; Lindsley & Wicke, 1974), positive alpha asymmetry scores are considered to reflect greater relative left frontal cortical activity, whereas negative scores reflect greater relative right frontal cortical activity.

The functional significance of asymmetrical frontal cortical activity is often interpreted from the perspective of the approach/withdrawal model (Davidson, Ekman, Saron, Senulis, & Friesen, 1990; Davidson, 1992; S. K. Sutton & Davidson, 1997) which relates asymmetries in frontal brain activity to basic motivational tendencies, with the left frontal areas subserving approach motivation and the right frontal areas subserving withdrawal motivation. A considerable body of research has shown that in adults, depression is associated with greater relative right frontal cortical activity (see Thibodeau, Jorgensen, & Kim, 2006, for meta-analytic evidence), even in individuals in remission from depression (Gotlib, Ranganath, & Rosenfeld, 1998; Henriques & Davidson, 1990). Right-sided frontal asymmetry may thus be an endophenotype of a trait-like withdrawal motivation associated with internalizing psychopathology such as depression and anxiety (Allen & Cohen, 2010; Davidson, Marshall, Tomarken, & Henriques, 2000).

The motivational model makes a crucial distinction between motivational direction and affective valence by arguing that asymmetrical frontal cortical activity promotes motivational tendencies to approach and withdraw independently of the affective valence underlying such tendencies (Harmon-Jones, Gable, & Peterson, 2010). Indeed, although left-sided frontal EEG asymmetry is associated with higher positive emotionality (Tomarken, Davidson, Wheeler, & Doss, 1992), negatively valenced externalizing behaviors that are related to approach (rather than withdrawal) tendencies, such as trait and state anger, have been shown to be associated with greater relative left frontal EEG activity as well (Harmon-Jones & Allen, 1998; Harmon-Jones & Sigelman, 2001; Verona, Sadeh, & Curtin, 2009).

Frontal cortical asymmetry is also influenced by the early social environment, with a particularly rich literature on the associations between maternal depression and

EEG asymmetry in infants and young children (see Field & Diego, 2008, for a review). Several studies have observed greater right frontal EEG asymmetry in infants of depressed vs. non-depressed mothers (e.g., Dawson, Frey, Panagiotides, Osterling, & Hessl, 1997; Diego, Field, Jones, & Hernandez-Reif, 2006; Field, Fox, Pickens, & Nawrocki, 1995), and similar findings have been observed in adolescents of depressed mothers regardless of the adolescents' own depression levels (Tomarken, Dichter, Garber, & Simien, 2004). Research investigating the influence of other types of psychosocial risk factors on children's patterns of frontal EEG asymmetry is scarce. A small number of studies have investigated whether the risks posed by insensitive maternal caregiving (Hane & Fox, 2006), parental alcohol dependence (Ehlers, Wall, Garcia-Andrade, & Phillips, 2001), and more severe conditions such as early institutionalization (McLaughlin, Fox, Zeanah, & Nelson, 2011) and child maltreatment (Curtis & Cicchetti, 2007; Miskovic, Schmidt, Georgiades, Boyle, & MacMillan, 2009) are also associated with right-sided EEG asymmetry. While some studies have documented strong effects of psychosocial risk factors in determining the extent and direction of children's frontal EEG asymmetry (Diego et al., 2006; Jones et al., 1998; Miskovic et al., 2009), others have found no significant differences between children experiencing high vs. low risk (Dawson, Klinger, Panagiotides, Hill, & Spieker, 1992; Lusby, Goodman, Bell, & Newport, 2014). One highly unexplored question concerns the ontogenetic mechanisms linking psychosocial risk to variations in frontal asymmetry, i.e., whether genetic or experience-based effects are more influential in shaping children's frontal cortical activity. While the large effects observed already in newborn infants of depressed mothers (Field, Diego, Hernandez-Reif et al., 2004) may be taken to indicate a genetic disposition to greater right-sided asymmetry in some individuals, longitudinal investigations aimed at producing long-

lasting changes in children's exposure to psychosocial risks are required to address this issue comprehensively.

Greater right-sided EEG asymmetry is commonly interpreted as a marker of heightened risk of psychopathology and emotional dysregulation in children. To directly test whether patterns of frontal EEG asymmetry can be considered as markers of vulnerability to the development of emotional and behavioral problems, studies have investigated the relations between EEG asymmetry and internalizing and externalizing behavior outcomes in preschool and school age children, with both concurrent and prospective study designs. In keeping with the model based on cortical asymmetries and direction of motivational tendencies in adults (Harmon-Jones et al., 2010), it can be hypothesized that internalizing symptoms (e.g., depression, anxiety, and social withdrawal) would show associations with greater relative right asymmetry, whereas externalizing symptoms (e.g., aggressive and impulsive behavior) are expected to be related to greater left asymmetry. The directional hypothesis has been supported by some studies (e.g., Gatzke-Kopp, Jetha, & Segalowitz, 2014; Jones, Field, Davalos, & Pickens, 1997b; Pössel, Lo, Fritz, & Seemann, 2008; Smith & Bell, 2010), whereas other studies have observed an opposite pattern of EEG activation (Baving, Laucht, & Schmidt, 2002; Santesso, Reker, Schmidt, & Segalowitz, 2006) or no direct associations between EEG and these outcomes (Fox, Schmidt, Calkins, Rubin, & Coplan, 1996; Theall-Honey & Schmidt, 2006).

In the present study we used meta-analysis to test a) the consistency of the association between having experienced psychosocial risk and frontal cortical EEG asymmetry and b) the effect sizes of the associations between frontal EEG asymmetry and internalizing and externalizing behavior outcomes. The first hypothesis was that

higher psychosocial risk including parental depression would be related to greater relative right frontal EEG asymmetry as compared to lower psychosocial risk. We incorporated data on all potential psychosocial risk factors and compared the effects of parental depression to those of other risk factors to obtain a more comprehensive picture of the range of psychosocial influences on children's EEG asymmetry. Second, we tested the hypotheses stemming from the motivational direction model (Harmon-Jones et al., 2010) that internalizing behavior outcomes are associated with greater relative right, and externalizing behavior outcomes with greater relative left EEG asymmetry. In addition to computing the combined effect sizes for the three sets of meta-analyses (i.e., psychosocial risk, internalizing, and externalizing), we ran moderator analyses to investigate whether effect size variations across studies are associated with sample characteristics or procedural differences between studies. In all meta-analyses, we tested the effects of participant age, gender, socioeconomic status (SES), and resting EEG recording duration (to test whether shorter recording durations are associated with larger effects, as in the meta-analysis of adult data by Thibodeau et al., 2006). Additional moderator analyses tested the effects of different types of psychosocial risk, different ways to assess psychosocial risks and child outcomes (i.e., diagnosed vs. self-reported parental depression measures and observed vs. reported child behavior), and the time lag between EEG recording and assessment of internalizing or externalizing.

Methods

Literature search

Figure 1 outlines the study selection process. To obtain data for the meta-analyses, we started with using PsycINFO and Google Scholar to search all empirical journal articles in the English language available by August 15th, 2013, with the key

words EEG or electroencephalogra*, and asymmetr* in the title or abstract (the asterisk indicating that the search contained the word or word fragment). We limited the search results to studies including participants younger than 18 years old. This search produced a total of 208 articles, the abstracts of which were screened. Studies were excluded if they did not report data on *resting* frontal alpha band EEG activity, for example if a) EEG asymmetry was reported only in response to a discrete stimulus or event, b) asymmetry scores or differences in alpha power in the left and right hemisphere were absent, or c) only non-frontal (e.g., parietal) asymmetry data was reported. Articles were also excluded if they did not provide data on associations between EEG asymmetry and psychosocial risk factors or on outcomes that could be defined in terms of internalizing or externalizing behavior.

The majority of studies including data on psychosocial risk factors investigated the association between maternal depression and children's EEG asymmetry. In Bruder, Tenke, Warner, and Weisman (2007), it was not specified which parent was affected and, therefore, children in the high risk group of this study had at least one parent (mother, father, or both) and at least one grandparent with a diagnosis of major depressive disorder. For the present meta-analysis, studies investigating more severe forms of psychosocial adversity such as childhood maltreatment (Curtis & Cicchetti, 2007; Miskovic et al., 2009) or institutionalization (McLaughlin et al., 2011) were also included, as well as two studies on the associations between child frontal EEG asymmetry and maternal caregiving insensitivity (Hane & Fox, 2006), and parental alcohol dependence (Ehlers et al., 2001). Studies contributing to the analyses of internalizing behaviors consisted of outcomes related to anxiety, fearfulness, depressiveness, social withdrawal, and shyness reported by the parent or the child, or observations of facial signs of fear or

inhibited behavior in novel or ambiguous contexts. The outcomes in the set of studies on externalizing behaviors included symptoms of aggression and oppositional defiance, which were in all cases reported by the caregiver (or teacher; Gatzke-Kopp et al., 2014). Whenever an article reported effects separately for female and male participants, these were considered as separate outcomes. Studies that included data on negative affect expressions that could not be clearly defined in terms of approach or withdrawal motivation were excluded from the internalizing and externalizing analyses. This resulted in the exclusion of studies investigating relations between EEG asymmetry and crying in response to maternal separation (Davidson & Fox, 1989), sad facial expressions (Jones, Field, Fox, Lundy, & Davalos, 1997a), or more global indices of negative emotionality (Dawson et al., 1999; Jones, McFall, & Diego, 2004; Shankman et al., 2011).

In the next step, articles were checked for partly overlapping samples and in such cases, the article with the largest sample size was selected. This ensured that no participants were included twice in the same meta-analysis. From the longitudinal temperament study conducted by Fox and colleagues, we selected Henderson, Fox, and Rubin (2001) to represent the internalizing data and Hane, Henderson, Reeb-Sutherland, and Fox (2010) to represent the externalizing data from this project. Although these publications do not report on the largest sample sizes in the context of this project (Degnan et al., 2011), they were considered most representative/adequate in terms of sample size, time between EEG measurement and outcome, and the availability of sufficient statistical information for effect size calculations. From the *Mannheim Study of Risk Children*, we selected the 8-year assessments (Baving, Laucht, & Schmidt, 2000; Baving et al., 2002) as these represent the midpoint of a longitudinal study from 4.5 to 11 years. After these steps, we identified 38 empirical

papers with 50 pertinent effect sizes, providing data for three sets of meta-analyses on psychosocial risk factors (k = 20, N = 1,291), internalizing (k = 20, N = 1,299), and externalizing (k = 10, N = 810) child behavior.

Figure 1 here

Moderators

Socioeconomic status (SES; low vs. middle/high) was coded as a categorical moderator for all analyses. For the set of articles contributing to the psychosocial risk analyses, we also coded risk type (parental depression vs. other adversity) and for the studies measuring parental depression, the type of depression assessment (diagnosis vs. self-report). For the associations with internalizing and externalizing behavior, additional categorical moderators were the temporal relationship between EEG recording and the outcome assessment (concurrent vs. predictive), and outcome assessment type (observed vs. reported behavior). Moderator subgroups with $k < 4$ were excluded from the categorical contrast analyses. Continuous moderators included age at the time of EEG measurement, age at the time of outcome assessment, gender (% of male participants), time lag (in years) between EEG recording and outcome assessment, and resting EEG recording duration. In cases where EEG was recorded twice (e.g., Smith & Bell, 2010), the data were averaged across the two assessments, as was age at the two assessment points. To assess intercoder reliability, 11 of the studies were coded by an independent coder. The agreement between the coders across the categorical moderator variables was 98% (κs > .86) and correlations between the continuous moderators were > .97.

Meta-analytic procedures

The meta-analyses were performed using the Comprehensive Meta-Analysis (CMA) program (Borenstein, Rothstein, & Cohen, 2005). For each study, an effect size (Cohen's *d*) was calculated as the standardized difference between high and low psychosocial risk conditions or between high and low manifestations of child internalizing or externalizing behavior in resting frontal EEG asymmetry values. For studies reporting correlational data, these were recomputed into Cohen's *d*. Except for Curtis and Cicchetti (2007) who reported the pertinent data from electrodes F7/8, and Gatzke-Kopp et al. (2014) who reported data only from electrodes AF3/4, the effect size calculations were based on data reported from mid-frontal electrodes F3 and F4. For the analyses of psychosocial risk and internalizing problems, effects of greater relative right-sided asymmetry were given a positive sign as they were in accordance with our hypotheses. As externalizing behavior was hypothesized to be associated with greater relative left-sided asymmetry, studies reporting effects of a greater relative right-sided asymmetry associated with externalizing behavior were given a negative sign (recall that due to the inverse relationship between alpha power and neural activity, right-sided asymmetry indicates lower alpha power/greater neural activity on the right frontal electrode sites).

CMA was used to compute combined effect sizes (weighted by the sample sizes within individual studies) and 95% confidence intervals (*CI*s) around the point estimates for the three separate sets of effects. Significance tests and moderator analyses were performed with the *Q*-statistic on the basis of random-effects models (Borenstein et al., 2005). Random-effects were favored over fixed-effects models as they allow for the possibility that there are random differences between studies that are associated with variations in procedures, measures, settings, that go beyond subject-level sampling error and thus point to different study populations (Lipsey &

Wilson, 2001). In case of statistically significant combined effect sizes, the robustness of these effects was estimated with the fail-safe number provided by the CMA program, which estimates the number of studies with null results that would be needed to reduce the overall significant result to non-significance. The *Q*-statistic was also used to assess the heterogeneity of the effect sizes across studies. Meta-regression was used to test the influence of continuous moderators. For each set of effect sizes, Fisher's *Z* scores were computed as equivalents for the effect size *d*, and the *Z* scores were then standardized to screen for potential outliers. No outliers (standardized Z scores ±3.29; Tabachnick & Fidell, 2001) were observed in the total set of studies or the three separate sets.

To calculate the effect of potential data censoring or publication bias on the significant outcomes of the meta-analyses, we used the trim-and-fill method. A funnel plot was constructed of each study's effect size on the x-axis against the inverse of the standard error on the y-axis. The plot is expected to have the shape of a funnel because studies with smaller sample sizes and larger standard errors have increasingly large variation in estimates of their effect sizes as random variation becomes increasingly influential, whereas studies with larger sample sizes have smaller variation in effect sizes, making the top portion of the plot narrower (Duval & Tweedie, 2000; A. J. Sutton, Duval, Tweedie, Abrams, & Jones, 2000). The plots would be expected to be shaped like a funnel if no data censoring is present. However, since smaller non-significant studies are less likely to be published, studies in the bottom left hand corner of the plot are often omitted. With the trim-and-fill procedure, the *k* right most studies considered to be symmetrically unmatched are trimmed and their missing counterparts are imputed or 'filled' as mirror images of the trimmed

outcomes. This leads to a new estimate of the combined effect size taking into account potential publication bias.

Finally, in case of statistically significant combined effect sizes, we performed a power analysis with the G*Power 3.1 program (Faul, Erdfelder, Lang, & Buchner, 2007). First, we calculated the sample size required for an individual study to reach the combined effect size (i.e., the assumed population effect size) with a power of 0.80 and a one-sided significance level of 0.05. Second, the actual power values of the individual studies were calculated to estimate the range of power of the included studies to detect the combined effect size.

Results

The combined effect sizes for the three sets of analyses and the primary categorical moderator contrasts are displayed in Table 1. Tables 2 – 4 list the studies contributing to the meta-analyses with descriptive data and forest plots representing the individual effect sizes.

Table 1 here

Psychosocial risk

Within the set of studies on the associations between psychosocial risk factors and EEG asymmetry, a significant combined effect size was observed (d = 0.36, *CI* 0.15 – 0.58, $p < .01$), indicating that the presence of psychosocial risk factors is associated with greater relative right-sided frontal EEG asymmetry. The set of outcomes was heterogeneous. The trim-and-fill method showed that one study had to be trimmed and filled, while the resulting combined effect size remained basically similar (d = 0.32, *CI* 0.09 – 0.54). The fail-safe number was 168, indicating a robust

effect. As can be observed from Table 1, the effect was of comparable magnitude between different types of risk (i.e., parental depression vs. maltreatment or institutionalization) and between different levels of SES. Within the set of depression studies (k = 14), no difference in the magnitude of effects was observed between the two types of depression assessment (diagnosis: k = 9, d = 0.35, *CI* 0.03 – 0.66; self-report: k = 5, d = 0.54, *CI* 0.14 – 0.95; Q [1] = 0.56, p = .45).

The power analysis indicated that a sample size of N = 194 would be required for an individual study to detect the combined effect size of d = 0.36 with a power of 0.80. The power values of the included studies to detect the combined effect size ranged from 0.21 for the study with the smallest sample size (Field, Pickens, Fox, Gonzalez, & Nawrocki, 1998) to 0.67 for the study with the largest sample size (Ehlers et al., 2001), with the median power of the included studies being 0.39.

Meta-regression analyses with the continuous moderators revealed that the effects were significantly moderated by gender but not age or resting EEG recording duration (both ps > .10). Gender (the percentage of males in each sample) yielded a significant negative regression weight (slope = -0.01, p = .04), indicating that studies with a larger percentage of females in the sample were associated with larger effects. When the continuous moderators were tested separately within the set of depression studies (k = 14), age emerged as a significant moderator (slope = -0.05, p = .02), with larger effect sizes in younger samples, while the effects of gender (p = .44) and recording duration (p = .68) were not significant.

Table 2 here

Internalizing

The combined effect size (d = 0.19, *CI* -0.03 – 0.41, p = .08) for the internalizing set was not significant. The set of studies was heterogeneous. Effect sizes were not associated with SES, EEG-outcome time lag (i.e., concurrent vs. predictive), or assessment type (observed vs. reported). Again, larger effects were observed in samples with higher percentages of females (slope = -0.01, p = .02). The associations between EEG asymmetry and internalizing behaviors were unrelated to age at the time of EEG recording or outcome assessment, time lag between EEG recording and outcome assessment, or resting EEG recording duration, all ps > .52.

Table 3 here

Externalizing

The combined effect size (d = 0.04, *CI* -0.27 – 0.35, p = .79) in a heterogeneous set of outcomes provided no support for the hypothesis that frontal EEG asymmetry would be related to externalizing behaviors in children. While no associations between effect sizes were observed with all other categorical or continuous moderators, gender was significantly associated with the magnitude of effects (slope = 0.02, p < .001), but in the opposite direction as was the case in the analyses of psychosocial risk and internalizing behavior. That is, stronger associations between left-sided EEG asymmetry and externalizing behaviors were observed in samples including relatively greater numbers of males.

Table 4 here

Discussion

The present meta-analytic study was designed to test whether the extent and direction of frontal EEG asymmetry is consistently associated with a) having experienced psychosocial risk and b) internalizing and externalizing behavior outcomes in studies of children ranging from newborns to adolescents. The results showed that the presence of psychosocial risk factors is significantly associated with greater relative right frontal EEG asymmetry, with a combined effect size of $d = 0.36$. While this association was of comparable magnitude between studies investigating parental depression and child maltreatment, the effects appeared to be larger in samples with a larger percentage of girls. Frontal EEG asymmetry showed a considerably weaker and non-significant relation to internalizing symptoms ($d = 0.19$) and no significant association with externalizing symptoms ($d = 0.04$).

The meta-analysis on studies of infants and children exposed to different kinds of psychosocial risk supports the view of greater relative right frontal asymmetry as a relatively consistent indicator of the exposure to familial stressors in children. While the association appeared quite robust and no signs of a systematic publication bias were observed, the studies included in the analyses of psychosocial risk were largely underpowered, which may have increased the risk of false positive findings. The effects of psychosocial risk were moderated by gender in that samples with a larger percentage of girls were associated with larger effects, indicating that girls were more susceptible (i.e., showed more right frontal asymmetry) to the presence of psychosocial risk. The effect was however not significant when tested only within the depression studies, and it appears to have been driven in the full set of studies by the large effect observed in the female-only study by Miskovic et al. (2009): after leaving out this study in an additional meta-regression, the effect was no longer significant ($p > .23$). Inspection of the two child maltreatment studies (Curtis & Cicchetti, 2007;

Miskovic et al., 2009) does not provide an obvious answer as to why the two effect sizes from maltreated female participants were highly different in magnitude. Among the potential factors could be some of the sample-related variation between these studies, such as different ways of documenting maltreatment history (official records vs. self-report), age, SES, and ethnicity (predominantly African American in Curtis & Cicchetti, 2007; no information on ethnicity was provided by Miskovic et al., 2009). Nevertheless, the finding of potentially greater susceptibility of girls merits further investigation as it appears to argue against findings from other developmental domains indicating boys' greater vulnerability to adverse experiences (Ramchandani, Stein, Evans, & O'Connor, 2005; Sharp et al., 1995).

In addition, although observed only within studies having parental depression as the risk factor, the moderation of the effect sizes by age is interesting as it seems to indicate that the association between parental depression and right frontal asymmetry may attenuate with extended exposure to parental depression (i.e., older age). In infants, on the other hand, large effects were observed even in neonates with obviously minimal experience of interaction with a depressed caregiver. As the number of studies including older children is very limited, more research documenting the effects across various age groups is needed before conclusions about potential age differences in frontal asymmetry in response to parental depression and other psychosocial risk factors can be made.

Apart from the common conceptualization of children's frontal asymmetry as a marker of vulnerability to later psychopathology, Saby and Marshall (2012) pointed out that our understanding of the ontogenetic origins of frontal asymmetry variations remains limited and a developmental model of EEG asymmetry has not been constructed. The large effects observed in newborn infants and the concordance in

patterns of EEG asymmetry between newborns and their mothers (Field et al., 2004) could be taken to indicate a genetic transmission. Field and colleagues have suggested, however, that the neonatal effects may also emerge through intrauterine exposure to a depressed mother's biochemical imbalance affecting the levels of cortisol and serotonin, which may have consequences on fetal brain development (Field & Diego, 2008; Field, Diego, Dieter et al., 2004). Furthermore, the influence of natural variations in maternal caregiving quality on children's frontal EEG asymmetry in low-risk samples (Hane & Fox, 2006; Hane et al., 2010) provide support for the role of early interpersonal experiences with caregivers in shaping the pattern of frontal cortical asymmetries. There is clearly a need for more research investigating the malleability of children's frontal asymmetry in response to changes in the social environment, e.g., with intervention designs targeting parental caregiving behaviors.

The functional significance of variations in children's frontal EEG asymmetry is best understood by investigating its associations with emotional and behavioral outcomes. In the present meta-analyses, however, the hypotheses derived from the motivational approach/withdrawal models (Davidson, 1992; Harmon-Jones et al., 2010) linking greater right frontal asymmetry to a greater risk of internalizing symptoms and greater left frontal asymmetry to externalizing symptoms were not supported. In the set of externalizing studies, in particular, the effects were rather evenly distributed into positive, negative, and null effects, yielding a combined effect size close to zero. The association between right frontal asymmetry and internalizing behavior was stronger but not statistically significant. The effect sizes were also not significantly dependent on the time lag between the EEG recording and internalizing assessment, or assessment type (observed vs. reported). The behavioral outcome effects were moderated by gender in directions that correspond to the higher rates of

internalizing symptoms in girls (Sterba, Prinstein, & Cox, 2007) and externalizing symptoms in boys (Alink et al., 2006). Stronger associations between right frontal asymmetry and internalizing symptoms were thus observed in samples with larger percentage of girls and, conversely, left frontal asymmetry was more strongly related to externalizing symptoms in samples with larger percentage of boys.

One reason for the lack of direct associations between frontal asymmetry and the outcomes may be the relatively imprecise nature of the outcome measures employed in many of the studies. For example, the Child Behavior Checklist (CBCL; Achenbach & Rescorla, 2000) internalizing scale that was used in seven studies included in the set of internalizing studies consists of separate subscales assessing emotional reactivity, anxiety/depression, somatic complaints, and withdrawal. It is possible that these subdomains of internalization are differentially associated with withdrawal motivation and right frontal asymmetry, and the link between asymmetry and internalizing may be attenuated when using the global internalizing scale. Indeed, Shankman et al. (2013) recently showed that in adults, reductions in left frontal asymmetry were uniquely associated with depression but not panic disorder, likely reflecting a more tonic withdrawal motivation or reduced reward sensitivity associated with depression vs. anxiety. In a similar vein, future studies with children should be more detailed as to which specific facets of internalizing symptomatology the patterns of frontal asymmetry are associated with (e.g., depression, anxiety, or observed withdrawal behavior). Regarding externalizing symptoms, one yet unexplored avenue for testing the contribution of left frontal asymmetry to externalizing could be to investigate differences in frontal asymmetry in children who manifest antisocial behavior but differ in the presence or absence of callous-

unemotional traits, as the aggression of children high in callous-unemotional traits tends to be more proactive in nature (Frick & Viding, 2009).

In all three meta-analyses, SES was not a significant moderator of the effects. The lack of moderation in the psychosocial risk studies therefore does not support the hypothesis of Lusby et al. (2014) who argued that the fairly large effects observed in many previous studies of infants of depressed mothers (e.g., Diego et al., 2006; Field et al., 1995; Jones et al., 1998) may at least partly reflect the influence of other stressors associated with the low socioeconomic status of the families in these studies.

It is also potentially important to point out that the analytical procedures of EEG data in many studies included in these meta-analyses were not always optimal. For example, whole-head EEG was measured in many studies with rather small number of electrodes but nevertheless referenced offline to an average reference configuration, which may be associated with biased estimation of the underlying sources of electrical activity due to inadequate spatial sampling of electrodes (cf. Keil et al., 2014). Given the often poorer signal-to-noise ratio in the EEG of infants and small children, computation of the average reference from a low number of electrodes may be a greater issue of concern in children than adults. To date, no studies have reported EEG asymmetry from young children with high-density electrode montages which provide a more complete coverage of the scalp and thereby also diminish the risk of biases in the average reference computation apparent with a low number of electrodes (e.g., variation in impedance and signal quality, or differences in scalp location between homologous electrodes).

Finally, resonating the currently active discussion on power issues and replicability in psychological and neuroimaging research (Bakker, van Dijk, & Wicherts, 2012; Button et al., 2013), it may be a cause of concern that many of the

studies contributing to the combined effect size of psychosocial risk were highly underpowered, with the median power of the included studies being 0.39. While the median power exceeds the estimated typical power of 0.35 in psychology (Bakker et al., 2012) and 0.21 in neuroscience studies (Button et al., 2013), it is nevertheless considerably lower than the ideal threshold of 0.80. Not only do small sample sizes decrease the possibility of detecting true effects, but, more worryingly, they may inflate the estimated effect size of the observed group differences, leading to an increasing likelihood of false positive findings. The problems associated with low power become even more pressing when additional factors (e.g., gender) are included in the statistical tests. Therefore, to be able to estimate the true effect sizes for the influence of psychosocial risk on frontal asymmetry and the associations between frontal asymmetry, internalizing, and externalizing, studies with larger sample sizes (possibly through consortia integrating data from multiple sites) are needed.

Taken together, the present meta-analyses showed that while the pattern of greater relative right frontal asymmetry is a fairly consistent marker of the presence of familial stressors in children, the power of frontal asymmetry to directly predict internalizing and externalizing behaviors is modest. The functional role of frontal asymmetry in internalizing and externalizing may be more subtle and better understood as a moderator of the influence of the environment or child dispositions on behavioral outcomes. Indeed, studies taking such approach have indicated, for example, that greater relative left frontal asymmetry may mitigate the influence of maternal depression on children's internalizing symptoms (Lopez-Duran, Nusslock, George, & Kovacs, 2012) and greater relative right frontal asymmetry may exacerbate the influence of inhibited temperamental disposition on later internalizing problems (Fox et al., 1996). Frontal asymmetry thus appears to foster children's tendencies to

approach or withdraw, but the relation of these tendencies to emotional and behavioral outcomes may be critically dependent on the affective features of the environment or the children themselves. Important challenges for future studies include investigating the malleability of children's frontal asymmetry in response to changes in parental caregiving behaviors and associating patterns of frontal asymmetry to behavioral outcomes more closely associated with motivational tendencies to approach and withdraw.

Notes

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Table 1. Combined effect sizes and categorical moderators

	k	*N*	*d*	*95% CI*	*Q-W*	*Q-B*[a]
Psychosocial risk						
Total	20	1291	0.36**	0.15 – 0.58	62.73**	
SES						0.16
Low	12	704	0.32*	0.03 – 0.62	38.45**	
Middle/high	8	587	0.42*	0.07 – 0.76	23.83**	
Risk type						0.44
Depression	14	872	0.42**	0.14 – 0.69	36.10**	
Maltreatment/ institution	4	224	0.22	-0.30 – 0.73	20.22**	
Insensitive parenting	1	59	0.61*	0.08 – 1.13		
Alcohol dependence	1	136	0.00	-0.38 – 0.38		
Internalizing						
Total	20	1299	0.19	-0.03 – 0.41	56.93**	
SES						0.07
Low	6	518	0.24	-0.16 – 0.65	21.24**	
Middle/high	14	781	0.17	-0.10 – 0.45	35.12**	
EEG/outcome time lag						1.59
Concurrent	13	869	0.08	-0.19 – 0.35	37.12**	
Predictive	6	314	0.39	-0.01 – 0.79	9.73	
Outcome first	1	116	0.46*	0.04 – 0.88		
Outcome assessment type						0.44
Observed	4	259	0.35	-0.16 – 0.86	6.28	
Reported	16	1040	0.16	-0.09 – 0.41	49.18**	
Externalizing						
Total	10	810	0.04	-0.27 – 0.35	35.83**	
SES						0.10
Low	5	479	-0.01	-0.48 – 0.46	17.88**	
Middle/high	5	331	0.09	-0.39 – 0.57	17.88**	
EEG/outcome time lag						
Concurrent	8	711	0.02	-0.35 – 0.38	33.60**	
Predictive	2	99	0.16	-0.62 – 0.94	2.08	

*$p < .05$, **$p < .01$

k = number of study outcomes, *N* = total sample size, *d* = effect size (Cohen's *d*), 95% *CI* = 95% confidence interval around the point estimate of the effect size, *Q-W* = a statistic testing for the homogeneity within a set of studies, *Q-B* = a moderation statistic testing for the significance of the contrast between different sets of studies.

[a]Subgroups with k < 4 excluded from contrast. Note: Outcome assessment type contrast not shown for the externalizing set because none of the studies in this set provided data on observational measures of externalizing.

Table 2. Effect sizes and descriptive statistics for the set of psychosocial risk studies. The forest plot represents the individual effect sizes (Cohen's *d* with 95% confidence interval).

Study	***d***	***p***	***N***	**Age**
Bruder 2007	-0.81	.02	35	13.20
Curtis 2007 (females)	0.00	1.00	43	10.30
Curtis 2007 (males)	-0.63	.04	44	10.30
Dawson 1992	0.00	1.00	27	1.18
Dawson 1997	0.54	.00	117	1.15
Diego 2006	1.12	.00	66	0.18
Ehlers 2001	0.00	1.00	136	10.50
Field 1995	0.74	.04	32	0.40
Field 1998	-0.75	.08	24	0.30
Field 2004	0.62	.00	119	0.01
Hane 2006	0.61	.02	59	0.75
Jones 1997a	0.63	.05	41	0.08
Jones 1998	0.86	.00	58	0.02
Jones 2001	0.78	.02	38	0.84
Lopez-Duran 2012	0.19	.30	135	7.65
McLaughlin 2011	0.24	.41	76	3.50
Miskovic 2009	1.22	.00	61	14.24
Tomarken 2004	0.59	.09	38	13.00
Jones 2004	0.63	.01	78	0.16
Lusby 2014	0.22	.39	64	0.38
Total	**0.36**	**.00**	**1291**	**4.41**

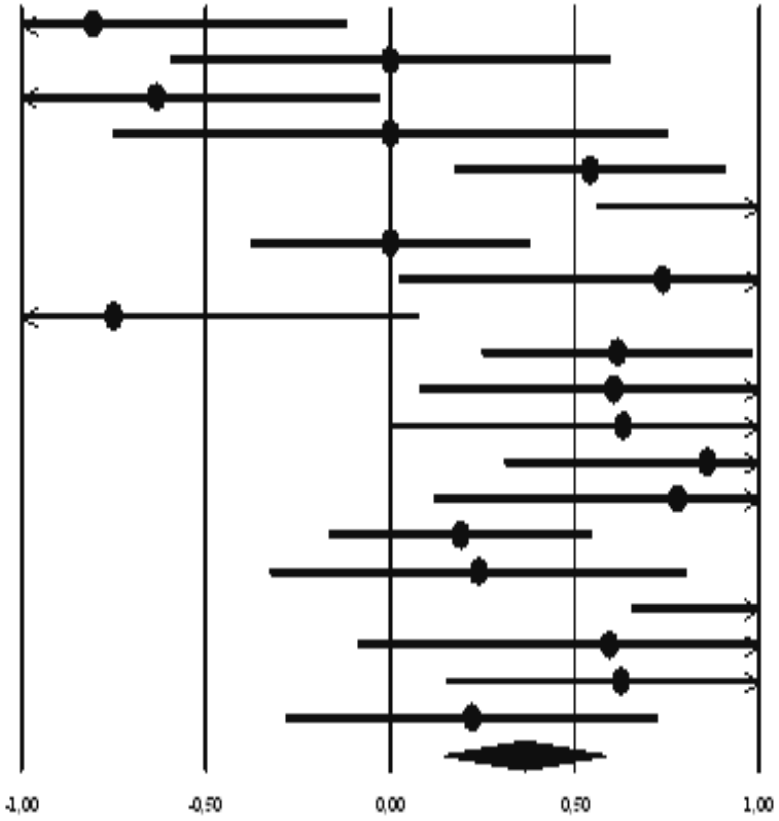

Table 3. Effect sizes and descriptive statistics for the set of internalizing studies. The forest plot represents the individual effect sizes (Cohen's *d* with 95% confidence interval).

Study	***d***	***p***	***N***	**Age**
Ehlers 2001	0.00	1.00	136	10.50
Lopez-Duran 2012	-0.18	.30	135	7.65
McLaughlin 2011	0.48	.05	76	4.50
Baving 2002 (females)	0.81	.01	47	8.00
Baving 2002 (males)	-1.05	.01	35	8.00
Buss 2003	-0.20	.58	31	0.50
Forbes 2006	-0.50	.04	74	5.08
Fox 1996	0.00	1.00	96	4.56
Gatzke-Kopp 2014	0.24	.09	209	6.03
Hannesdóttir 2006	-0.90	.14	16	4.50
Hayden 2008	0.20	.66	22	6.16
Henderson 2001	0.24	.25	97	0.75
Jones 1997b	1.81	.02	15	3.00
Kentgen 2000	0.16	.74	18	15.50
Pössel 2008	0.90	.00	80	13.92
Schmidt 2008	1.46	.00	20	0.75
Schmidt 1999	0.28	.57	17	7.00
Smith 2010	0.70	.14	23	1.42
Theall-Honey 2006	0.00	1.00	36	4.50
McManis 2002	0.46	.03	116	11.00
Total	**0.19**	**.08**	**1299**	**6.17**

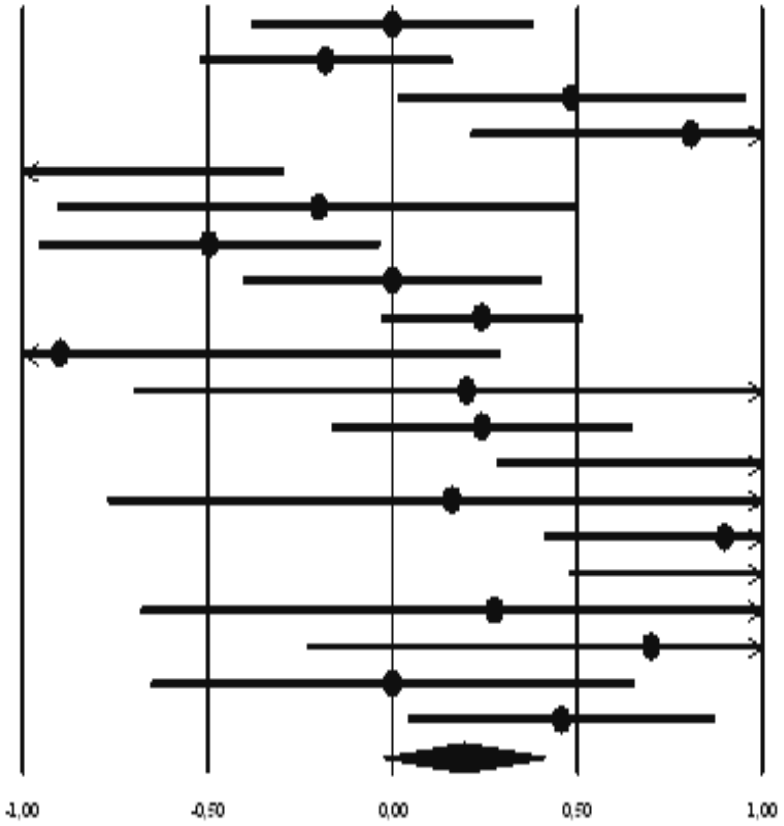

Table 4. Effect sizes and descriptive statistics for the set of externalizing studies. The forest plot represents the individual effect sizes (Cohen's *d* with 95% confidence interval).

Study	***d***	***p***	***N***	**Age**
Ehlers 2001	0.00	1.00	136	10.50
Hane 2010	0.03	.89	98	3.00
McLaughlin 2011	-0.13	.59	76	4.50
Baving 2000 (females)	-1.21	.00	33	8
Baving 2000 (males)	0.92	.03	25	8
Forbes 2006	0.77	.00	74	5.08
Fox 1996	0.00	1.00	96	4.56
Gatzke-Kopp 2014	0.28	.04	209	6.03
Santesso 2006	-0.98	.01	40	10.10
Smith 2010	0.63	.18	23	1.42
Total	**0.04**	**.79**	**810**	**6.12**

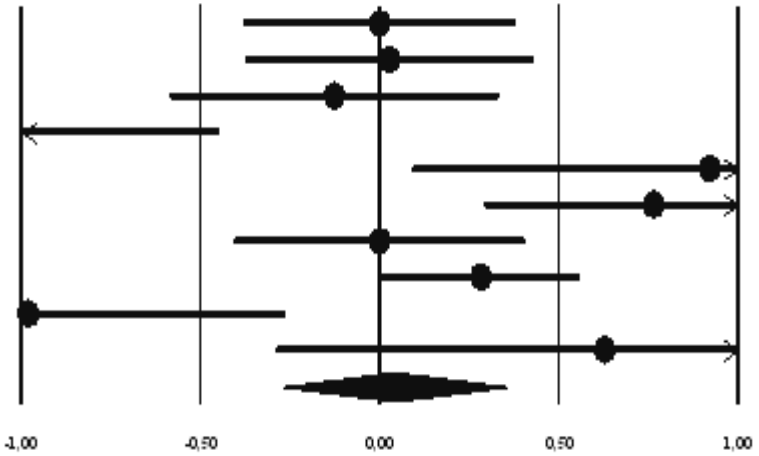

Figure Captions

Figure 1. A flow chart of the study selection process.

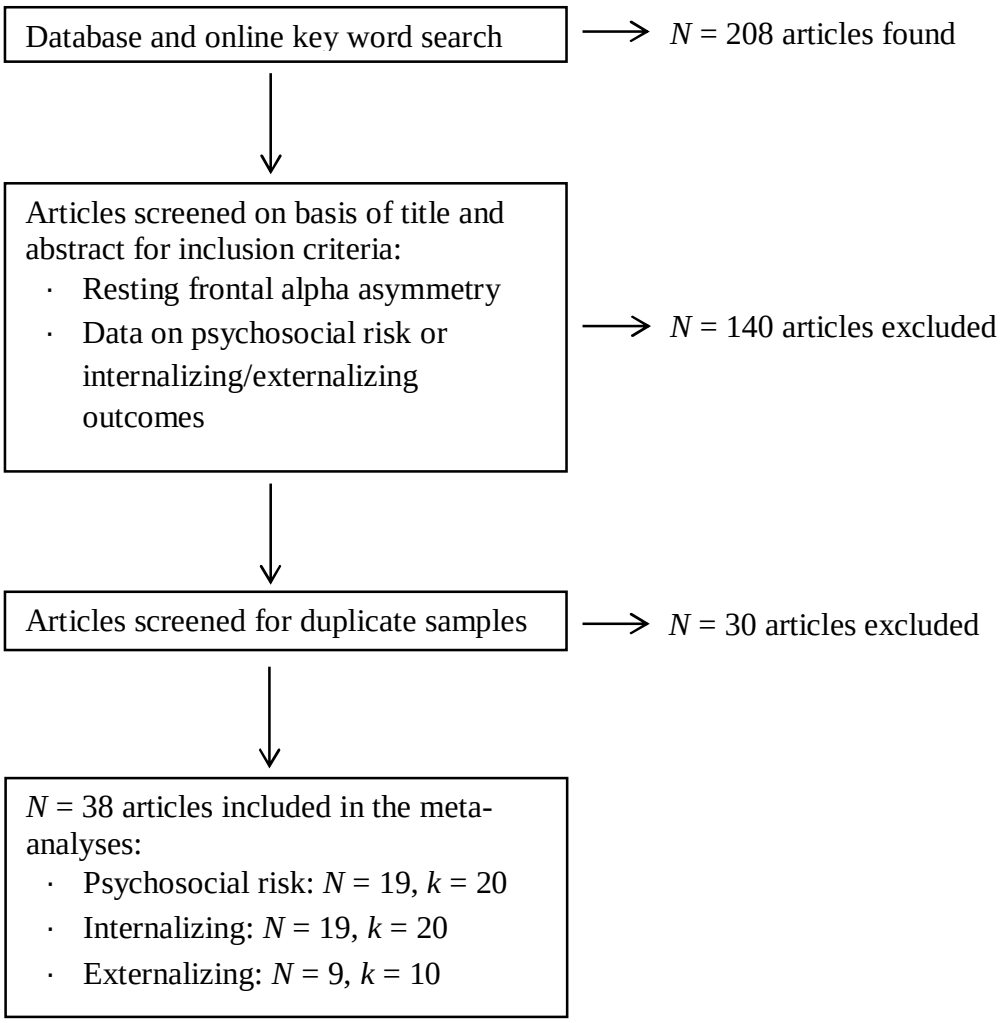
Database and online key word search
N = 208 articles found
Articles screened on basis of title and abstract for inclusion criteria:
· Resting frontal alpha asymmetry
· Data on psychosocial risk or internalizing/externalizing outcomes
N = 140 articles excluded
Articles screened for duplicate samples
N = 30 articles excluded
N = 38 articles included in the meta-analyses:
· Psychosocial risk: N = 19, k = 20
· Internalizing: N = 19, k = 20
· Externalizing: N = 9, k = 10

Figure 1.