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Childhood EEG frontal alpha power as a predictor of adolescent antisocial behavior: A twin heritability study

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ABSTRACT

High EEG frontal alpha power (FAP) is thought to represent a state of low arousal in the brain, which has been related in past research to antisocial behavior (ASB). We investigated a longitudinal sample of 900 twins in two assessments in late childhood and mid-adolescence to verify whether relationships exist between FAP and both aggressive and nonaggressive ASB. ASB was measured by the Child Behavioral Checklist, and FAP was calculated using connectivity analysis methods that used principal components analysis to derive power of the most dominant frontal activation. Significant positive predictive relationships emerged in males between childhood FAP and adolescent aggressive ASB using multilevel mixed modeling. No concurrent relationships were found. Using bivariate biometric twin modeling analysis, the relationship between childhood FAP and adolescent aggressive ASB in males was found to be entirely due to genetic factors, which were correlated $r = 0.22$.

1. Introduction

In the 1970s, a theory of antisocial behavior (ASB) emerged that is referred to as the Slow Arousal Theory. Posited by Robert Hare, a researcher of psychopathy, this theory sought to explain several findings of low arousal levels in individuals prone to crime and violence. Early research witnessed increased levels of slow-wave brain waves, including theta (4–8 Hz) and delta (1–4 Hz) in the adult brains of incarcerated psychopaths (Ellingson, 1954) and violent criminals (Hill, 1952). Since these early studies, lower prefrontal activation has been found in males with past aggressive behavior (Volkow et al., 1995), in 9 males and 1 female with repetitive violent behavior (Critchley et al., 2000), and in violent psychiatric inpatients (Kuruoglu, Arikan, Karatas, Arac, & Isik, 1996). In examining these findings, the Slow Arousal Theory suggests a 'stimulus hunger' in brains marked by slow-wave activity. A stimulus hungry and poorly aroused brain, it is suggested, may require risky, impulsive, or other 'high excitement' external stimuli to achieve the arousal levels that a normally aroused brain typically experiences. The theory that ASB often relates to difficulty in inhibiting behavior is highly prevalent and longstanding (Eysenek, 1964; Gray, 1972,1987), although ASB can also stem from failure to correctly estimate punishment (Gray, 1972). Impulsive and risk-taking behavior, as well as the ability to evaluate reward or punishment, are frequently thought to be related to the prefrontal cortex, which is also related to behavioral inhibition. This may explain higher propensity toward violent, delinquent or criminal activity in adolescence, when the frontal lobes are not yet fully developed. The frontal region thus appears to be a fruitful area for research on the prediction of ASB.

In children, slow arousal as marked by high alpha power, has also been predictive of criminal activity. EEG alpha power, typically measured as 8–13 Hz in adults and 8–10.5 Hz in children of the age examined in the present research (Gasser, Verleger, Bacher, & Sroka, 1988), is representative of a sleepy quality. In children, increased cortical alpha power (a marker of slow arousal) has been associated with later crime (Mednick, Volavka, Gabrielli, & Itil, 1981; Petersén, Matousek, Mednick, Volavka, & Pollock, 1982). In a group

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of 24-year-old male criminals, retrospective analysis found high alpha power at age 15 years (Raine, Venables, & Williams, 1990). Reduced frontal activation was also found in children with oppositional defiant disorder, using single-photon emission computerized topography (SPECT; Amen & Carmichael, 1997). The majority of this research, both in adults and in children, was conducted with male subjects, and an aim of the present research is to investigate these relationships in females as well.

One important question that has arisen in research of ASB is that of differences between aggressive and nonaggressive ASB. Nonaggressive ASB is also referred to as delinquency or rule-breaking behavior, and is captured by the Delinquency scale of the Child Behavior Checklist (CBCL; Achenbach, 1991), which is the instrument used in this research.

Aggressive and nonaggressive ASB both show heritability, with a recent metaanalysis estimating 65% heritability for aggressive ASB and 48% for nonaggressive ASB (Burt, 2009). Alpha power has also shown high heritability, ranging from 63 to 89% in children and adolescents (Van Baal, De Geus, & Boomsma, 1996; van Beijsterveldt, Molenaar, de Geus, & Boomsma, 1996). In our sample, full range frontal alpha power (FAP) at 8–13 Hz was found to have 71–85% genetic influence, with the rest of the variance accounted for by non-shared environmental factors (Gao, Tuvblad, Raine, Lozano, & Baker, 2009). However, no past study has examined potential genetic correlations between FAP and any form of ASB, of either aggressive or nonaggressive nature. We hypothesized that a significant genetic correlation would emerge to explain the phenotypic relationship between the FAP and both forms of ASB, as all variables have been found to have genetic roots.

2. Methods

2.1. University of Southern California (USC) study of risk factors for ASB (RFAB)

The data in the present research are drawn from RFAB, a longitudinal project currently in its fifth wave of data collection, which focuses on genetic, environmental, social, and biological influences on the development of ASB. The twins and their families were recruited from the greater Los Angeles area, and are ethnically and socioeconomically representative of the region in terms of parental occupation and income, education levels, race, and ethnicity (Baker et al., 2013). The present research uses data from Waves 1 and 3, when twins were 9–10 and 14–15 years old, respectively. EEG data were only collected in Wave 1 (605 families; $N = 1219$ twins/triplets). Caregivers (91.4% biological mothers) were administered the Child Behavior Checklist (CBCL) in both Waves 1 and 3. In accordance with past research of EEG (Baving, Laucht, & Schmidt, 2003; Deckel, Hesselbrock, & Bauer, 1996), only right-handed twins were included (i.e., 169 left-handed participants were excluded from the present analyses). In total, 431 males and 469 females provided EEG data, all of whom also provided Wave 1 ASB data, and 311 and 378 of whom, respectively, also provided Wave 3 ASB data. The number of participants used in longitudinal analyses, not counting single twins, was 383 (90 MZ male, 105 MZ female, 47 DZ males, 54 DZ females, 87 opposite sex). Attrition analyses found no significant predictors for discontinuation for socioeconomic, gender, language, or ASB factors (Baker et al., 2013). Zygosity was determined with microsatellite DNA concordance analysis for over 87% of pairs, and a twin similarity questionnaire for inconclusive samples (usually due to poor signal quality). No significant mean differences were found between MZ and DZ twins for age, FAP, or ASB.

2.2. Aggressive and nonaggressive ASB, Waves 1 and 3, parent-ratings

The CBCL measures a wide range of behavior problems in children over the prior 6 months. This instrument has 113 items that use a three-point scale (0 for not true, 1 for sometimes true, and 2 for very or often true). The Aggressive ASB subscale consists of 13 items and includes behaviors such as arguing, fighting with other children, and bullying others. The internal consistencies in Waves 1 and 3 were found to be 0.61 and 0.71, respectively. The nonaggressive ASB subscale (CBCL Delinquency) consists of 20 items gauging cheating, lying, petty crimes, and other nonaggressive ASB activities, and showed internal consistencies of 0.88 and 0.89 at Waves 1 and 3, respectively. In this sample, the highest count of data for the CBCL were available from Waves 1 and 3 with approximately 600 pairs of twins participating in each, and only 205 pairs participating in Wave 2. For sample consistency, Waves 1 and 3 were included in the analysis.

2.3. EEG collection and processing, Wave 1

First, a 3-min rest period baseline of activity was obtained prior to any task completion after which the twin participated in approximately 1.5–2 h of tasks. In this condition, the children kept their eyes open and paid attention to a blue cross on a computer screen, as it was observed that 9–10 year old participants had trouble keeping eyes closed for three full minutes. The rest period was selected as it likely represented the most naïve brain activity before a series of tasks.

EEG data were collected from 30 scalp sites and left and right mastoids based on the 10–20 international system (Jasper, 1958) using James Long Inc. amplification system (Caroga Lake, NY). A lycra Electro-Cap (Eaton, OH) was used in the data collection. Anterior midline site (AFz) was the ground electrode whereas the central midline site (Cz) served as the reference site. The scalp recording sites were prepared with conductive abrasive, which aids in increasing conductivity. Impedances were kept below 10 KOhms. To account for eye blink artifacts, a bipolar electrooculogram (EOG) channel was recorded from surrounding (above and below the supra and infra-orbital eye ridges of the left eye). Tester comments and observations, as well as unusual datum values, were taken into account in evaluating the validity of each participant's data for inclusion in data analysis. The majority of manually excluded cases were excluded for tester-reported excessive movements and fidgetiness. Artifact removal was initially performed by hand to remove eye or muscle movement, and later again with the addition of MatLab artifact removal algorithms.

2.4. Derivation of FAP

Data from selected electrodes, nine frontal and prefrontal electrodes: Fp1, Af3, F7, F3, Fz, F4, F8, Af4, Fp2, were filtered into the low alpha band (8–10.5 Hz), appropriate for analysis of resting-state EEG recordings from children aged 9–10 years. This range was selected due both to past research that found alpha to reside in such ranges in children (Gasser et al., 1988) and because many children show delayed alpha quickening, which raises the risk of targeting Beta waves with a higher frequency. We were specifically interested in the behavioral disinhibition that stems from excessive alpha. The FAP is estimated via a weighted sum of this data across electrodes. The weights were calculated via principal components analysis (PCA), which attempts to maximize the power in the weighted sum, given that the weights squared sum to 1 (that is, they are normalized). Thus, the FAP analyzed is the strongest average power over all possible normalized weightings of the frontal electrode data.

2.5. Statistical analyses

Data were visualized, cleaned and analyzed for descriptive statistics and phenotypic correlations in Statistical Analysis Software (SAS 9.1.3, 2005). Mixed modeling regression was also conducted in SAS, using family ID to nest twins, taking into account non-independent observations between family members. Outcome variables were aggressive and nonaggressive ASB in Waves 1 and 3.

2.6. Genetic analyses

ASB and EEG data were transformed using Blom Normalization option to reduce skewness in the ASB data. Analyses were conducted using raw data in Mx GUI (Neale, Boker, Xie, & Maes, 2003), a structural equation-modeling program specialized for twin data. Genetic model comparison in Mx relies on raw maximum-likelihood estimation procedures, which yield a likelihood ratio statistic (−2LL) based on differences between observed and expected values. Calculating the difference between two models' −2LL values yields a chi-square (χ^2) test of significance with degrees of freedom (df) equal to the differences in df between the two compared models (Neale & Cardon, 1992). All models were compared to a baseline saturated model which freely estimates means, variances and covariance for each zygosity group. Model fit was also assessed by comparing the Akaike's Information Criterion (AIC; Akaike, 1987). Lower values of AIC indicate more parsimonious fit and a better model.

Bivariate Cholesky decomposition models were used to determine proportions of A (additive genetic), C (shared environmental), and E (nonshared environmental) influences on FAP and aggressive ASB or nonaggressive ASB separately as well as the shared A, C, and E influences between them. This model uses covariances between twins for given traits and across traits, hence partitioning the influences that are unique or shared between variables. Different models were examined to verify whether sexes could be equated, and whether types of influences (e.g., shared environment) could be dropped to explain observed results most parsimoniously. Non-additive genetic effects (i.e. due to dominance or epistasis) were also tested and found not to explain the data as well.

3. Results

Males showed higher aggressive ASB levels than females in both Wave 1 ($t = 5.08$, df = 887, $p = 0.02$) and Wave 3 ($t = 2.36$, df = 596, $p = 0.02$). Nonaggressive ASB was higher in males than in females in Wave 1 ($t = 3.975$, df = 887, $p < 0.01$), but not Wave 3 ($t = 1.21$,