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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 $r = 0.22$.

Keywords

EEG; Aggression; Frontal alpha power Twins; Heritability

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

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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 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, df = 596, p = 0.23). No significant sex differences emerged in FAP. Mean levels of aggressive ASB in Wave 1 were 6.12 (SD = 5.16) in males and 5.10 (SD = 4.78) in females, and in Wave 3 were 5.35 (SD = 6.00) in males and 4.32 (SD = 4.80) in females. Mean levels of nonaggressive ASB were 1.49 (SD = 1.81) in males and 1.06 (SD = 1.36) in females in Wave 1, and 1.79 (SD = 2.34) in males and 1.55 (SD = 2.52) in females in Wave 3. Mixed Model repeated measures analyses of variance revealed that aggressive ASB decreased between Waves 1 and 3 ($F_{(1,179)}$ = 8.16, $p < 0.01$), and nonaggressive ASB increased ($F_{(1,179)}$ = 22.12, $p < 0.01$), with no significant interactions with sex.

Mixed model regressions of ASB on FAP conducted with both sexes did not yield significant regression coefficients in Wave 1 for either aggressive or non-aggressive ASB. In Wave 3, significant main effects of FAP were deemed not interpretable due to significant interactions of sex with FAP. When the two sexes were analyzed separately, FAP was significantly predictive of aggressive ASB in Wave 3 for males (Beta = 0.99, SE = 0.44, p = 0.03) but not females (Beta = 0.31, SE = 0.24, p = 0.26). FAP did not significantly predict Wave 3 nonaggressive ASB in males (Beta = 0.05, SE = 0.17, p = 0.76), or in females (Beta = −0.18, SE = 0.12, p = 0.09).

Twin and cross-twin cross-trait correlations are presented in Table 1 for the two variables that were found to be phenotypically related – FAP and Wave 3 aggressive ASB. Twin correlations are consistently higher in the MZ than in the DZ twins, suggesting heritability. Similarly, the cross-trait correlations were slightly higher in the male MZ twins than in the male DZ twins, although no differences were found between MZ and DZ females, suggesting genetic overlap between FAP and aggressive ASB in males but not females.

Genetic models were only explored for the relationship that demonstrated significant phenotypic relationships – namely, FAP with Wave 3 male aggressive ASB. Model fits and comparisons are shown in Table 2. Comparing models 1 and 1a demonstrates that equating the estimates on the two sexes significantly worsens model fit, and so sexes were estimated separately. Comparing Models 1 and 2 demonstrates that dropping C improved model fit, suggesting that shared environment was not significant in any of these relationships. Finally, dropping E correlations provided the best fit to the data (Models 9). Bivariate genetic analysis found that influences on FAP are 78% A (raw variance 0.88^2) and 22% E (raw variance 0.47^2) in males. Influences on Wave 3 aggressive ASB were found to be 65% A (raw variance 0.81^2) and 35% E (raw variance 0.59^2) in the males. The covariation between FAP and male Wave 3 aggressive ASB was accounted for exclusively by significant genetic correlation at Rg = 0.22 (95% CI 0.16–0.30).

Although the models investigated included both males and females, and estimated influences on them separately, Fig. 1 depicts only the relationships that demonstrated significant bivariate relationships between FAP and ASB, for which the 95% CI did not include zero. Female FAP was not significantly related to aggressive ASB in either wave through bivariate genetic or environmental influences. These findings are consistent with phenotypic mixed modeling results. This figure presents squared standardized estimates of the influences.

4. Discussion

This study sought to investigate the phenotypic and genetic relationships between frontal alpha power and antisocial behavior, both aggressive and nonaggressive forms, in childhood and adolescence. It emerged that in males but not females, frontal alpha power at the age of 9–10 years was predictive of aggressive behavior at the ages of 14–15 years. No significant concurrent relationships emerged in either sex, and no relationships emerged with nonaggressive antisocial behavior, which largely reflects rule-breaking tendencies. Frontal power and aggressive behavior both showed high heritability and no shared environmental influences. Significant genetic correlations emerged in the males but not females between aggressive behavior and frontal alpha power. Although these genetic correlations were modest, they constituted 100% of the influences accounting for the phenotypic relationship between frontal alpha power and antisocial behavior.

These findings are novel in the literature of developmental psychopathology. While past studies found links between alpha power and criminality (Kuruoglu et al., 1996; Mednick et al., 1981; Raine et al., 1990), this relationship had never been demonstrated in a community sample. These findings suggest that high FAP, representing slow wave activity, is predictive of male aggressive behavior in the general population. Most importantly, the majority of past research examined male participants exclusively, usually using clinical or criminal populations, or sons of offenders. This is one of the first examinations of female neural arousal and its relationship to ASB, and hence the results are of considerable interest as they suggest that past findings may be significant exclusively within males. Lastly, this was the first demonstration of joint genetic etiology to frontal alpha power and aggressive behavior.

The genetic findings that emerged in this research are novel. While the heritability of aggressive behavior and the heritability of alpha power have been demonstrated in past research, as well as the phenotypic relationship between them, the genetics of that relationship had not been explored. Our findings suggest very moderate but significant heritability overlap to these two variables. This suggests that while some of the same genes that contribute to frontal alpha power are those that determine aggressive behavior in adolescents, there are many genetic influences that do not overlap. It would be beneficial for researchers to continue exploring biological markers that could account for a greater proportion of genetic influence on aggressive behavior.

One notable finding is that childhood frontal alpha power was not correlated with concurrent childhood aggressive behavior, but was significantly correlated with adolescent aggressive behavior. This may imply that frontal alpha power is a pre-dispositional marker of children likely to develop more aggressive behavior response patterns in the future. It may also imply that frontal alpha power is more strongly related to a qualitatively different form of aggressive behavior that emerges in adolescence over childhood. As EEG data from Wave 3 were not collected, it is not possible to say whether concurrent relationships in adolescence would have emerged.

Inarguably, the barrier to antisocial behavior is higher for females than for males. Female aggression is both rarer, uniformly and consistently across studies, and may be regarded with less social acceptance (Bjorkvist, 1994). This may explain why a low-arousal brain in the males may default to thrill or risk-seeking behavior but not so in the females. It is also theorized by some that female aggression regularly takes alternative forms. In recent years, attention has been paid to the concept of ‘relational aggressive ASB, which comprises excluding others from social groups, gossiping, purposely withdrawing friendship, and other forms of indirect and non-physical attacks (Crick & Grotpeter, 1996). It is possible that the absence of correlation with aggressive behavior that is observed in these results fails to capture aggressive behavior as it is most pertinent to females in mid-adolescence. Future research may benefit from exploring relationships between frontal alpha power and relational aggressive behavior in females.

While aggressive behavior was associated with lower arousal, the same was not true for nonaggressive antisocial behavior. This pattern of results may provide further support to the distinction between aggressive and nonaggressive antisocial behavior. Much past research concerning slow-arousal has focused on violent offending, which is a subset of aggression. Nonaggressive antisocial behavior, e.g., rule-breaking, may in fact stem from neural mechanisms that are distinct from low arousal. This finding should be further investigated in other samples for the purpose of replication.

Two additional configurations of electrodes were tested: five frontal electrodes (F7, F3, Fz, F4, F8) and three frontal regions (F3, Fz, F4) without prefrontal electrodes. Genetic correlations between frontal alpha power and male Wave 3 aggressive ASB were significant using five but not three electrodes, but were slightly lower (Rg = 0.17, $p < 0.05$). Neither of the other configurations showed significant correlations with female ASB in either wave. This suggests that that inclusion of prefrontal electrodes is beneficial for prediction of

aggressive behavior. It should be noted that when activation was simply averaged across electrodes rather than calculated using PCA weighting, correlations were significant and higher than those reported here ($r = 0.30$, $p < 0.01$ in males; $r = -0.19$, $p = 0.02$ in females). However, using this evenly weighted sum may not be as able to find the strongest activations in the frontal area. Hence, we believe that the PCA method of calculating alpha power is more accurately reflective of frontal activation. Also, the weighting procedure derived from PCA may be less likely to inflate correlations between EEG and behavior as opposed to an even weighting, because the PCA procedure tries to reduce the effects of noise from other regions or the EEG equipment. In addition, frontal asymmetry was also tested for relationship with aggressive behavior, and was not found to correlate significantly with behavior. Despite great interest in frontal asymmetry over the recent decades, our findings suggest that overall frontal power may be more predictive of aggressive behavior.

Slow Arousal Theory is further informed by another prominent and influential theory of the past few decades, the somatic marker hypothesis (Damasio, Tranel, & Damasio, 1991). This theory stemmed from a set of findings that showed that in most individuals, risky actions are accompanied by a somatic nervousness, typically measured with skin conductance response. In psychopathic individuals, however, this somatic response was absent, leading to the suggestion that psychopathic individuals experience lower arousal, as well as an absence of discomfort that prevents most individuals from taking risky actions. In a recent project conducted with the present twin sample, it was found that males with reduced orienting response to novelty, as measured by skin conductance, showed higher levels of psychopathy (Tuvblad, Gao, Niv, Raine, & Baker, 2011). In these analyses, children who had persistently high or persistently low skin conductance response over the course of three waves of data collection were compared; boys with persistently low skin conductance orienting response also showed significantly higher psychopathy scores in adolescence, suggesting some longitudinal stability. This analogous finding lends support to the gender difference explanation rather than necessarily differences between aggressive and nonaggressive behavior, as the aggressive behavior instrument in these analyses was the Child Psychopathy Scale (Lynam, 1997) a measure of personality rather than aggressive behavior. However, because both of these findings emerged in the same sample, it is crucially important for large twin studies other than the present study to replicate these promising findings.

Lastly, this research has mechanistic implications for antisocial behavior. A question to consider in future research is the directionality of the relationship, and perhaps whether the two are commonly influenced by a third variable. While Slow Arousal Theory suggests that a stimulus hunger is at the core of the tendency toward stimulation seeking, in this case through violence, it may instead be that aggressive behavior affects mental stimulation, leading to less frontal activity. It is also possible that other factors, such as neurotransmitter prevalence and binding properties, affect both phenotypes. If the mechanisms of this relationship were discerned, treatment implications would emerge for methods such as neurofeedback, meditation, or medication.

In conclusion, this paper found a small but significant genetic correlation between frontal alpha power in childhood, and aggressive behavior in adolescence. There are a few important conclusions to draw here. First, these findings suggest that childhood brain

patterns are a potentially useful marker for identify children who are susceptible to difficulties later in their lives, and may serve as useful tools for early detection. Second, the heritable underpinnings of this relationship are a useful first step in the search for genetic origins of certain aggressive behaviors. Third, there are differences between the biological bases of male and female aggression, which may explain why they manifest in different forms. On the whole, this research helps us elucidate a few more important points about the origins of antisocial behavior.

References

Achenbach, TM. Manual for the Child Behavior Checklist/4–18 and 1991 profile. Burlington, VT: University of Vermont Department of Psychiatry; 1991.

Akaike AC. Factor analysis and AIC. Psychometrika. 1987; 52:317–332.

Amen DG, Carmichael BD. High-resolution brain SPECT imaging in ADHD. Annals of Clinical Psychiatry. 1997; 9:81–86.

Baker LA, Tuvblad C, Wang P, Gomez K, Bezdjian S, Niv S, et al. The Southern California Twin Register at the University of Southern California: III. Twin Research. 2013; 16:336–343.

Baving L, Laucht M, Schmidt MH. Frontal EEG correlates of externalizing spectrum behaviors. Eur Child Adolesc Psychiatry. 2003; 42:36–42. [PubMed: 12601563]

Bjorkvist K. Sex differences in physical, verbal, and indirect Aggression: A review of recent research. Sex Roles. 1994; 30:177–188.

Burt SA. Are there meaningful etiological differences within antisocial behavior? Results of a meta-analysis. Clinical Psychology Review. 2009; 2:163–178. [PubMed: 19193479]

Crick NR, Grotpeter JK. Children's treatment by peers: Victims of relational and over aggression. Developmental Psychopathology. 1996; 8:267–280.

Critchley HD, Simmons A, Daly EM, Russell A, van Amelsvoort T, Robertson DM, et al. Prefrontal and medial temporal correlates of repetitive violence to self and others. Biological Psychiatry. 2000; 47:928–934. [PubMed: 10807966]

Damasio, AR.; Tranel, D.; Damasio, H. Somatic markers and the guidance of behavior. In: Levin, H.; Eisenberg, H.; Benton, A., editors. Frontal lobe function and dysfunction. New York: Oxford University Press; 1991. p. 217-228.

Deckel AW, Hesselbrock V, Bauer L. Antisocial personality disorder, childhood delinquency, and frontal brain functioning: EEG and neuropsychological findings. J Clinical Psychology. 1996; 52:639–650.

Ellingson RJ. The incidence of EEG abnormality among patients with mental disorders of apparently nonorganic origin: A critical review. American Journal of Psychiatry. 1954; 111(4):263–275. [PubMed: 13197589]

Eysenek, HJ. Crime and personality. London: Routledge and Kegan Paul; 1964.

Gao Y, Tuvblad C, Raine A, Lozano DI, Baker LA. Genetic and environmental influences on frontal EEG asymmetry and alpha power in 9–10-year-old twins. Psychophysiology. 2009; 46(4):787–796. [PubMed: 19386046]

Gasser T, Verleger R, Bacher P, Sroka L. Development of the EEG of school age children and adolescents. I. Analysis of band power. Electroencephalography and Clinical Neurophysiology. 1988; 69:91–99. [PubMed: 2446839]

Gray, JA. The psychophysiological basis of introversion-extraversion: A modification of Eysenck's theory. In: Nebylitsyn, VD.; Gray, JA., editors. The biological bases of individual behavior. San Diego, CA: Academic Press; 1972. p. 182-205.

Gray, JA. The Psychology of Fear and Stress. New York: Cambridge University Press; 1987.

Hill D. EEG in episodic psychotic and psychopathic behaviour: A classification of data. Electroencephalography and Clinical Neurophysiology. 1952; 4(4):419–442. [PubMed: 12998591]

Jasper HH. The 10–20 electrode system of the International Federation. Electroencephalography and Clinical Neurophysiology. 1958; 10:371–437.

Kuruoglu AC, Arikan Z, Karatas M, Arac M, Isik E. Single photon emission computerized tomography in chronic alcoholism: Antisocial personality disorder may be associated with decreased frontal perfusion. British Journal of Psychiatry. 1996; 16:348–354. [PubMed: 8879722]

Lynam DR. Pursuing the psychopath: Capturing the fledgling psychopath in a nomological net. Journal of Abnormal Psychology. 1997; 106:425–438.

Mednick SA, Volavka J, Gabrielli WF, Itil TM. EEG as a predictor of ASB. Criminology. 1981; 19:219–229.

Neale, MC.; Boker, SM.; Xie, G.; Maes, H. Mx: Statistical modeling. Richmond, VA: Department of Psychiatry, Medical College of Virginia; 2003.

Neale, MC.; Cardon, LR. Methodology for genetic studies of twins and families. Dordrecht, The Netherlands: Kluwer Academic Publications; 1992.

Petersén KG, Matousek M, Mednick SA, Volavka J, Pollock V. EEG antecedents of thievery. Acta Psychiatrica Scandinavica. 1982; 65(5):331–338. [PubMed: 7113731]

Raine A, Venables PH, Williams M. Relationships between central and autonomic measures of arousal at age 15 years and criminality at age 24 years. Archives of General Psychiatry. 1990; 47(11): 1003–1007. [PubMed: 2241502]

Tuvblad C, Gao Y, Niv S, Raine A, Baker LA. The skin conductance orienting response and psychopathic personality: A longitudinal twin study. Biol Psychol. 2011; 89:47–53. [PubMed: 21945549]

Van Baal GC, De Geus EJ, Boomsma DI. Genetic architecture of EEG power spectra in early life. Electroencephalography and Clinical Neurophysiology. 1996; 98(6):502–514. [PubMed: 8763510]

van Beijsterveldt CE, Molenaar PC, de Geus EJ, Boomsma DI. Heritability of human brain functioning as assessed by electroencephalography. American Journal of Human Genetics. 1996; 58(3):562–573. [PubMed: 8644716]

Volkow ND, Tancredi LR, Grant C, Gillespie H, Valentine A, Mullani N, et al. Brain glucose metabolism in violent psychiatric patients: A preliminary study. Psychiatry Research. 1995; 61:243–253. [PubMed: 8748468]

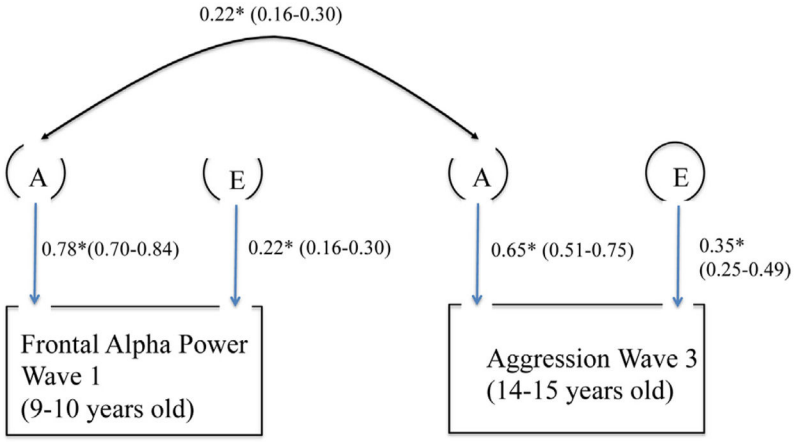

Fig. 1.

Bivariate heritability model of Wave 1 EEG alpha power with Wave 3 aggressive ASB in males. This figure demonstrates the small but significant genetic correlation between frontal alpha power at the age of 9–10 years and aggression at the age of 14–15 years. No shared environmental factors emerge as significant for either variable, and no environmental correlation emerges as significant to the relationship between them. The genetic correlation is significant only in the males.

Table 1

Twin and cross-twin cross-trait correlations for FAP and Wave 3 aggressive ASB by zygosity.

	Mz male		Mz female		Dz male		Dz female		Dz opposite	
	FAP	**Agg-3**	**FAP**	**Agg-3**	**FAP**	**Agg-3**	**FAP**	**Agg-3**	**FAP**	**Agg-3**
FAP	**0.73***	–	**0.64***	–	**0.60***	–	**0.54***	–	**0.37***	–
Agg-3	0.22*	**0.74***	−0.07	**0.72***	−.0.06	**0.11**	−0.10	**0.42***	0.05	**0.52***

Note. Bolded values represent twin correlations, and non-bolded values cross-twin cross-trait correlations.

* $p < 0.05$.

Table 2

Bivariate model fit indices for FAP and aggressive ASB in Wave 3.

Model	Likelihood values				Overall fit		Model comparison		
	−2LL	DF	AIC	BIC	χ^2 (Δdf)	*p*	Compared to Model	$\Delta\chi^2$ (Δdf)	*p*
0 Saturated model (means constrained)	3281.56	1509	263.56	−3082.07					
1 ACE	3345.97	1557	231.97	−3299.10	64.41 (48)	0.06			
1a. ACE M = F	3364.39	1566	232.39	−3219.06	83.83 (57)	0.02	1	7.61 (9)	0.57
2 Drop shared environmental effects (AE)	3351.39	1563	225.39	−3216.17	69.83 (54)	0.07	1	6.14 (6)	0.41
3 Drop genetic effects (CE)	3405.03	1563	279.03	−3189.35	123.47 (54)	<0.01	1	60.03 (6)	<0.01
4 Drop genetic and shared environmental effects (E)	3636.89	1569	498.89	−3092.20	355.33 (60)	<0.01	1	371.32 (12)	<0.01
5 No genetic covariance	3350.88	1559	232.88	−3203.91	69.32 (50)	0.04	1	5.25 (2)	0.07
6 No common environmental covariance	3349.59	1559	231.59	−3204.55	68.03 (50)	0.05	1	3.98 (2)	0.14
7 No A or C covariance	3355.34	1561	233.34	−3207.94	73.78 (52)	0.03	1	11.34 (4)	0.02
8 No nonshared env. covariance	3348.71	1559	230.71	−3204.99	67.15 (50)	0.05	1	2.86 (2)	0.24
9 AE, no nonshared env. covariance	**3353.39**	**1565**	**223.39**	**−3221.43**	**71.83 (56)**	**0.08**	2	6.38 (8)	0.38

Note: Best-fitting model denoted in boldface type.