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Regular Article

Gene-environment interplays between family chaos and emotional problems among Nigerian adolescents: A twin study

Olakunle A. Oginni[1,2] and Yoon-Mi Hur[3]

[1]Department of Mental Health, Obafemi Awolowo University, Ile-Ife, Nigeria, [2]Social, Genetic and Developmental Psychiatry Centre, Institute of Psychiatry, Psychology and Neuroscience, King's College London, London, UK and [3]General College of Education, Kookmin University, Seoul, South Korea

Abstract

Gene-environment correlations and interactions for the relationship between emotional problems (EP) and family environment in adolescents in low- to middle-income countries (LMIC) have been rarely investigated. In total, 3207 adolescent twins aged 12–18 (Mean = 14.6 ± 1.73) years attending public schools in Lagos State in Nigeria completed measures of EP and Family Chaos (FC). Model-fitting analyses suggested that genetic and non-shared environmental influences on EP were 21% and 71%, respectively, and the corresponding estimates were 23% and 71% for FC. Shared environmental influences were not significant (8% and 6% respectively). Phenotypic correlation between EP and FC was .30 (95% CI = .27–.34), which was significantly influenced by genetic (A – 49%, 95% CI: 0.01–0.97) and non-shared environmental factors (E – 32%, 95% CI: 0.10–0.54). Shared environmental influences were not significant (C – 19%, 95% CI: −0.13 to 0.50). Moderation effects were significant whereby as FC increased, A on EP decreased (βA = −0.07, 95% CI: −0.12 to −0.02) while E increased (βE = 0.06, 95% CI: 0.03–0.09). Our findings indicate that genetic and non-shared environmental risk factors may mediate the relationship between EP and FC, and that as FC increases, protective genetic influences on EP may be attenuated, whereas environmental influences may become stronger in adolescents in LMIC.

Keywords: adolescents; emotional problems; family chaos; gene-environment correlation; gene-environment interaction

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Introduction

Emotional problems (EP) including anxiety and depressive disorders are the commonest mental health conditions (World Health Organization, 2017), and are first and sixth leading contributors to non-fatal health loss (World Health Organization, 2017). These conditions arise during childhood with the burden being highest in low- and middle-income countries. For example, up to 50% of Nigeria's population of 200 million are aged 18 years or less (United Nations, Economic, & Social Affairs, 2017), with one out of every seven adolescents under 16 years experiencing mental health difficulties (Cortina et al., 2012).

However, despite this burden, there is little research investigating the determinants of child and adolescent mental health in these settings (Owen et al., 2016). In Nigeria, only a few studies have investigated the associations between childhood risk exposures and mental health conditions. Of these, most have tested bivariate associations between childhood risk exposures such as chaotic households and child and adolescent mental health conditions. Furthermore, others have investigated associations between retrospectively assessed childhood risk and adult mental health conditions (e.g., Oladeji et al., 2010). In contrast, extensive research from higher-income settings have investigated the causal roles of such adverse environmental exposures in the etiology of childhood mental health conditions (Fogelman & Canli, 2019; Lupien et al., 2009). Unfortunately, it is unclear to what extents findings from higher-income countries can be generalized to low- to middle-income countries.

Furthermore, considering that even environmental risk exposures can be influenced by genetic factors (Plomin et al., 1977), it is possible that the associations between the risk factors and childhood mental health conditions identified in Nigeria are partly genetically mediated. For example, using a United Kingdom-based twin cohort a significant gene-environment correlation (i.e., overlap between genetic influences and environmental measures) was demonstrated for family chaos (FC) and depressive symptoms (Wilkinson et al., 2013). Specifically, chaotic households are characterized by high levels of disorganization, and lack of routines and structure in daily activities (Weisner, 2010), which is associated with adverse childhood mental health outcomes (Marsh et al., 2019). Thus, while children may inherit genetic risk for EP directly from their parents, genetic risk may also be transmitted indirectly via passive gene-environment correlation. This means that parents create genetically-influenced risk environments (e.g., chaotic family households, Plomin et al., 1977) associated with childhood EPs. In addition, children with inherited risk for EP may contribute to

Corresponding author: Yoon-Mi Hur, email: ymhur@kookmin.ac.kr

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FC (active gene-environment correlations). These associations can explain phenotypic as well as genetic correlations between EPs and risk environments such as chaotic family households in childhood and adolescence. Alternatively, chaotic households may moderate the expression of genetic risk for EPs (Wilkinson et al., 2013) such that genetic influences are differentially manifest in adverse environments – a stress-diathesis gene-environment interaction (Dick, 2011). However, these possibilities have not been previously examined in a non-Western sample.

Considering the high burden of childhood mental health conditions and the under-prioritization of mental health in low- to middle-income countries settings, it is important to start investigating the etiological mechanisms of childhood mental health conditions in these settings. A readily available and affordable approach is to use population-based genetically informative designs such as twin studies to disentangle the genetic and environmental mechanisms of childhood mental health risk. Findings from such studies can help focus further research efforts and policy changes on risk factors which can be targeted for preventive mental health intervention in low- to middle-income countries.

The objectives of the present study were therefore to use the genetically informative twin design to investigate (1) the extent to which genetic and environmental influences explain the relationship between chaotic family environments and EP among adolescents in Nigeria, and (2) whether the genetic and environmental influences on EP change as a function of chaotic family environments.

Method

Sample

The sample comprised 3207 adolescent twins aged 12–18 (Mean = 14.6 ± 1.73) years including 768 monozygotic (MZ) (Mean age = 14.6 ± 1.71) and 2439 dizygotic (DZ) (Mean age = 14.6 ± 1.74) twins drawn from the Nigerian Twin and Sibling Registry (Hur et al., 2019). Of these, 1458 (45.5%) and 1749 (54.5%) were male and female respectively. Twins were recruited from 272 public junior and senior secondary schools in Lagos state in Nigeria. Questionnaires were administered to twins in the school libraries or special classrooms. Research assistants and school teachers were present in the testing room to monitor twins and give instructions to twins. During the testing session, a saliva sample was taken and analyzed to determine twins' zygosity. Eighteen microsatellite markers of DNA including amelogenin were analyzed to test zygosity of twins. A much larger number of DZ than MZ twins in the present sample likely reflects twin birth rates in Nigeria (MacGillivray, 1986). Further details of the recruitment procedures have been previously described (Hur et al., 2013, 2017, 2019). Approval to conduct this research was obtained from the Ministry of Education in Lagos state and the Health Research and Ethics Committee of the Lagos State University Teaching Hospital in Nigeria. Our research has been conducted according to the ethical principles expressed in the Declaration of Helsinki.

Measures

EPs

These were assessed using the five items of the EP scale of the Strength and Difficulties Questionnaire (Goodman, 1997). The Strength and Difficulties Questionnaire is one of the most commonly used instruments for screening psychopathology in children and adolescents and has been translated into over 70 languages worldwide. It contains five scales which assess EP, Peer Problems, Hyperactivity, Conduct Problems, and Prosocial Behavior (five items per scale). Participants completed all five scales of the Strength and Difficulties Questionnaire. However, only the EP scale was used in the present study. The items of the EP scale include: *"getting a lot of headaches, stomach-aches, or sickness"*; *"worrying a lot"*; *"being often unhappy or tearful"*; *"being nervous in new situations"*; and *"having many fears."* These items assess emotional difficulties which are associated with an increased risk for internalizing disorders (Stone et al., 2010). Twins were asked to report each of these experiences on a 3-point Likert scale ranging from *"Not true"* (scored 0) to *"Certainly true"* (2). The responses were summed with higher scores indicating higher EP, and the Cronbach's alpha was 0.55 in the present sample.

All scales have good correlations with those of the Child Behavior Check List (Vogels et al., 2009), and yield good sensitivities in detecting psychiatric disorders in the community as well as in clinical samples (Goodman et al., 2004). A review that examined 48 studies ($N = 131{,}223$) from various countries examining the psychometric properties of the Strength and Difficulties Questionnaire concluded that the internal consistency, test–retest reliability, and validity of the subscales were satisfactory across different age groups and sexes (Stone et al., 2010).

FC

FC was assessed using the family adaptability scale (ten items) of the 20-item Family Adaptability and Cohesion Evaluation Scales (FACES III, Olson, 1986) which assesses family functioning in terms of adaptability (change) and cohesion (Place et al., 2005). The Family Cohesion scale (also comprising ten items) assesses the degree to which family members are connected to or separated from each other. Only the Family Adaptability subscale was included in the present analyses consistent with the study's objectives. The Family Adaptability subscale measures the family's capacity to change its power structure, role relationships and rules in response to situational or developmental needs. High scores are indicative of unstable routines and roles within the family (i.e., FC) while lower scores indicate more structured family environments (Place et al., 2005). Sample questions include *"Different persons act as leaders in our family," "The rules seem to change in our family"* and *"The children make the decisions in our family."* Each item was scored on a 3-point Likert scale ranging from *"Almost never"* (scored 0) to *"Almost always"* (2). Nine items from this subscale were used with one item excluded: *"Our family changes its way of handling tasks."* to improve the internal consistency of the subscale from .56 for all 10 items to .60 for 9 items. This may reflect the stability of the roles of children and adults in Nigerian households (Ajayi & Owumi, 2013; Ogunola, 2018). The responses to the items were summed with high and low scores indicating chaotic and structured family environments respectively. The Cronbach's alpha in the present sample was 0.60.

Statistical analysis

The data were prepared using SPSS (IBM SPSS vs 26) while biometric genetic analyses were carried out in OpenMx (Neale et al., 2016) using maximum likelihood estimation procedures. As is standard practice (McGue & Bouchard, 1984), FC and EP were adjusted for the main effects of age and sex by using regression analysis and standardized residuals were used in subsequent analyses.

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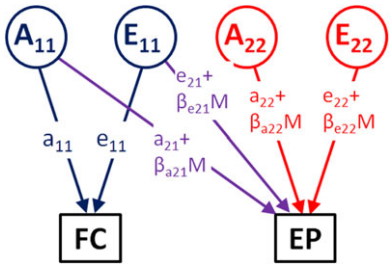

Figure 1. Bivariate moderation model with the moderator specified as a dependent variable. *Note*. In this model, family chaos (FC) is modeled as a dependent variable, allowing for moderation of its covariance with emotional problems (EP). A_{11}, E_{11}, A_{22} and E_{22} represent unique additive genetic and non-shared environmental influences on FC and EP respectively while a_{11}, e_{11}, a_{22} and e_{22} denote their respective unmoderated path coefficients, a_{21} and e_{21} denote the additive genetic and non-shared environmental coefficients of the covariance between FC and EP; ($\beta_{a22}M$, $\beta_{e22}M$) and ($\beta_{a21}M$, $\beta_{e21}M$) denote the respective moderation terms on the a_{22} and e_{22}, and a_{21} and e_{21} paths where M indicates possible values of the moderator (ranging between +2SD and −2SD).

Phenotypic correlations were computed using maximum likelihood estimation and constraints were applied whereby within-person correlations were equal across birth order and zygosity. This was to enable the estimation of many correlations using a reduced set of statistics as is typically done in structural equation modeling using twin data. To gain an initial impression of the genetic and environmental influences on each variable, maximum likelihood twin correlations were estimated across MZ and DZ twin pairs for FC and EP.

The variances and covariance of the variables were parsed into additive genetic (*A*), and shared (*C*) and non-shared (*E*) environmental components. This is achieved by comparing within-pair correlations in MZ and DZ twins raised together. The method assumes that MZ and DZ twins are 100% and 50% genetically identical; and that both types of twins share their family environment to the same extent (Rijsdijk & Sham, 2002). *A* influences indicate the extent to which individual differences between individuals reflect genetic differences. *C* influences indicate aspects of the environment that make twin pairs similar to each other and may include aspects of the home environment, while *E* influences indicate environmental influences that make twins different from each other including random idiosyncratic events and measurement error (Rijsdijk & Sham, 2002).

To investigate the genetic and environmental etiology of the correlation between FC and EP (first objective), we specified a bivariate *ACE* Cholesky model and interpreted a correlated solution (Loehlin, 1996). The decision to specify an *ACE* model was based on DZ cross-twin within-trait correlations being greater than half the corresponding MZ correlations (Rijsdijk & Sham, 2002). However, as *C* influences were not statistically significant in the bivariate *ACE* model, C parameters were dropped from the full model ($\chi^2[3] = 2.31$; $p = 0.51$; Supplementary Table S2). We thus reported the results of the *AE* model.

To investigate the second objective, we specified a bivariate moderation model in which FC was modeled as a moderator (Figure 1; Purcell, 2002). This model allowed us to explore whether the *A* and *E* component influences on EP varied as a function of FC (the effect of moderation) in addition to the genetic/environmental correlations between FC and EP. Specifically, moderation coefficients (β) were included in the expression of the path coefficients for the latent *A* and *E* influences on EP and its covariance with FC that is, $a + \beta_A M$ and $e + \beta_E M$ respectively where *M* indicates different values of the moderator (−2SD to +2SD). Furthermore, we adjusted for the direct effects of the cotwin's moderator variable as recommended by van der Sluis et al. (2012) when the moderator variable is not obligatorily shared by twin pairs to reduce the likelihood of false positive moderation effects. 95% confidence intervals of all estimates including moderation coefficients are reported and statistical significance inferred by intervals not including the null value of 0. As a preliminary exploratory step, we specified a bivariate phenotypic moderation model to test whether the variance in EP and its covariance with FC were moderated by FC and the results are reported in the Supplementary material.

Results

Descriptive statistics

Table 1 gives descriptive statistics for FC and EP by zygosity. The mean age of the participants was 14.6 (±1.73) years (Table 1), the mean FC score in the whole sample was 16.6 (±3.39) and that for EP was 3.8 (±2.41). Means (SDs) of these variables were not significantly different across zygosity or birth order within each zygosity group. Male participants ($n = 1458$, 45.5% of total sample) were older than female participants ($n = 1749$). Although the difference was statistically significant, the magnitude was small (Cohen's $d = 0.07$, $p = 0.03$). In contrast, female participants had significantly higher EP compared to male participants (Cohen's $d = 6.75$; $p < 0.001$). There was no significant sex difference in FC. Considering that there were no sex differences in the variances of the variables ($p > 0.05$ using Levine's test) and main effects of sex and age had been adjusted for, we did not further investigate sex differences in subsequent analyses.

Phenotypic correlations

Table 2 shows phenotypic, cross-twin within-trait, and cross-twin cross-trait correlations between FC and EP. There was a moderate positive correlation between FC and EP ($r = 0.30$, 95% CI: 0.27–0.34, Table 2) whereby higher FC was significantly associated with more EP. The cross-twin within-trait correlations among MZ twins for FC and EP were both 0.29 (95% CI: 0.20–0.38, the similarities in estimates are due to approximation). However, they were both less than 1, indicating the presence of E influences (including measurement error). Although the MZ cross-twin within-trait correlations were greater than the corresponding DZ correlations ($r = 0.18$ and 0.19, 95% CIs: 0.12–0.24 and 0.13–0.24 respectively) indicating the presence of A influences, the MZ correlations were less than twice the DZ correlations, which suggested some C influences.

Bivariate Cholesky model fitting

Bivariate twin model fitting indicated significant genetic influences on FC (32%, 95% CI: 0.25–0.38; Table 3) and EP (32%, 95% CI: 0.25–0.39), and the covariance between them (74%, 95% CI: 0.58–0.91). There were significant E influences on FC (68%, 95% CI: 0.62–0.75), EP (68%, 95% CI: 0.61–0.75) and their covariance (26%, 95% CI: 0.09–0.42). Genetic and environmental correlations (*rA* and *rE* respectively) between FC and EP were significant: 0.70 and 0.11 (95% CIs: 0.55–0.86 and 0.04–0.19 respectively). Significant genetic correlation between EP and FC implicates the presence of gene-environment correlation (Quinn & D'Onofrio, 2020; Rijsdijk & Sham, 2002). The estimates for the full *ACE* model are reported in Supplementary Table S1.

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Table 1. Characteristics of study sample

Variable	MZ	DZ	Male	Female	Total
Age (years)					
n	768	2439	1458	1749	3207
Mean (±*SD*)	14.6 (1.71)	14.6 (1.74)	14.7 (1.77)	14.6 (1.71)	14.6 (1.73)
Family chaos					
n	758	2400	1439	1719	3158
Mean (±*SD*)	16.5 (3.40)	16.8 (3.38)	16.7 (3.40)	16.7 (3.38)	16.7 (3.39)
Emotional problems					
n	733	2339	1401	1671	3072
Mean (±*SD*)	3.6 (2.40)	3.9 (2.41)	3.5 (2.39)	3.9 (2.37)	3.8 (2.41)

Note. MZ = Monozygotic twins; DZ = Dizygotic twins.

Table 2. Phenotypic and twin correlations and their 95% confidence intervals for family chaos (FC) and emotional problems (EP)

Variable	FC	EP
Within-person		
FC	1	
EP	0.30 (0.27–0.34)	1
MZ cross-twin		
FC	0.29 (0.20–0.38)	
EP	0.21 (0.14–0.27)	0.29 (0.20–0.38)
DZ cross-twin		
FC	0.18 (0.12–0.24)	
EP	0.13 (0.09–0.17)	0.19 (0.13–0.24)

Note. MZ = Monozygotic twins; DZ = Dizygotic twins. 95% confidence intervals are in parenthesis.

Table 3. Genetic and non-shared environmental influences on variances and covariance of family chaos (FC) and emotional problems (EP), and genetic and non-shared environmental correlations between FC and EP

	FC	EP	Correlation
a^2			
FC	0.32 (0.24–0.38)		
EP	0.74 (0.58–0.91)	0.32 (0.25–0.39)	*rA* = 0.70 (0.56–0.86)
e^2			
FC	0.68 (0.62–0.75)		
EP	0.26 (0.09–0.42)	0.68 (0.61–0.75)	*rE* = 0.11 (0.04–0.19)

Note. a^2 and e^2 = standardized genetic and non-shared environmental influences; *rA* and *rE* = Genetic and non-shared environmental correlations. 95% confidence intervals are in parenthesis.

Moderation analyses

Exploratory phenotypic moderation analyses indicated that FC significantly moderated the variance of EP (β = 0.09, 95% CI = 0.02–0.15) such that the variance in EP increased as FC increased while moderation of the covariance was not significant (Table S3 and Figure S1, Supplementary material). In line with these findings, genetic bivariate moderation analyses showed that the genetic and non-shared environmental influences on the variance of EP were significantly moderated by FC (Table 4, Figure 2) such that genetic influences decreased ($\beta_A = -0.07$, 95% CI: −0.12 to −0.02) and non-shared environmental influences increased ($\beta_E = 0.06$, 95% CI: 0.03–0.09) as FC increased. In contrast, the genetic and non-shared environmental influences on the covariance between FC and EP were not significantly moderated by FC.

Table 4. Moderation coefficients of component influences on variance of emotional problems (EP) and its covariance with family chaos (FC)

Moderation coefficients	Covariance of FC and EP	Variance of EP
β_A	0.03 (−0.02 to 0.08)	−0.07 (−0.12 to −0.02)
β_E	0.00 (−0.04 to 0.04)	0.06 (0.03–0.09)

Note. FC and EP = Family chaos and Emotional problems respectively, β_A and β_E = Moderation coefficients of additive genetic and non-shared environmental influences from genetic moderation model.

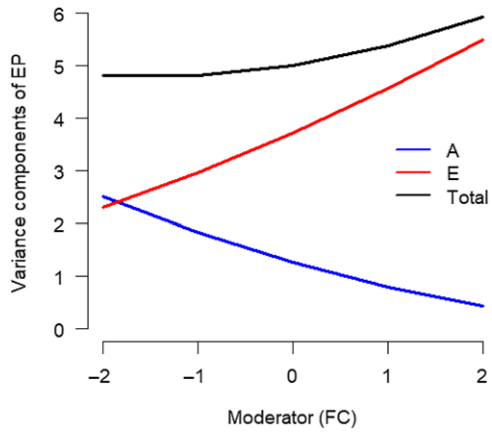

Figure 2. Moderation of the unstandardized variance component influences on emotional problems. *Note*. A = Additive genetic influences, E = Non-shared environmental influence; EP = Emotional problems; FC = Family chaos.

Discussion

This is the first study to investigate the etiology of the relationships between EP and an environmental risk factor – FC in adolescents

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using a genetically informative design in a low-/middle-income setting which are under-represented in global genetic research (Tucci & Akey, 2019). The present study confirmed a significant positive correlation between FC and EP and demonstrated significant genetic and non-shared environmental influences on both variables and their covariance. Furthermore, it was found that as FC increased, the genetic variance in EP decreased, whereas the non-shared environmental variance increased.

The heritability of FC found in the present study is consistent with heritability estimates of FC previously shown in higher-income countries, indicating that environmental variables (e.g., FC) are influenced by genetic factors (Plomin et al., 1977, Polderman et al., 2015). However, shared environmental (C) influences were smaller than non-shared environmental (E) influences and not significant in the present sample, suggesting that environmental influences on FC predominantly made children in the same family different from rather than similar to each other. The genetic and non-shared environmental influences on adolescent EPs are consistent with genome-wide polygenic and non-genetic (including early-life stressful experiences) influences on depressive and anxiety symptoms (Heim & Binder, 2012; Howard et al., 2019; Lupien et al., 2009).

Consistent with previous research (Wilkinson et al., 2013), the present study demonstrated that chaotic home environments were associated with higher EPs. A possible explanation is that the stress associated with adverse home environments may adversely impact on the developing brain which may manifest as an increased risk for EPs (Lupien et al., 2009). This is consistent with the correlation between the non-shared environmental influences on FC and EPs which suggests that FC and EPs are influenced by similar individual-specific factors. An alternative explanation suggested by the genetic correlation between FC and EPs is that the same genetic factors that predispose to EPs can also result in chaotic family settings which are independently associated with EPs (Kendler et al., 2002). Thus, parents can transmit the genetic risk for EPs to their children directly as well as indirectly through genetically mediated chaotic family settings. In contrast to this passive gene-environment correlation, it is also possible that children with genetic risk for EPs evoke chaos-promoting reactions from their parents (Plomin et al., 1977). However, these bidirectional effects need to be specifically tested in longitudinal studies.

A final mechanism for the etiological relationship between FC and EPs among the adolescents in the present study is via moderation effects. Specifically, we showed that the genetic contribution to individual differences in EPs decreased as FC increased. This finding contrasts with the stress-diathesis effect whereby genetic influences on an adverse health outcome manifest more strongly in increasingly stressful situations (Dick, 2011; Manuck & McCaffery, 2014). Instead, our finding is consistent with the prediction offered by the bio-ecological model (Pennington et al., 2009), where adverse environmental factors are hypothesized to influence the development of the disorder. According to this model, environmental sources of variance are strongest in the presence of environmental risk, whereas genetic influences are most prominent in the absence of environmental risk. Prior studies have provided some support for this model to explain attention deficit hyperactivity disorder (Nikolas et al., 2012). Further studies are needed to confirm whether this model can be applied to EPs. Another interpretation is that the etiological influences on EPs can be conceptualized as including those that are protective [e.g., (de Vries et al., 2021) and those that confer risk (Howard et al., 2019)]. Our finding may thus suggest that protective genetic influences on EPs among the Nigerian adolescents in the present study diminish as FC increases. This finding is also consistent with a previous phenotypic finding in which early childhood adversities were significantly associated with lower resilience and self-esteem among Nigerian adolescents (Folayan et al., 2020). Similarly, an animal study showed that early-life environmental stress among rats was epigenetically associated with diminished resilience (Meaney & Szyf, 2005). Thus, in a non-Western setting like Nigeria, diminished genetic resilience may be more salient than increased genetic risk for understanding the role of gene-environment interactions in the etiology of mental health difficulties. However, this interaction needs to be specifically investigated using measured genetic influences (such as polygenic scores) for EP and measured environments such as FC. Furthermore, our finding of stronger non-shared environmental influences on EPs with increasing FC is consistent with the kindling effect in depression. This phenomenon describes an environment-environment interaction whereby repeated stressful life events increase sensitivity to the psychopathogenic effects of subsequent stressful events (Kendler et al., 2000). Thus, in addition to being genetically correlated with EPs, chaotic family environments may further confer risk by attenuating protective genetic processes and increasing sensitivity to non-genetic risk influences.

Limitations

While the present study adds to the limited understanding of genetic and non-genetic influences on the mental health of children and adolescents in a non-Western setting such as Nigeria, the following limitations should be considered in interpreting our results.

Firstly, the classical twin design estimates genetic and environmental influences as latent constructs which means that further research is needed to identify specific genetic and environmental risk and protective influences which can then be targeted for later screening or intervention efforts. Furthermore, the variance components are population-based estimates which are time- and population-specific and may not provide information about individual-level processes (Bronfenbrenner & Ceci, 1994). However, findings from twin studies can help inform phenotypes and etiological mechanisms to prioritize in other genetic and epidemiological studies. Concerns have also been raised about the impacts of violating the underlying assumptions of the classical twin design but have been shown not to significantly impact on the derived estimates (Rijsdijk & Sham, 2002).

Although we estimated genetic and environmental components, the cross-sectional design of the present study limits the inference of causation. Such causal effects will be better estimated using longitudinal designs which can also be used to investigate how genetic and environmental risk and protective influences on childhood mental health change during development. Related to this, although adolescence is a critical developmental period (Lupien et al., 2009), early childhood may even be more critical as the developing neurobiological processes are relatively less developed and may be more vulnerable to the psychopathogenic impacts of environmental insults (Heim & Binder, 2012; Lupien et al., 2009).

We used the Adaptability scale of the FACES III to assess FC (i.e., unstable family structures and routines) and it may be argued that some of its items do not necessarily appear negative. However, the items of this scale are more likely to reflect instability in Nigerian families wherein children are expected to be submissive

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to the adults; and failure to conform to these roles are punished by parents without negotiation with the children (Ajayi & Owumi, 2013; Ogunola, 2018). Furthermore, several items from this scale load on the Chaos subscale of the FACES IV questionnaire (Martínez-Pampliega et al., 2017). It has also been suggested that FC in a high-income setting may not reflect the same construct in low- and middle-income settings, with families in both settings being typically nuclear and extended respectively (Ajayi & Owumi, 2013; Alabi & Olonade, 2022). However, a trend towards nuclear family structures has been increasingly noted in the last two decades (Rotimi, 2005) and this is more so in cities such as Lagos, Nigeria (Alabi & Olonade, 2022), where the present study was carried out. This trend may in turn suggest that the FACES III assessed construct of FC in the present sample is similar to that in high-income countries. However, the equivalence of the FC construct as assessed by the FACES III in high- versus low- and middle-income settings needs to be specifically tested in future studies.

Finally, although we detected statistically significant effects, the wide confidence intervals suggest the possibility that some of the non-significant effects may reflect low power and the need for larger samples in future studies examining similar relationships in Nigeria.

Future Directions

Our study highlights the need for more genetically informative mental health studies in non-Western settings. In the short term, the roles of specific protective genetic factors can be further tested by investigating genetic correlations between childhood EPs and intrinsic (e.g., self-esteem and resilience) and extrinsic protective factors (e.g., parental warmth and supportive home environments; (Bowes et al., 2013) using the classical twin design. Such models can also be extended to investigate the moderation of these relationships by childhood stresses including chaotic home environments.

In the longer term, future studies could utilize longitudinal twin family designs incorporating twins recruited during earlier childhood, and other family members such as parents and siblings. This can allow the assessment of the roles of specific risk and protective factors on mental health risk through development. The incorporation of non-twin family members will also allow adjustment for violations of the assumptions of the classical twin design, and the derivation of less biased estimates of genetic and environmental influences. The longitudinal design will also allow for the investigation of causal mechanisms and genetic/environmental influences on these and changes over time.

Conclusions

Our findings provide evidence for gene-environment interplays for EPs for the first time among adolescents in Nigeria – a Low-/Middle-Income country. We demonstrated significant genetic overlap between FC and EPs among the participants. Furthermore, we found that FC can increase the likelihood of EPs among Nigerian adolescents through diminished protective genetic influences and increased sensitivity to adverse effects of non-shared environmental risk influences. The need for replication is also emphasized given the dearth of genetically informative studies in Low- and Middle-Income countries.

Supplementary material. The supplementary material for this article can be found at https://doi.org/10.1017/S0954579422000943

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Conflicts of interest. None.

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