Polygenic risk for autism, attention deficit hyperactivity disorder, schizophrenia, major depressive disorder, and neuroticism are associated with experience of childhood abuse

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Abstract

People who experience childhood abuse are at increased risk for mental illness. Twin studies suggest that inherited genetic risk for mental illness may account for some of these associations. Yet the hypothesis that individuals who have experienced childhood abuse may carry genetic loading for mental illness has never been tested with genetic data. Using polygenic risk scores for six psychiatric disorders - attention-deficit hyperactivity disorder (ADHD), autism spectrum disorder (ASD), bipolar disorder (BPD), major depressive disorder (MDD), neuroticism, and schizophrenia – we tested whether genetic risk for mental illness was associated with increased risk of experiencing three types of childhood abuse: physical/emotional abuse, physical assault, and sexual abuse, in a cohort of white non-Hispanic women (N=11,315). ADHD and MDD genetic risk scores were associated with higher risk of experiencing each type of childhood abuse, while neuroticism, schizophrenia, BPD, and ASD genetic scores were associated with higher risk of experiencing physical/emotional abuse and physical assault, but not sexual abuse. Sensitivity analyses examining potential bias from differential recall of childhood trauma, parental socioeconomic status, and population stratification were consistent with the main findings. A one standard deviation increase in genetic risk for mental illness was associated with modestly elevated risk of experiencing childhood abuse (OR range: 1.05-1.19). Therefore, inherited genetic risk may partly account for the association of childhood abuse with mental illness. In addition,
future treatments for mental illness will benefit from taking into consideration the co-occurrence of childhood trauma and genetic loading.

Introduction

Nearly 30% of US children are exposed to physical, sexual, or emotional abuse\(^1\) that is associated with serious physical and mental health outcomes across their lifespans.\(^2\) The strong association between childhood abuse and mental illness is assumed to be due to the causal effects of child abuse on later mental illness. However, it has also been proposed that individuals who experienced childhood abuse may carry genetic loading for mental illness, which may account for some of the association between experiencing childhood abuse and mental illness.\(^3\)–\(^6\) Twin studies that compare the mental health outcomes of twins who did and did not experience childhood abuse suggest that the association between childhood abuse and borderline personality disorder,\(^3\) major depression,\(^4\) panic disorder,\(^4\) and alcoholism\(^4\) may be in part due to genetic loading. The co-occurrence of environmental factors such as childhood abuse and genetic risk is termed gene-environment correlation (rGE). Prior studies using genome-wide data have found several early-life environmental factors, including maternal and paternal age at gestation,\(^7\)–\(^8\) maternal years of education, and maternal alcohol use and anxiety during pregnancy\(^7\) associated with genetic risk for mental illness.

Three potential pathways exist by which the genetic risk for mental illness could confound the association between mental illness and experiences of childhood abuse (Figure 1). First, passive rGE posits that mentally ill parents (e.g., generation 0, G0) are more likely than those without mental illness both to pass on genetic risk for mental illness and to have children (generation 1, G1) who are abused (Figure 1, Panel A).\(^9\) Several studies have shown that parental schizophrenia, bipolar disorder (BPD), ADHD, and mood and anxiety disorders are associated with increased risk of offspring experiencing childhood abuse.\(^10\)–\(^17\) Such mental illnesses are also genetically influenced, such that the child would be at greater risk for these because of genetics inherited from the parents. Thus, rather than abuse causing mental illness in this scenario, abuse and mental illness co-occur due to shared genetic risk.\(^18\)

Second, in the evocative rGE pathway, or “challenging child hypothesis”, persons (G1) with genetic risk for mental illnesses may exhibit psychopathology-associated difficult behaviors as children, leading to an increased risk of experiencing child abuse (Figure 1, Panel B).\(^3\) For example, behavioral difficulties in children with ADHD and ASD,\(^19\) such as aggression, inattention, and oppositional behavior, have been linked to negative and controlling parental practices and reduced parental affection.\(^20\)–\(^25\)

Finally, mate selection may lead to rGE (Figure 1, Panel C). Persons (G0) who have experienced childhood abuse may be more likely as adults to select mates (also G0) with mental illness. Studies have indicated, for example, that persons exposed to child abuse are more likely to select mates with autistic traits\(^26\) and alcoholism.\(^27\) Their children (G1) would be at elevated risk of experiencing abuse due to behavioral transmission of abuse from the
parent who experienced abuse, and would be at risk of inheriting the mentally ill parent’s genetic loading for mental illness.\(^{15, 28}\)

However, the hypothesis that genetic risk for mental illness confounds the association between experiencing childhood abuse and later mental illness, which could occur through any of the three pathways depicted in Figure 1, has never been tested with molecular genetic data. The question of the degree to which childhood abuse causes psychopathology is a key question, relevant to both etiologic research and the development of interventions.\(^{29}\) Individual-level genetic risk for mental illness can be estimated using polygenic risk scores (PRS) that apply results from published genome-wide association studies (GWAS) to individuals’ measured genetic variants to estimate their genetic risk. The present study investigated rGE for child abuse and mental illness using molecular genetic data. We applied publicly available summary statistics from GWAS of ADHD,\(^{30}\) ASD,\(^{31}\) bipolar disorder,\(^{32}\) major depressive disorder (MDD),\(^{33}\) neuroticism,\(^{34}\) and schizophrenia\(^{35}\) to genetic data from the Nurses’ Health Study 2 (NHS2) to examine whether genetic risk for mental illness was associated with childhood physical, emotional, and sexual abuse (through any of the pathways described). We aimed to assess the presence of rGE, but not to distinguish among the specific pathways described in Figure 1.

**Materials and methods**

The NHS2 is an on-going cohort of 116,430 female nurses recruited in 1989 and assessed every two years. Participants were 24-44 years old at baseline. Blood samples were collected from 29,611 participants between 1996 and 1999, as previously described.\(^{36}\) Genome-wide data was available for 13,313 women based on three genotyping platforms: 1) Illumina Human Hap Array (N = 781), 2) Illumina OncoArray (N = 2,722), and 3) Illumina HumanCore Exome Chip (Batch 1 N = 3,276; Batch 2 N = 4,568). Following a standard quality control pipeline (call rate >0.90), participant genotype data were imputed using 1000 Genomes phase 3 reference data.\(^{37}\) Participants were restricted to those of European ancestry, given that PRS for mental illness were developed from GWAS of Europeans and may perform poorly for other ancestries due to differences in linkage disequilibrium patterns and the frequency of minor alleles.\(^{38}\) Informed consent was received from all participants and the study protocol was approved by the Institutional Review Boards of the Brigham and Women’s Hospital and the Harvard T.H. Chan School of Public Health.

**Polygenic Risk Scores**

PRS for ADHD,\(^{30}\) ASD,\(^{31}\) BPD,\(^{32}\) MDD,\(^{33}\) neuroticism,\(^{34}\) and schizophrenia\(^{35}\) were calculated using the summary statistics from the largest published GWAS, with p-value thresholds, clumping parameters, minor allele frequencies, and imputation score cutoffs based on those found to explain maximum variance based on Nagelkerke’s R\(^2\) from each analysis (see Supplemental Table 1).\(^{39, 40}\) Participant’s PRS for each mental illness were calculated by taking the weighted sum of risk alleles, with each allele weighted by the log odds ratio reported in published GWAS summary statistics using PRSice-2.\(^{41, 42}\) PRS were then standardized using z-score transformations.
**Childhood Abuse**

Childhood physical, emotional, and sexual abuse were assessed in a 2001 questionnaire. More than 11,000 women with genetic data responded to this questionnaire (n=11,347, 85% of women with genetic data). Five questions from the Physical and Emotional Abuse Subscale of the Childhood Trauma Questionnaire (CTQ) were used to assess physical/emotional abuse before age 12 years. The frequency of family members: 1) hitting so hard it left bruises, 2) punishing in a way that seemed cruel, 3) insulting, 4) screaming and yelling, and 5) punishing with a belt or other hard object was queried. In a validation study for the Childhood Trauma Questionnaire, these questions were found to load on a common factor of physical and emotional abuse (Cronbach’s alpha = 0.94, test-retest reliability = 0.82). Responses of never (0), rarely (1), sometimes (2), often (3), or very often (4) were summed, with the resulting scale dividing into quartiles.

Physical assault before age 18 was assessed for two time periods, ages 0-11 and 12-17 years, using items from the Physical Assault Subscale of the Revised Conflict Tactics Scale (CTS). Five questions were asked for each time period about whether a participant’s parent, step-parent, or adult guardian ever 1) pushed, grabbed or shoved you, 2) kicked, bit, or punched you, 3) hit you with something that hurt your body, 4) choked or burned you, and 5) physically attacked you in some other way. Response options were “Never” (0), “Once” (1), “A few times” (3), and “More than a few times” (4). Responses were summed, following coding recommendations. 46% of respondents reported no events; the remainder were classified as having experienced mild, moderate, or severe physical assault.

Sexual abuse was assessed with the Sexual Maltreatment Scale of the Conflict Tactics Scales, which consists of two questions regarding unwanted sexual touching and forced or coerced sexual contact by an adult or older child before age 12 years and two questions about experiences between ages 12-17 years. Participants could respond, “No this never happened” (0), “Yes this happened once” (1), or “Yes this happened more than once” (2). Responses were summed and individuals classified as having experienced none (score=0), infrequent (score=1 or 2), moderately frequent (score=3 or 4), or frequent sexual abuse (score ≥5).

**Covariates**

In 2005, the highest level of education completed by parents was queried, which were coded as: ≤ high school, some college, or ≥ 4 years college. Parents’ occupations were classified as: “skilled/service”, “unskilled”, “farmer”, “managerial/professorial”, or “other” worker. Depressive symptoms were ascertained by the 5-item Mental Health Inventory (MHI-5) in 1993, 1997 and 2001. Women who scored <60 at any of these time points were considered to have had probable depression before or at the time of the childhood abuse questionnaire. In addition, we account for residual population stratification – systematic differences in allele frequencies among those of varying exposures to childhood abuse across ancestries that can lead to spurious results – by including 10 principal components derived from the GWAS data as covariates.
Statistical Analyses

We first examined the distribution of parental education and occupation across each level of childhood abuse. Next, to determine the co-occurrence of the three types of child abuse, we calculated Spearman rank correlations. To determine the clustering of mental illness PRS in our sample, we calculated Pearson correlations between each pair of PRS.

To ascertain whether genetic risk for mental illness was associated with childhood abuse, we fit separate ordinal logistic models to estimate odds ratios (ORs) of child abuse level (e.g., quartiles for the CTQ and severity levels for the CTS and sexual abuse) in association with a one-standard deviation increase in PRS score. All models adjusted for parental education, parental occupation, genomic assay, and 10 GW AS principal components. The validity of the proportional odds assumption was assessed using Brant tests, graphical assessment of the parallel slopes across all levels of each outcome, and a likelihood test comparing an ordinal logistic model and multinomial (unconstrained) logistic model.

We conducted three sensitivity analyses. First, as depressive symptoms may influence the reporting of childhood abuse and are also likely associated with the MDD and neuroticism PRS, we calculated associations between PRS of MDD and neuroticism and each of the child abuse measures among women without probable depression at or before the 2001 assessment of childhood abuse (e.g., in 1993, 1997, or 2001). Second, to determine whether there may have been residual confounding by childhood socioeconomic status, we compared associations of PRS with child abuse with and without adjustment for parental education and occupation. Third, to examine whether there was an association with type rather than frequency of sexual abuse, we fit an ordinal logistic model using an alternative coding for sexual abuse, where individuals were classified as experiencing no sexual abuse, touching only, or forced sexual activity.

We also assessed potential bias from residual population stratification within our data, which may affect PRS by fitting our original ordinal logistic regression model without GWAS principal components, to assess whether population stratification affected the results. Additionally, we fit a second set of models including both GWAS principal components and self-reported ancestry (Southern European, Scandinavian, mixed, or other Caucasian) to investigate whether residual population stratification existed after controlling for GWAS principal components. Finally, we assessed whether residual population stratification within the NHS2 population was driving the association between PRS for mental illnesses and childhood abuse by simulating 1,000 sets of summary statistics containing 500,000 randomly selected SNPs and effect sizes (mean = 0, SD = 0.3, based on the SNPs we selected from the mental illness GWAS). These summary statistics, unassociated with childhood abuse but reflective of underlying allele frequency differences across populations within the NHS, were then used to create 1000 PRS for each participant. We estimated the association between each PRS and measure of child abuse and calculated the false positive rate as the percentage of simulated PRS associated with childhood abuse.

To assess whether a single PRS was responsible for the associations between each PRS and the outcome, for each outcome we fit an ordinal logistic regression with all six PRS, adjusted for parental education, parental occupation, assay, and GWAS principal.
components. Additionally, we generated new PRS for each mental illness by removing SNPs that were included in more than one PRS. Thus, these new PRS included SNPs unique to each mental illness.

Results

Women who experienced childhood abuse were more likely to have parents who had a high school degree or less and were unskilled workers, compared with women who experienced the lowest level of childhood abuse (Table 1). As anticipated, physical/emotional abuse (the CTQ) was strongly associated with physical assault (the CTS, r = 0.69). Sexual abuse was moderately associated with physical/emotional abuse (r = 0.23) and physical assault (r = 0.21) (Supplemental Table 2).

Correlations among the six mental illness PRS revealed three clusters: 1) schizophrenia, BPD and ADHD; 2) neuroticism and MDD; and 3) ASD (Figure 2). The schizophrenia, BPD, and ADHD PRS were each negatively correlated with the neuroticism and MDD PRS. The ASD PRS was uncorrelated with any other PRS.

A one standard deviation increase in any of the six PRS was associated with increased risk of experiencing physical/emotional abuse: ADHD (OR=1.19, 95% CI=1.12, 1.26), MDD (OR=1.14, 95% CI=1.10, 1.18), neuroticism (OR=1.11, 95% CI=1.07, 1.15), schizophrenia (OR=1.11, 95% CI=1.07, 1.16), BPD (OR=1.09, 95% CI=1.03, 1.15), and ASD (OR=1.05, 95% CI=1.02, 1.09; Figure 3, Panel 1; Supplemental Table 3). PRS were similarly associated with physical assault (Figure 3, Panel 2). However, only the ADHD (OR=1.15, 95% CI=1.07, 1.23), and MDD (OR=1.07, 95% CI=1.03, 1.11) PRS were associated with sexual abuse (Figure 3, Panel 3).

Sensitivity Analyses

Depressive symptoms may influence the reporting of childhood abuse and induce an association of experiencing childhood abuse with the MDD PRS. However, in the sensitivity analysis restricted to women with no probable depression, associations between the PRS for MDD and neuroticism, which is closely linked to MDD, with physical/emotional abuse, physical assault, and sexual abuse were consistent with those in the full sample (Figure 4; Supplemental Table 4).

Results of models excluding parental occupation and education were consistent in direction and significance with the main results (Supplemental Table 5). Analyses examining residual population stratification in our population did not indicate bias. Models excluding GWAS principal components (Supplemental Table 5) and those including self-reported ancestry (Supplemental Tables 5, 6) were consistent with the main results. Simulated PRS indicated that GWAS principal components provided adequate adjustment for population structure, with false positive rates within the expected range: physical/emotional abuse 5.2%, physical assault 4.8%, and sexual abuse 4.7%. Analyses with childhood sexual abuse coded as none, touching-only, or forced sexual activity were consistent with the main results (Supplemental Table 7).
In analyses with PRS for all disorders included in a single model, results were consistent in direction with the main results (Supplemental Table 8). In models using PRS comprised only of SNPs unique to each disorder, associations were also consistent with the main results, except ASD was more strongly associated with childhood abuse in these analyses (Supplemental Table 9).

Discussion

This is the first study to use molecular genetics to examine the association of genetic risk for mental illnesses with the experience of childhood physical, emotional, and sexual abuse. We found that genetic loading for ADHD and MDD were associated with higher likelihood of experiencing childhood physical/emotional abuse, physical assault, and sexual abuse. In addition, we found that genetic risk for neuroticism, schizophrenia, BPD, and ASD were associated with higher risk of experiencing physical/emotional abuse and physical assault. These results were robust to possible recall bias, confounding by parental education and occupation, and residual population stratification. Furthermore, the effect of the PRS for each mental illness was independent of the other mental illness PRS, implying no single PRS was driving the associations between mental illness and experiencing childhood abuse.

Associations between genetic risk for mental illness and sexual abuse were smaller compared to associations with physical/emotional abuse and physical assault. This may be because physical and emotional abuse questions specifically queried actions by people in the family, parents, or step-parents, whereas sexual abuse queried actions by any adult or older child. It is therefore possible that the physical and emotional abuse items better captured family dysfunction that may be related to heritable genetic risk of mental illness.

Our findings suggest that rGE may explain part of the association between childhood abuse and adulthood mental illness. However these results do not discount other pathways. First, childhood abuse can lead to dysregulation of the HPA-axis, which controls the body’s stress response, with repeated activation resulting in deleterious effects on the brain that have been associated with developing MDD. Second, experiencing childhood abuse can lead to poor behavioral regulation, poor social support, and maladaptive coping that put individuals at risk for later mental illness. As genetic risk scores become further refined, the role of genetic factors in these associations can be explored more precisely.

Maternal abuse has also been associated with elevated risk of mental illness among offspring, including depression, anxiety, ADHD, and ASD. Our findings suggest that genetic pathways may partly explain the co-occurrence of maternal experiences of childhood abuse with offspring mental illness. Previous studies have focused on two explanations for this intergenerational association. First, women who experience abuse are more likely to have children who also experience abuse, via behavioral transmission of childhood abuse across generations. Therefore, these children are also at increased risk of mental illness as experiencing childhood abuse is a strong risk factor for many mental illnesses. Second, women who experience childhood abuse are more likely to have perinatal risk factors that may place their children at increased risk of mental illness, including smoking and alcohol use during pregnancy, gestational diabetes, and preterm birth.
Both pathways may be confounded by rGE, though no studies have examined the association between genetic risk for mental illness and perinatal risk factors for mental illnesses. As our understanding of the biology and genetics of these mental illnesses improves, interventions may become more targeted and effective.

Our study has at least four limitations. First, the PRS for ADHD, ASD, BPD, MDD, and neuroticism have been found to explain a small proportion of the variation when predicting out-of-sample mental health outcomes (Nagelkerke $R^2$ range: 0.01 to 0.05, Supplemental Table 1). The use of PRS that explain little of the variation in mental illness may lead to attenuated estimates of the true associations between genetic risk and childhood abuse. This possibility is supported by our finding that the MDD PRS ($R^2 = 0.05$) was associated with sexual abuse, while the neuroticism PRS ($R^2 = 0.028$) was not significantly associated with sexual abuse, despite the neuroticism and MDD PRS being highly correlated (Figure 2). As sample sizes in discovery GWAS increase and the variability explained increases, we would expect concordance of associations across highly correlated PRS. Second, childhood abuse was queried retrospectively at ages 36-56 years, which may have resulted in misclassification. Third, while the inclusion of the first 10 principal components controlled for populations stratification in our sample, and our simulations also showed no bias, we are unable to adequately assess the residual population structure from the cohorts that were used to develop the PRS. Fourth, our study was conducted in a sample of high-functioning participants who are unlikely to include individuals with the greatest levels of risk for mental illness. Fifth, we were unable to elucidate which rGE pathway(s) affect the association between childhood trauma and later mental illness.

Future studies with measurement of mental illness, childhood trauma, and genetics in both parents and offspring will be needed to provide evidence for passive rGE, evocative rGE, or mate selection. For example, the passive and evocative rGE pathways could be examined through the associations of parent and offspring genetic risk, parental mental illness, offspring behaviors, and offspring experiences of childhood abuse. The presence of mate selection could be explored by examining the extent to which childhood abuse is associated with mate’s PRS, and whether this differs by sex. Although large samples would be likely be required, such studies could identify the degree to which each pathway accounts for the association between experiencing childhood abuse and later mental illness, albeit with the caveat that PRS currently explain only a portion of genetics-related phenotypic variance.

Our results suggest possible future research directions relevant to the etiology of mental illness. First, our findings suggest that rGE may be occurring in other circumstances. For example, rGE may account for some of the association between maternal perinatal health behaviors, e.g. smoking, and offspring neurodevelopmental risk. Maternal perinatal smoking has been found to be associated with offspring cognitive development. However, in a large sibling study (N=52,919) that examined discordant smoking behaviors across births using a sibling matching analysis to account for shared genetic risk, perinatal smoking was not associated with cognitive development, suggesting that rGE may account for the association of some perinatal risk factors with cognitive development.

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Our results indicate that genetic risk for mental illness is associated with modestly elevated risk of experiencing childhood abuse. Therefore, inherited genetic risk may account for part of the increased risk for mental illness in individuals who have experienced childhood abuse, and the increased risk for mental illness in their children. Improved genetic instruments will assist in developing targets for intervention to prevent mental illness and to better elucidate the role that experiencing childhood abuse has on later mental illness. Finally, future treatments for mental illness will benefit from taking into consideration the co-occurrence of childhood trauma and genetic risk.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.
Potential genetic pathways account for the correlation between risk for mental health disorders and childhood abuse. G0 = Generation 0, the Nurses’ Health Study 2 participants’ parents, G1 = Generation 1, the NHS2 participants.
Figure 2.
Correlations between polygenic risk scores for ADHD, ASD, BPD, MDD, neuroticism, and schizophrenia.
Figure 3. 
Odds ratios and 95% confidence intervals (CI) associated with a one standard deviation increase in each mental health polygenic risk score (PRS) for being in a higher level of childhood abuse adjusted for parental education, parental occupation, genomic assay, and 10 GWAS principal components.
Figure 4.
Odds ratios and 95% confidence intervals (CI) associated with a one standard deviation increase in the MDD and neuroticism polygenic risk score (PRS) for being in a higher level of childhood abuse, adjusted for parental education, parental occupation, genomic assay, and 10 GWAS principal components among all participants ($N_{\text{physical/emotional abuse}} = 11,315$, $N_{\text{physical assault}} = 11,322$, $N_{\text{sexual abuse}} = 11,317$) and among those with no probable depression ($N = 7,994$).
Table 1.

Descriptive statistics for age, parent education, and parent occupation across child abuse outcomes.

| Outcome                          | Levels                        |
|----------------------------------|-------------------------------|
|                                  | Quartile 1: Low | Quartile 2 | Quartile 3 | Quartile 4: High | Total  |
| Physical/Emotional Abuse (CTQ)   | N               | Age, Mean (SD) | Parent Education |
|                                  | 3,749           | 46.629 (4.491) | HS or less | 1681 (44.8%) |
|                                  |                 |               | 1132 (47.9%) | 1141 (50.6%) | 1567 (53.2%) | 5521 (48.8%) |
|                                  |                 | 2,363         | Some college | 1014 (27.0%) |
|                                  |                 | 46.337 (4.393) | 656 (27.8%) | 622 (27.6%) | 831 (28.2%) | 3123 (27.6%) |
|                                  |                 | 2,257         | College or more | 1054 (28.1%) |
|                                  |                 | 46.488 (4.441) | 575 (24.3%) | 494 (21.9%) | 548 (18.6%) | 2671 (23.6%) |
|                                  |                 | 2,946         | Parent Occupation |
|                                  |                 | 46.858 (4.279) | Skilled / Service Worker | 1871 (49.9%) |
|                                  |                 | 46.600 (4.409) | 1296 (55.9%) | 1712 (58.1%) | 6141 (54.3%) |
|                                  |                 |               | Unskilled Worker | 306 (8.2%) |
|                                  |                 |               | 209 (8.8%) | 201 (8.9%) | 345 (11.7%) | 1061 (9.4%) |
|                                  |                 |               | Farmer | 276 (7.4%) |
|                                  |                 |               | 151 (6.4%) | 139 (6.2%) | 145 (4.9%) | 711 (6.3%) |
|                                  |                 |               | Managerial/Professorial | 1176 (31.4%) |
|                                  |                 |               | 629 (26.6%) | 555 (24.6%) | 602 (20.4%) | 2962 (26.2%) |
|                                  |                 |               | Other | 120 (3.2%) |
|                                  |                 |               | 78 (3.3%) | 100 (4.4%) | 142 (4.8%) | 440 (3.9%) |
| Physical Assault (CTS)           | None | Mild | Moderate | Severe | Total |
|                                  | N               | Age, Mean (SD) | Parent Education |
|                                  | 5,044           | 46.415 (4.492) | HS or less | 2280 (45.2%) |
|                                  |                 |               | 1088 (48.4%) | 1470 (52.1%) | 685 (56.8%) | 5523 (48.8%) |
|                                  |                 | 2,247         | Some college | 1401 (27.8%) |
|                                  |                 | 46.645 (4.344) | 621 (27.6%) | 795 (28.2%) | 306 (25.4%) | 3123 (27.6%) |
|                                  |                 | 2,824         | College or more | 1363 (27.0%) |
|                                  |                 | 46.628 (4.402) | 538 (23.9%) | 559 (19.8%) | 216 (17.9%) | 2676 (23.6%) |
|                                  |                 | 1,207         | Parent Occupation |
|                                  |                 | 47.225 (4.129) | Skilled / Service Worker | 2565 (50.9%) |
|                                  |                 | 46.600 (4.409) | 1244 (55.4%) | 1637 (58.0%) | 697 (57.7%) | 6143 (54.3%) |
|                                  |                 |               | Unskilled Worker | 422 (8.4%) |
|                                  |                 |               | 201 (8.9%) | 281 (10.0%) | 157 (13.0%) | 1061 (9.4%) |
|                                  |                 |               | Farmer | 366 (7.3%) |
|                                  |                 |               | 140 (6.2%) | 149 (5.3%) | 56 (4.6%) | 711 (6.3%) |
|                                  |                 |               | Managerial/Professorial | 1516 (30.1%) |
|                                  |                 |               | 575 (25.6%) | 633 (22.4%) | 243 (20.1%) | 2967 (26.2%) |
|                                  |                 |               | Other | 175 (3.5%) |
|                                  |                 |               | 87 (3.9%) | 124 (4.4%) | 54 (4.5%) | 440 (3.9%) |
| Outcome                  | Levels            | Quartile 1: Low | Quartile 2 | Quartile 3 | Quartile 4: High | Total  |
|--------------------------|-------------------|----------------|------------|------------|-----------------|--------|
| Physical/Emotional Abuse (CTQ) |                   |                |            |            |                 |        |
| N                        |                   | 7,213          | 2,797      | 847        | 460             | 11,317 |
| Age, Mean (SD)           |                   | 46.442 (4.454) | 46.855 (4.334) | 46.864 (4.252) | 46.941 (4.349) | 46.596 (4.410) |
| Parent Education         |                   |                |            |            |                 |        |
| HS or less               |                   | 3421 (47.4%)   | 1396 (49.9%) | 452 (53.4%) | 250 (54.3%)     | 5519 (48.8%) |
| Some college             |                   | 1999 (27.7%)   | 776 (27.7%)  | 221 (26.1%) | 127 (27.6%)     | 3123 (27.6%) |
| College or more          |                   | 1793 (24.9%)   | 625 (22.3%)  | 174 (20.5%) | 83 (18.0%)      | 2675 (23.6%) |
| Parent Occupation        |                   |                |            |            |                 |        |
| Skilled / Service Worker |                   | 3841 (53.3%)   | 1571 (56.2%) | 478 (56.4%) | 250 (54.3%)     | 6140 (54.3%) |
| Unskilled Worker         |                   | 650 (9.0%)     | 261 (9.3%)   | 100 (11.8%) | 48 (10.4%)      | 1059 (9.4%) |
| Farmer                   |                   | 439 (6.1%)     | 175 (6.3%)   | 61 (7.2%)   | 36 (7.8%)       | 711 (6.3%)  |
| Managerial/Professorial  |                   | 2013 (27.9%)   | 684 (24.5%)  | 174 (20.5%) | 96 (20.9%)      | 2967 (26.2%) |
| Other                    |                   | 270 (3.7%)     | 106 (3.8%)   | 34 (4.0%)   | 30 (6.5%)       | 440 (3.9%)  |