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Building conditions, 5-HTTLPR genotype, and depressive symptoms in adolescent males and females

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Abstract

Purpose—Emerging work suggests that both environmental and genetic factors contribute to risk of depression in adolescents, and that these factors may differ between genders. We assessed whether features of the social environment (SE), measured at varying levels, and genetic factors jointly shape the risk of depression in adolescent males and females.

Methods—Using data from a national survey of U.S. adolescents, we applied cross-sectional, multilevel mixed models to assess the contribution of: (i) 5-HTTLPR genotype and respondent-level building conditions to depressive symptom score (DSS); and (ii) 5-HTTLPR genotype and neighborhood-level building conditions to DSS. Models testing potential gene-SE ($G \times SE$) interactions were also conducted. All models were stratified by gender and adjusted for age, race/ethnicity, family structure, parental education and social support.

Results—Among females, adjusted analyses indicated that *sl* genotype carriers enjoyed a marginally significant ($p=0.07$) protective effect against higher DSS in models assessing respondent-level building conditions. In contrast, among males, adjusted analyses predicted significantly higher DSS for residents of neighborhoods with relatively poor building conditions ($p<0.01$). No significant $G \times SE$ interactions were detected for either gender.

Conclusions—These results suggest that adverse, macro-level SE effects increase risk of depression to a greater extent in adolescent males than females. Intervention strategies designed to improve mental health in adolescent populations should consider a growing body of work suggesting that the contextual effects conferring increased risk of depression differ among males and females.

Keywords

social epidemiology; genetics; mental health; gender

Introduction

Depression is a commonly occurring mental illness characterized by persistent sad feelings, low energy, loss of interest in activities that were once pleasurable, feelings of guilt or low

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self-worth, disturbed sleep or appetite, and poor concentration, among other symptoms [1]. From a developmental perspective, the risk of major depression is known to increase with increasing age, with an estimated 2 percent of young children, 4 percent of young adolescents and 16 percent of older adolescents suffering from this disorder each year [2]. Although rates of major depression are approximately equal among boys and girls during childhood, during adolescence the prevalence for girls becomes almost twice that for boys [3]. Similarly, a marked increase in prevalence of depressive symptoms during adolescence among girls, but not boys, has been reported in studies based on self-reported depressive symptom scales [4]. The gender difference that emerges during adolescence persists until adulthood, when the lifetime relative risk of major depression is approximately twice as high among women compared to men [3].

These developmental patterns have motivated investigations of the etiology of depression focused on adolescents. Current knowledge suggests that environmental factors measured at the individual and family levels, such as childhood maltreatment [5], family dysfunction [6], and family socioeconomic status [7], are important contributors to depression in adolescents. Notably, some of these factors have shown differential associations depending on participants' gender [8]. To date, however, there has been a dearth of research regarding how factors operating more distal to the individual, such as residential segregation and the aesthetic environment, contribute to depression in adolescents. Nevertheless, the plausibility of such associations in this age group remain, given the documented associations between these more distal factors and depression in adult populations, including neighborhood socioeconomic status [9], urbanicity [10], and poor quality built environments measured at both the building [11] and neighborhood [12] levels. Indeed, such social factors, some of which lie well beyond the individual, are of prime importance in the production of human population health, and suggest that macrosocial determinants of human health should be a particular focus of research among investigators interested in improving public health [13].

Genetic factors are also known to be important contributors to adolescent depression. Twin and adoption studies have confirmed the existence of genetic influences on adolescent depression, with heritability estimates generally ranging from 30 to 50 % (reviewed in [14]). Of note, conflicting evidence exists on the relative importance of genetic contributions to depression in adolescent boys vs. girls, with some work finding these endogenous factors to be more important in boys compared to girls [15] and other work finding genetic influences to be more important in girls compared to boys [16]. These variable findings may be due in part to the fact that interacting effects of genetic and environmental factors are greater than their separate, independent effects; and, further, that these effects may differ by gender. From a biological perspective, sex-specific patterns of gene expression due to stress have been demonstrated in work based on animal models, with some genes showing opposite patterns of expression in males vs. females following administration of glucocorticoids [17], a known regulator of the stress response. Similarly, work in humans suggests that hypothalamic-pituitary-adrenal (HPA) axis stress responsivity differs by gender, with males showing a stronger stress-related cortisol response than females in both adult [18] and adolescent [19] samples. These findings help to contextualize the $G \times E$ literature focused on adolescents, which has found evidence for increased risk for depression and depressive symptoms among male carriers of the *ll* serotonin transporter promoter (*SLC6A4*) promoter (*5-HTTLPR*) genotype according to type of residence and parental separation [20], and female carriers of the *ss* genotype according to social adversity [21] and traumatic conflicts within the family [20, 21]. More generally, these sex-specific effects render plausible that different triggers, or stressors, may be salient to depression in adolescent males and females. Indeed, our own work has suggested that adolescent boys are more susceptible to macro- (i.e., county) level contextual effects on depressive symptoms than their female counterparts,

who showed stronger genetic effects on their risk for depression [22]. However, the extent to which this gender difference exists at other contextual levels is currently unknown.

With this background in mind, we conducted this study to investigate whether features of the social environment (SE), measured at varying levels, and genetic factors jointly shape the risk of depression in a sample of U.S. adolescents. Specifically, we examined whether building conditions, measured at the respondent- and neighborhood levels, respectively, genetic variation at the *5-HTTLPR* locus, and interactions between these factors contribute to risk for depressive symptoms in male and female adolescents sampled in the National Longitudinal Study of Adolescent Health (AddHealth). A functional polymorphism in the *5-HTTLPR* region is associated with differential uptake of serotonin *in vivo* [23] and has previously been implicated in differential susceptibility to depression (reviewed in [24]). Evidence for sex differences in serotonergic function is provided by animal models showing sex-specific differences in serotonin levels detected in the brain [25] and blood [26]. Thus, consistent with recent recommendations regarding G X E studies involving this locus [24], and depression more generally [27], we conducted this examination separately for males and females, and report results separately for each genotype.

Methods

Sample

Data for our analysis comes from the wave I in-home subsample (N = 20,745) of respondents from AddHealth. AddHealth is a nationally representative, school-based sample of US adolescents initially sampled in 1994 – 1995 and in three subsequent follow-up waves. DNA samples were collected from a subsample of siblings (n = 2,574) participating in the in-home questionnaire in 2002 as a part of wave III. The in-home and genetic data are part of the restricted use/contractual AddHealth dataset [28] and IRB approval to work with this dataset was secured from the University of Michigan prior to conducting these analyses.

Our respondent-level building analyses include 1,084 individuals from the sibling subsample who provided DNA, belonged to a same sex sibling cluster, and had complete data for each of the measures included in our models. Our neighborhood-level building analyses include 795 individuals meeting these same criteria.

Measures

Individual- and family-level health indicators

Depressive symptom scores (DSS) were obtained using a shortened, 17-item version of the Center for Epidemiological Studies Depression (CESD) Scale [29]. The CESD internal consistency was 0.85 based on the dataset used in the respondent-level analyses, and 0.86 based on the dataset used in the neighborhood-level analyses. Responses to the 17 questions ranged from 0 (never or rarely) to 3 (most or all of the time) and were summed for use as the outcome variable in all analyses. Respondents were required to answer all 17 questions in order to be included in our analyzed sample. DSS scores were classified as minimal (0–18), somewhat elevated (19–28), and very elevated (29–51) based on an adaptation of scores reported in Poulin et al.'s [30] study of 12 item CES-D scores in community-dwelling adolescents. The final DSS was standardized to the mean for all analyses.

Siblings were classified as monozygotic twins, dizygotic twins, full siblings, half siblings, or cousins, as indicated in the AddHealth data files.

Genotype—The *5-HTTLPR* locus is characterized by a variable number of tandem repeat (VNTR) polymorphism with two predominant alleles: the long (*l*) allele with 16 repeats and

the short (s) allele with 14 repeats, the latter of which corresponds to a ~44bp deletion in reference to the long allele [31]. Respondents were assigned one of three possible 5-HTTLPR genotypes: homozygote long (ll; referent category), homozygote short (ss), and heterozygote (sl).

Age and race/ethnicity—Age was calculated using date of birth and date of interview and left as a continuous variable in the model. Race/ethnicity was self-reported using the following categories: White (reference), African-American, Hispanic, Asian, and other race. Those respondents identifying as multi-racial were coded as other race.

Family structure categorized respondents as belonging to a two-biological parent family (referent category), a one-biological parent family (i.e. single biological parent or one biological parent and a stepparent) or “other family structure.”

Family-level socioeconomic status (SES) assessed the highest level of resident parental education attained (typically from the mother). This variable was dichotomized into high school graduate (or equivalent) or greater, and less than high school (referent).

Social support averaged the responses to eight questions that queried respondents on their perceived value and support from family members, friends and teachers; responses ranged from 1 (not at all) to 5 (very much). If respondents missed one or more of the questions, the average was determined from the remaining answered questions. The internal consistency of the social support scale was 0.78 based on the dataset used in the respondent-level analyses, and 0.77 based on the dataset used in the neighborhood-level analyses

Environmental measure

Building upkeep was selected as a measure of exposure to poor SE using the following two questions: 1) “how well kept is the building in which the respondent lives?” and 2) “how well kept are most of the buildings on the street?” from the in-home questionnaire, *Section 40 – Interviewer comments*. Responses were coded as “0” if the interviewer marked “very well kept” and “1” if the interviewer responded with “fairly well kept (needs cosmetic work), poorly kept (needs minor repairs) or very poorly kept (needs major repairs).” This coding created categories that were balanced between groups and improved the inter- and/or intra-rater reliability of the original measures (data not shown). Responses coded in this manner are referred to as well kept (0) and poorly kept (1) in the remainder of the manuscript. Separate analyses were run for each level of environmental measure (i.e. one model for the respondent’s building condition and another for building conditions in the respondent’s neighborhood).

Statistical Analysis

Hardy Weinberg Equilibrium—Genotype frequencies were assessed for Hardy-Weinberg Equilibrium (HWE) using Rodriguez et al.’s [32] online HWE Chi Square calculator by randomly sampling one sibling per family cluster in each stratum (well kept vs. poorly kept building conditions). Calculations were performed separately for each gender.

Analytic Model—A multi-level, mixed model was employed in our study, as described by the following equation:

$$CESD_{ij(s)} = \beta_0 \cdot X_{ij} + \beta_1 \cdot 5-HTTLPR_{ij} + \beta_2 \cdot \text{family structure}_{ij} + \beta_3 \cdot \text{SES}_{ij} + \beta_4 \cdot \text{support}_{ij} + \beta_5 \cdot \text{Building condition} + u_{j(s)} + e_{ij(s)}$$

where i, j indicate individual and sibling cluster, respectively. Each beta represents a single coefficient or a vector of coefficients for each predictor component in the model; X

represents age and race, *5-HTTLPR* represents the serotonin transporter promoter genotype, family structure represents the variants in resident parents, SES refers to resident parent's highest level of educational attainment, support refers to social support, and building condition refers to the overall level of building upkeep in which the respondent lives or, for the neighborhood-level model, the overall level of building upkeep on the respondent's street. The random effect of the family cluster is represented by $u_{j(s)}$, and $e_{ij(s)}$ is the error term, allowing the random effect of family cluster and the error term to vary by sibling type [33]. Interactions between *5-HTTLPR* genotype and respondent- or neighborhood-level building condition were explored in the interaction models in which the *ll* genotype and good building condition were the referent categories and all other covariates were maintained. All models were stratified by gender, and all analyses were conducted using SAS v. 9.2.

Results

Respondent-level building conditions

Table 1 presents the descriptive statistics and unadjusted associations for the individual-, family- and building-level predictors included in our final model based on respondents' building condition. The average age in both our male (n=510) and female (n=574) samples was approximately 16 years (range: 12–19 yrs, males; 12–20 yrs, females). The average DSS was significantly higher in female (11.1) vs. male (9.4) adolescents ($p<0.0001$). The male and female analytic samples did not differ from the excluded samples with respect to genotype, respondent-level building condition, or DSS, i.e. the main variables in the study (data not shown). For the sample stratified by both gender and respondent-level building condition, females showed genotype frequencies in Hardy-Weinberg Equilibrium (HWE) in both strata. In contrast, males residing in poorly kept (but not well kept) buildings showed *5-HTTLPR* genotypes that deviated from HWE ($X^2 = 4.35$, $df=1$, $p=0.037$), with an excess of *s* allele carriers.

Table 2 presents the results of our multivariable, multilevel main effects model for respondent-level building condition, which mirror many of the results obtained in unadjusted analyses (Table 1). However, adjusted analyses attenuated to non-significance the previously observed, positive relation between building condition and DSS in both males and females (Table 2). Similarly, the previously observed significant relation between *ss* genotype and DSS in females was no longer apparent in the adjusted model. However, the *sl* genotype showed a marginally significant, protective effect against DS in this gender ($b=-0.17$, 95% CI $-0.35, 0.01$, $p=0.07$). Interaction models did not show significant $G \times SE$ interactions for either gender (data not shown).

Neighborhood-level building conditions

Table 3 presents the descriptive statistics and unadjusted associations for the individual-, family- and building-level predictors included in our final model assessing neighborhood-level building conditions. The average age in both our male (n=377) and female (n=418) samples was approximately 16 years (range: 12–19 yrs, males; 12–20 yrs, females). As in the respondent-level building model, the average depressive symptom score was significantly higher in female (11.4) vs. male (9.5) adolescents ($p<0.0001$). The male analytic sample did not differ from the male excluded sample with respect to genotype, neighborhood-level building condition or DSS (data not shown). In females, the proportion of *sl* genotype carriers was significantly higher in the included sample (50.24% vs. 44.15%; $p=0.04$). All other major study variables were comparable in included vs. excluded female samples. For the sample stratified by both gender and neighborhood building condition, genotypes were in HWE in all strata.

Table 4 presents the fully adjusted results of our neighborhood-level building analyses. Results mirror many of the relationships observed in unadjusted analyses (Table 3), including the significant and positive association between residing in a neighborhood with less well kept buildings and DSS in males only ($b=0.29$, 95% CI: 0.12, 0.47, $p<0.01$); however, adjusted models attenuated the previously observed, marginally significant relationship between these two variables in females (Table 4). Interaction models did not show significant $G \times SE$ interactions for either gender (data not shown); however, in these models, males continued to show a significant positive association between poorer neighborhood-level building conditions and DSS ($b=0.33$, 95% CI: 0.03, 0.63, $p=0.03$).

Discussion

Our work sought to assess the combined and interacting effects of environmental and genetic features on adolescent depression at multiple levels, controlling for a number of factors previously associated with depression in this population. Respondent-level building analyses provided evidence for increased DSS among adolescent males and females residing in buildings with relatively poor upkeep in unadjusted, but not adjusted, results. In addition, these analyses provided some evidence for genetic influences on DSS in adolescent females. In contrast, neighborhood-level building analyses provided evidence for increased DSS among adolescent males only residing in neighborhoods with poorer building conditions, in both unadjusted and adjusted results. No similar association was observed in females in these models that assessed larger-scale (i.e. neighborhood-level) features of the SE. Taken together, these results suggest that adolescent males may be more susceptible to macro-level SE influences on mental illness than their female counterparts.

Our choice of building upkeep as a measure of exposure to poor SEs contributes to a very limited, but growing, literature on the relation between housing and mental health. Studies focusing specifically on the overall quality of the housing environment have found that housing quality shows a positive correlation with psychological well-being; for example, a study of adults residing in the U.K. found that those residing in housing that was in a “poor state of repair” were four times as likely to experience isolation, depression and worries than those residing in well-kept housing [34]. Similarly, in studies involving children and adolescents, housing quality predicted mental health [35], symptoms of psychological distress [36], psychosomatic illnesses [37] and professional referrals for mood/conduct/stress disorders [38]. Certain housing-related measurements have also shown a differential effect by gender: among children randomly assigned to reside in 3- story vs. 14-story public housing buildings, teachers’ ratings of behavioral disturbances were higher for boys residing in 14-story buildings [39]. Notably, this difference was not observed in girls [39], suggesting that boys may be more susceptible to the contextual effects of suboptimal housing than girls.

Results of the current study should be considered in light of previous findings based on the same sample. In particular, our previous study assessing county-level SE exposures in the AddHealth sample identified a protective effect among female carriers of the *sl* genotype in both unadjusted and adjusted main effect results, and a $G \times SE$ interaction effect among males, with those carrying the *sl* genotype showing a protective effect against higher DSS in counties with higher levels of deprivation [22]. The present work did identify a marginally significant, similarly protective effect among female *sl* carriers in adjusted results from the respondent-level building models (Table 2); and a significant adverse effect of poor neighborhood-level building conditions on DSS in unadjusted and adjusted models in males. Notably, both of these findings remain, or become stronger, after including the county-level deprivation variable used in our earlier work: females *sl* carrier show a significantly lower DSS in adjusted models for respondent-level building condition ($p=0.04$, data not shown); and males continue to show a significant adverse effect of poor neighborhood-level building

conditions on DSS ($p < 0.001$, data not shown). Taken together, both our earlier work and the present findings suggest that females may have a more “endogenous” contribution to DS than their adolescent male counterparts, consistent with previous suggestions [16]; and that males are more susceptible to contextual effects, as has been demonstrated for other, behavior-related outcomes (e.g. [39]).

The present study did not find evidence for $G \times SE$ interaction effects, in contrast to other work focused on adolescents. Although our sample size was larger than many $G \times E$ studies conducted to date involving the *5-HTTLPR* locus, it remains possible that we were underpowered to detect a true $G \times SE$ interaction in these analytic samples. An alternative interpretation, however, may be that our lack of $G \times SE$ positive results are attributable to the subjectivity with which the SE feature was measured. A recent review found that the likelihood of detecting a $G \times E$ effect was found to be highest among those studies that used objective measures to assess environmental exposures (i.e. records obtained independent of the participants’ report) and lowest among those studies that used subjective measures (i.e. participant self report) [40]. The present study assessed SE exposures using a measure intermediate between these two extremes, i.e. the interviewers’ judgement of respondent- or neighborhood-level building conditions—an assessment method which is less likely to yield replications of earlier positive $G \times E$ findings at the *5-HTTLPR* locus [40]. This interpretation is plausible in light of our earlier study that used more objective SE measures (i.e. county-level census data) in the same AddHealth population and detected a $G \times SE$ interaction among adolescent males [22].

Our study should be interpreted in light of a number of limitations. Our sample size was modest and may have been underpowered to detect $G \times SE$ interactions. In addition, in our respondent-level building analyses, *5-HTTLPR* genotype frequencies did not meet HWE among males residing in buildings with relatively poor upkeep, raising the possibility of reverse causation; however, our lack of $G \times SE$ findings in this gender minimizes concerns about false positives. Similarly, in our neighborhood-level building analyses, the included vs. excluded female samples differed on *sl* genotype prevalence, which could have affected our ability to detect main genetic effects or $G \times SE$ interactions in this gender. We note, however, that even if this were the case, it would not alter our finding of a differential main effect of neighborhood level building condition between genders, i.e. the main finding of the study. Third, although our goal in this study was to assess the effect of building conditions on adolescent males’ and females’ mental health at both the respondent and neighborhood levels, adjusted results for both models attenuated the effect of these variables. This raises the possibility that our results do not reflect the effect of building conditions per se, but rather what the building conditions may represent; for example, quality of schools, hospitals or other services, the presence of environmental pollutants, peer influences, or other, unmeasured variables. Finally, although we made use of a longitudinal study, we conducted cross-sectional analyses due to the likely use of different interviewers (and their differing subjectivity on neighborhood building conditions) between data collection waves. Thus, our work cannot assess the extent to which residing in a building or a neighborhood with relatively poor building conditions “causes” or precedes increased DSS, suggesting an area ripe for future research.

Despite these limitations, our results are consistent with a growing body of work suggesting that adolescent males differ from their female counterparts in their susceptibility to social environmental influences on depressive phenotype, and that this difference in susceptibility may occur at multiple levels. Intervention strategies designed to improve mental health in adolescent populations should consider a growing body of work suggesting that the contextual effects conferring increased risk for depression differ among adolescent males and females.

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Table 1

Sample descriptives and unadjusted associations of respondent-level building model predicting standardized depressive symptom score

	Males (n=510)			Females (n=574)			Males (n=510)			Females (n=574)		
	n	mean	% /std	n	mean	% /std	b	p	95% CI	b	p	95% CI
Genotype												
SS	114	22.35		100	17.42	0.11	0.26	0.02	0.01	0.10	0.02	0.01
SL	233	45.69		279	48.61	-0.13	0.09	0.02	-0.29	0.02	-0.15	0.11
LL	163	31.96		195	33.97	0.07	0.41	0.24	-0.10	0.24	0.01	0.92
Demographics												
Age	16.2	1.66		16.0	1.68	0.06	0.02	0.01	0.01	0.10	0.02	0.44
White	280	54.90		362	63.07	-0.27	<0.01	-0.44	-0.09	-0.13	0.20	-0.34
Black	82	16.08		78	13.59	0.21	0.08	-0.03	0.45	-0.03	0.85	-0.32
Hispanic	84	16.47		75	13.07	0.01	0.94	-0.23	0.25	-0.02	0.90	-0.31
Asian	27	5.29		22	3.83	0.29	0.14	-0.10	0.67	0.57	0.04	0.03
Other	37	7.25		37	6.45	0.29	0.09	-0.04	0.63	0.33	0.11	-0.08
Family structure												
Two biological parents	330	64.71		346	60.28	-0.27	<0.01	-0.45	-0.08	-0.35	<0.001	-0.55
One biological parent	152	29.80		184	32.06	0.28	<0.01	0.09	0.47	0.30	0.01	0.09
Other family structure	28	5.49		44	7.67	0.05	0.79	-0.34	0.44	0.28	0.15	-0.10
SES												
Parent is a high school graduate	448	87.84		469	81.71	-0.45	<0.001	-0.72	-0.19	-0.27	0.04	-0.53
Social support	4.0	0.53		4.0	0.59	-0.60	<0.0001	-0.73	-0.47	-0.85	<0.0001	-0.98
Respondent-level building condition												
Poor building condition*	218	42.75		261	45.47	0.26	<0.01	0.10	0.43	0.30	<0.01	0.11
17-Item CESD												
Depressive Symptom Score	9.4	5.92		11.1	7.26							
Levels of depression												
Minimal (0-18)	470	92.16		494	86.06							
Somewhat elevated (19-28)	38	7.45		65	11.32							
Very elevated (29-51)	2	0.39		15	2.61							

Significant effect estimates at 5% level are bold-faced. b is the model parameter estimate, p is the p-value, and CI is the parameter estimate confidence interval

* Results for unadjusted models reflect the association between poor building condition and depressive symptom score

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Table 2
Adjusted main effects respondent-level building model predicting standardized depressive symptom score

	Male (n=510)			Female (574)		
	b	p	95% CI	b	p	95% CI
Genotype						
SS	0.04	0.72	-0.17 0.24	0.05	0.67	-0.19 0.29
SL	-0.08	0.35	-0.25 0.09	-0.17	0.07	-0.35 0.01
Demographics						
Age	0.02	0.27	-0.02 0.07	-0.03	0.27	-0.07 0.02
Black	0.26	0.03	0.03 0.48	-0.09	0.50	-0.34 0.17
Hispanic	-0.04	0.71	-0.28 0.19	-0.03	0.80	-0.30 0.23
Asian	0.47	0.01	0.12 0.83	0.60	0.01	0.14 1.05
Other	0.18	0.29	-0.15 0.50	0.04	0.83	-0.31 0.38
Family structure						
One biological parent	0.13	0.15	-0.05 0.32	0.30	0.00	0.11 0.48
Other family structure	0.00	0.98	-0.34 0.35	0.24	0.15	-0.09 0.56
SES						
Parent is a high school graduate	-0.39	0.00	-0.65 -0.12	-0.30	0.01	-0.53 -0.07
Social support	-0.57	<.0001	-0.70 -0.43	-0.84	<.0001	-0.98 -0.71
Respondent-level building condition						
Poor building condition	0.13	0.11	-0.03 0.29	0.14	0.10	-0.03 0.31

Significant effect estimates at 5% level are bold-faced

b is the model parameter estimate, p is the p-value, and CI is the parameter estimate confidence interval

Table 3

Sample descriptives and unadjusted associations of neighborhood-level building model predicting standardized depressive symptom score

	Males (n=377)		Females (n=418)		Males (n=377)		Females (n=418)					
	n / mean	% / std	n / mean	% / std	b	p	95% CI	b	p	95% CI		
Genotype												
SS	89	23.61	70	16.75	0.11	0.34	-0.11	0.32	0.24	0.11	-0.05	0.53
SL	173	45.89	210	50.24	-0.10	0.27	-0.28	0.08	-0.04	0.72	-0.25	0.18
LL	115	30.50	138	33.01	0.04	0.73	-0.16	0.24	-0.10	0.39	-0.33	0.13
Demographics												
Age	16.2	1.67	16.0	1.69	0.05	0.06	0.00	0.11	0.03	0.28	-0.03	0.10
White	189	50.13	245	58.61	-0.30	<0.01	-0.50	-0.10	-0.14	0.26	-0.37	0.10
Black	61	16.18	55	13.16	0.28	0.04	0.02	0.55	0.07	0.69	-0.27	0.41
Hispanic	77	20.42	69	16.51	-0.09	0.47	-0.35	0.16	-0.07	0.68	-0.38	0.25
Asian	25	6.63	19	43.18	0.28	0.16	-0.11	0.66	0.72	0.01	0.15	1.29
Other	25	6.63	30	7.18	0.52	0.01	0.13	0.92	0.13	0.57	-0.32	0.58
Family structure												
Two biological parents	235	62.33	246	58.85	-0.24	0.02	-0.45	-0.04	-0.37	<0.01	-0.60	-0.14
One biological parent	119	31.56	135	32.30	0.26	0.02	0.04	0.47	0.29	0.02	0.05	0.54
Other family structure	23	6.10	37	8.85	0.03	0.88	-0.39	0.46	0.31	0.13	-0.09	0.72
SES												
Parent is a high school graduate	328	87.00	337	80.62	-0.43	<0.01	-0.72	-0.14	-0.11	0.45	-0.41	0.18
Social support	4.0	0.53	4.0	0.59	-0.59	<0.0001	-0.75	-0.44	-0.86	<0.0001	-1.02	-0.71
Neighborhood-level building condition												
Poor neighborhood-level building condition *	181	48.01	200	47.85	0.36	<0.001	0.18	0.55	0.21	0.07	-0.02	0.43
17-item CESD												
Depressive Symptoms Score	9.5	5.88	11.4	7.42								
Levels of depression												
Minimal (0–18)	347	92.04	356	85.17								
Somewhat elevated (19–28)	28	7.43	49	11.72								
Very elevated (29–51)	2	0.53	13	2.11								

Significant effect estimates at 5% level are bold-faced

b is the model parameter estimate, p is the p-value, and CI is the parameter estimate confidence interval

* Results for unadjusted models reflect the association between poor building conditions and depressive symptom score

Table 4

Adjusted main effects neighborhood-level building model predicting standardized depressive symptom score

	Male (n=377)				Female (418)			
	b	p	95% CI	b	p	95% CI	b	95% CI
Genotype								
SS	0.00	1.00	-0.23 0.23	0.07	0.65	-0.23 0.37		
SL	-0.06	0.51	-0.26 0.13	-0.05	0.65	-0.26 0.16		
Demographics								
Age	0.01	0.57	-0.04 0.06	-0.01	0.77	-0.06 0.05		
Black	0.23	0.08	-0.02 0.49	0.04	0.83	-0.28 0.35		
Hispanic	-0.14	0.29	-0.39 0.12	-0.03	0.85	-0.33 0.28		
Asian	0.45	0.01	0.11 0.80	0.68	0.01	0.17 1.19		
Other	0.40	0.03	0.04 0.76	-0.04	0.83	-0.43 0.35		
Family structure								
One biological parent	0.13	0.21	-0.07 0.32	0.30	0.01	0.08 0.52		
Other family structure	-0.03	0.86	-0.41 0.34	0.21	0.26	-0.16 0.59		
SES and social support								
Parent is a high school graduate	-0.39	0.01	-0.68 -0.10	-0.20	0.16	-0.48 0.08		
Social support	-0.57	<0.0001	-0.72 -0.42	-0.86	<0.0001	-1.02 -0.70		
Neighborhood-level building condition								
Poor neighborhood-level building condition	0.29	<0.01	0.12 0.47	0.04	0.70	-0.17 0.25		

Significant effect estimates at 5% level are bold-faced

b is the model parameter estimate, p is the p-value, and CI is the parameter estimate confidence interval