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Executive Functioning Mediates the Effect of Behavioral Problems on Depression in Mothers of Children With Developmental Disabilities

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Abstract

The present investigation explored long-term relationships of behavioral symptoms of adolescents and adults with developmental disabilities with the mental health of their mothers. Fragile X premutation carrier mothers of an adolescent or adult child with fragile X syndrome ($n = 95$), and mothers of a grown child with autism ($n = 213$) were included. Behavioral symptoms at Time 1 were hypothesized to predict maternal depressive symptoms at Time 3 via maternal executive dysfunction at Time 2. Results provided support for the mediating pathway of executive dysfunction. Additionally, the association of behavioral symptoms with executive dysfunction differed across the two groups, suggesting that premutation carriers may be more susceptible to caregiving stress due to their genotype.

Keywords

Behavioral problems; executive functioning; depressive symptoms; fragile X syndrome; autism

Caring for an adolescent or adult child with a developmental disability has substantial psychological impacts on parents. It accounts for a 10% increase in the prevalence of maternal depression (Singer, 2006). For example, mothers of children with autism spectrum disorders are more likely to have emotional disorders than mothers of children without disabilities (Totsika, Hastings, Emerson, Lancaster, & Berridge, 2011). Fragile X syndrome also is highly stressful for parents as children with fragile X syndrome often have co-occurring anxiety, aggression, inattention, and autism symptoms (Bailey, Raspa, Holiday, Bishop, & Olmsted, 2009). Notably, many studies have shown that exposure to child behavioral symptoms or psychopathology, rather than the degree of intellectual disability or functional limitations, is the most significant source of poor well-being for parents of children with developmental disabilities (e.g., Bailey, Golden, Roberts, & Ford, 2007; Johnston et al., 2003). In the present study, we examined how the behavioral symptoms of adolescents and adults with two developmental disabilities (i.e., autism spectrum disorders and fragile X syndrome) are associated with maternal executive functioning limitations and depressive symptoms.

Autism spectrum disorders are characterized by qualitative impairments in social communication and interaction, as well as restrictive and repetitive behaviors (e.g., American Psychiatric Association, 2013), with the current prevalence estimated to be 1 in 68 children (Centers for Disease Control and Prevention, 2014). Although it is understood to be a genetic disorder, it is likely the result of the interaction of hundreds of genes (Ch'ng,

Kwok, Rogic, & Pavlidis, 2015; Eapen & Clarke, 2014; Liu & Takumi, 2014; Persico & Napolioni, 2013), and thus is distinct from fragile X syndrome which is caused by a single gene mutation. The behavioral problems of individuals with autism, as well as fragile X syndrome have been found in many studies to be predictive of parenting stress and compromised maternal well-being (e.g., Baker, Seltzer, & Greenberg, 2012; Huang, Yen, Tseng, Tung, Chen, & Chen, 2014; Jones, Hastings, Totsika, Keane, & Rhule, 2014; Taylor & Warren, 2012; Zablotsky, Anderson, & Law, 2013).

Fragile X Premutation Carriers

Fragile X syndrome is the most prevalent inherited cause of intellectual disability (Crawford, Acuña, & Sherman, 2001; Hagerman & Hagerman, 2002). Most mothers of children with fragile X syndrome are premutation carriers (defined as having between 55 and 200 CGG repeats in the *FMR1* gene). This genetic variant is relatively prevalent in the United States, affecting 1 in 148–209 females (Maenner, Baker, et al., 2013; Seltzer, Baker, Hong, Maenner, Greenberg, & Mandel, 2012; Tassone et al., 2012). Premutation carriers have a much milder phenotype than their children with fragile X syndrome, including elevated risk of executive functioning limitations (e.g., Berry-Kravis & Hall, 2011; Schneider, Ligsay, & Hagerman, 2013; Wheeler et al., 2014). For example, they have been found to have worse performance on central executive subcomponent of working memory than normal controls starting at 40 years of age, and such age differences increased over time (Cornish, Kogan, Li, Turk, Jacquemont, & Hagerman, 2009). Spatial and language processing also have been shown to have age-related declines in premutation carriers (Goodrich-Hunsaker et al., 2011). Sterling, Mailick, Greenberg, Warren, and Brady (2013) reported greater linguistic dysfluencies among premutation carrier mothers of adolescents and adults with fragile X syndrome than among mothers of those with autism spectrum disorders. Such dysfluencies have been shown to be associated with executive functioning limitations (Turkstra, Fuller, Youngstrom, Green, & Kuegeler, 2004). Notably, in the Sterling et al. study, these dysfluencies were more severe in older premutation carrier mothers but there was no age effect in mothers of individuals with autism. Thus, the empirical evidence suggests that executive functioning limitations may be linked to the premutation phenotype, with a pattern of increasing limitations associated with aging.

Additionally, an association between the degree of caregiving stress and the level of anxiety, depression, and other mental health outcomes has been demonstrated in premutation carrier mothers of children with fragile X syndrome. Having a greater number of children with fragile X syndrome in the family and higher levels of child problem behaviors have been shown to contribute to increased risks for anxiety disorder (Roberts et al., 2009) and cortisol dysregulation (Hartley et al., 2012) among premutation carrier mothers. It also has been reported that when a subgroup of premutation carrier mothers of children with fragile X syndrome (i.e., those with midrange CGG repeat length, equivalent to 90–105 repeats) experienced the greatest number of negative life events during the previous year, they suffered the highest levels of depressive and anxiety symptoms, and the most abnormal cortisol responses (Seltzer, Barker, Greenberg, Hong, Coe, & Almeida, 2012). Thus, environmental stressors may be implicated in elevated risks for psychological and

physiological dysregulation of premutation carrier mothers of children with fragile X syndrome.

Executive Functioning Limitations as a Mediator

Executive functioning is a comprehensive construct for cognitive capacity representing higher-level cognitive processes, including planning, working memory, and inhibition (Gilbert & Burgess, 2008; Packwood, Hodgetts, & Tremblay, 2011). Executive functioning is critical for self-regulation in everyday life, and it is susceptible to environmental influences. For example, stress exposure can undermine cognitive and physiological functioning, potentially resulting in poor mental health (Marin et al., 2011; Sandi, 2013). A growing body of evidence suggests that caregiving for a family member with a chronic condition can be detrimental to the cognitive functioning of caregivers (e.g., Pinquart & Sörensen, 2003; Vitaliano, Murphy, Young, Echeverria, & Borson, 2011).

In the context of developmental disabilities, the child's level of psychopathology may constitute a significant source of chronic stress exposure that could erode cognitive abilities in parent caregivers (e.g., Caicedo, 2014; Hastings, 2002; Ritzema & Sladeczek, 2011). Lovell, Elliot, Liu, and Wetherell (2014) reported that family caregivers of individuals with autism reported more problems in both their prospective and retrospective memory than their counterparts with a typically developing child. Similarly, Song, Mailick, Greenberg, and Ryff (2016) found that older mothers of a son or daughter with developmental disabilities had worse episodic memory than younger mothers, and that those who experienced high levels of negative parenting experiences showed the worst cognitive functioning.

Poorer executive functioning has been found to be associated with higher levels of depressive symptoms in older populations and in persons with major depressive disorder (e.g., Elderkin-Thompson, Moody, Knowlton, Helleman, & Kumar, 2011; Snyder, 2013; Thomas & O'Brien, 2008). Similarly, executive functioning deficits have emerged as a risk factor for depressive symptoms in clinical and community samples (Feldman, Knouse, & Robinson, 2013; Knouse, Barkley, & Murphy, 2013; Wingo, Kalkut, Tuminello, Ascnap, & Han, 2013). Thus, negative impact of stress on executive functioning may be a plausible mediating mechanism linking stress exposure to levels of depression among maternal caregivers of adolescent or adult children with developmental disabilities.

The Current Study and Hypotheses

The present investigation examined the extent to which executive functioning in caregiving mothers mediates the association between behavioral symptoms in the son or daughter with developmental disabilities and maternal depression, and whether the strength of this mediation effect differs between families affected by fragile X syndrome versus autism. The study was developed to address gaps in prior research. First, longitudinal data allows for a more rigorous assessment of the temporal ordering of the relationships among behavioral symptoms, executive functioning, and maternal well-being. Second, by comparing mothers whose son or daughter had distinct types of developmental disabilities (i.e., fragile X

syndrome vs. autism spectrum disorders), diagnostic-specific mechanisms can more fully be investigated.

We advanced three hypotheses for testing mediating processes. First, we hypothesized that prior levels of behavioral symptoms in the son or daughter with developmental disabilities would predict subsequent maternal depressive symptoms. Second, we hypothesized that behavioral symptoms would also predict the subsequent executive functioning limitations of these mothers. With these first two hypotheses established, the third hypothesis was that executive functioning limitations would mediate the association of behavioral symptoms in the son or daughter with developmental disabilities and maternal depressive symptoms. Finally, we further explored whether the magnitude of the mediating effect was stronger in premutation carrier mothers of adolescent and adult children with fragile X syndrome than in mothers of individuals with autism.

Method

Participants

Fragile X syndrome—The current sample included mothers participating in an ongoing longitudinal study of families of adolescents and adults with fragile X syndrome (Mailick et al., 2014). In order to be eligible for the study, respondents had to be the biological mother of a son or daughter with full mutation of fragile X syndrome ($N = 147$) who was at least 12 years old. In addition, the child had to reside with his or her mother, or maintain contact at least every week (i.e., by phone or in person; Baker et al., 2012; Seltzer, Barker, et al., 2012). For the current study, three waves of data were used. Each wave was approximately 18 months apart. The sample for the current study was restricted to mothers who carried the fragile X premutation (i.e., whose CGG repeats ranged from 55 – 200), whose child with fragile X syndrome did not have co-occurring autism spectrum disorders (Smith, Barker, Seltzer, Abbeduto, & Greenberg, 2012), and for whom complete data on the child behavioral symptoms were available for analyses ($n = 95$). Mothers who had a child with co-occurring autism spectrum disorders were not included in order to sharpen the diagnostic contrast in the research design.

The majority of mothers of individuals with fragile X syndrome were non-Hispanic White (95.8%). On average, they were in their early fifties at the start of the current study ($M = 51.10$ years old, $SD = 7.85$; ranged from 37 to 79 years of age). The majority (83.2%) had obtained an associate's degree or higher education. At Time 1, their mean household income fell between \$70,000 and \$79,999. More than half of these mothers (55.7%) had more than one child with a disability ($M = 1.73$, $SD = .74$). The target children with fragile X syndrome were in their early twenties at the start of the study ($M = 21.61$ years old, $SD = 7.63$; ranged from 12 to 49 years of age).

Autism spectrum disorders—The second study group consisted of mothers with an adolescent or adult child with autism. They were recruited from a larger longitudinal investigation (Adolescents and Adults with Autism Study, $N = 406$; Seltzer et al., 2011) with a parallel research design and measures as the fragile X syndrome study. All respondents had a child with autism who was at least 10 years old at the entry to the study. In total, 10

waves of data were collected for this longitudinal study. Similar to the fragile X syndrome sample, three waves of data were utilized for the present analysis (i.e., Time 8, 9, and 10 from the larger autism study). Since the measure of executive functioning limitations was first collected in Time 9, data from Time 8 and 10 were included to be parallel with the fragile X syndrome sample (i.e., one wave lag between each key variable). Each wave was approximately two years apart. For inclusion in the present study, participants had to have data on child behavioral symptoms and valid data on executive functioning limitations at Time 9, resulting in 213 cases for the present analysis. For clarity of the presentation, data collected at Time 8, 9 and 10 for the autism sample are referred to as Time 1, 2, and 3, respectively, in the present report.

Similar to the fragile X syndrome sample, the majority of mothers whose child had autism were also non-Hispanic White (95.8%). However, they were nearly 10 years older than their counterparts with fragile X syndrome ($M = 60.39$ years old, $SD = 9.44$; ranged from 43 to 89 years of age) and their mean household income was lower (between \$60,000 – \$69,999 as of Time 8). The majority (79.9%) had some college education or obtained an associate's or bachelor's degree. The adult with autism was in the adult's early 30s ($M = 31.16$ years old, $SD = 8.87$; ranged from 20 and 59 years old). Some of these families (43.2%) also had more than one child with disability ($M = 1.64$; $SD = 1.08$).

For the entire sample, missing data affected between 14% – 23% on executive functioning or depressive symptoms variables due to attrition and selection criteria. Group difference tests showed that the mothers included in the present analysis were more likely to be married ($\chi^2(1) = 14.30, p < .001$), to have higher levels of education ($t(306) = 2.70, p = .007$) and to have younger sons or daughters with developmental disabilities ($t(49.04) = 2.38, p = .021$; $t(85.66) = 3.70, p < .001$) than those not included.

The institutional review boards at the universities involved in data collection approved the protocol and written informed consent was obtained from all participants.

Note that although the people with developmental disabilities were adolescents and adults, because we are studying the relationship between their characteristics and the mental health of their mothers, we refer to them as adolescent or adult sons and daughters or grown children in this report.

Measures

In addition to diagnostic group (fragile X syndrome and autism), the primary variables in our longitudinal analysis were child behavioral symptoms (measured at Time 1), maternal executive functioning limitations (measured at Time 2), and maternal depression (measured at Time 3).

Adolescent/adult child behavioral symptoms—The total problems score of the Child or Adult Behavior Checklist (CBCL/ABCL; Achenbach & Rescorla, 2001, 2003) was used to assess behavioral symptoms in the individual with developmental disabilities. For those younger than 18.5 years, the CBCL was administered. For those 18.5 years or older, the ABCL was administered in order to ensure the measurement was age-appropriate (e.g.,

Greenberg et al., 2012). Respondents indicated the extent to which their son or daughter exhibited behavioral symptoms over the past 6 months on a scale from 0 (*not true*) to 2 (*very true*). Age normed T-scores were used for the analysis so as to enable us to integrate scores from two different age groups (e.g., Baker, Smith, Greenberg, Seltzer, & Taylor, 2012). T-scores at 64 or above reached the range of clinical significance, while scores below 60 were considered as in the normal range. T-scores between 60 and 63 were interpreted as borderline (Achenbach & Rescorla, 2001). Reliabilities have been well established for the CBCL and ABCL (Achenbach & Rescorla, 2001, 2003).

Maternal executive functioning—The Behavior Rating Inventory of Executive Function-Adult version (BRIEF-A; Roth, Isquith, & Gioia, 2005) was used to assess executive functioning. It consists of 75 items in 9 domains, i.e., Inhibit, Shift, Emotional Control, Self-Monitor, Initiate, Working Memory, Plan/Organize, Task Monitor, and Organization of Materials. Mothers were asked to indicate the extent to which they experienced each problem in daily life on a scale from 1 (*never*) to 3 (*often*) in the past month. A composite T-score was used to represent global executive functioning limitations (Roth et al., 2005). T-scores of 50 is the mean of distribution while T-scores of 65 or greater (i.e., 1.5 standard deviation above the mean) represented a threshold of being abnormally elevated in executive dysfunction (Roth et al., 2005). Excellent internal consistency were reported across samples (Cronbach alpha = .97 for fragile X syndrome sample; Cronbach alpha = .96 for autism sample). The validity of the BRIEF-A was well established in prior research (Roth et al., 2005; Roth, Lance, Isquith, Fischer, & Giancola, 2013).

Maternal depressive symptoms—The Center for Epidemiological Studies - Depression Scale (CES-D; Radloff, 1977) was used to measure depressive symptoms. Mothers reported the number of days in the previous week they experienced each of 20 symptoms of depression. Each symptom was rated on a scale from 0 (*never*) to 3 (*5 to 7 days*) and a sum score was computed. Higher scores represented more symptoms. Scores of 16 or greater were considered as clinically significant. The CES-D has been shown to have high levels of reliability (Cronbach alpha = .93 for fragile X syndrome sample; Cronbach alpha = .92 for autism sample).

Covariates—Age, maternal education (1 = *graduated from high school or less*; 4 = *post bachelor's or graduate degree*), number of offspring with disabilities in the family, and maternal marital status (1 = *married*; 0 = *other*), and coresidence status (1 = *living together*; 0 = *other*) were included as covariates. Given that the age of the son or daughter was highly correlated with maternal age ($r = .87, p < .001$), only the age of the son or daughter was included in the analyses.

In addition, the level of independence in daily activities of the son or daughter was covaried to control the effect of this additional source of stress exposure. Level of independence was measured by the Waisman Activities of Daily Living Scale (W-ADL; Maenner, Smith, Hong, Makuch, Greenberg, & Mailick, 2013; Smith, Maenner, & Seltzer, 2012). The W-ADL consists of 17 items measuring functional abilities in the areas of personal hygiene, mealtime skills, and housekeeping on a scale ranging from 0 (*does not perform the task at all*) to 2 (*performs the task independently*). Summary scores were computed, with higher

scores representing greater levels of independence. The scale has high levels of internal consistency (Cronbach alpha = .86 for fragile X syndrome sample; Cronbach alpha = .94 for autism sample).

Analytic Plans

Our models consisted of three major variables. Child behavioral symptoms at Time 1 was the key predictor. Maternal executive functioning limitations at Time 2 and depressive symptoms at Time 3 were the mediator and outcome variables, respectively. To examine our hypotheses, two steps were involved.

First, we used the entire sample (those whose child had fragile X syndrome and those whose child had autism) in path analyses using the Mplus program (version 7.4, Muthén & Muthén, 2012). Maternal depression at Time 3 was regressed onto child behavioral symptoms at Time 1. Next, executive functioning at Time 2 was added as a mediator and the indirect effects were examined. The son or daughter's age, daily living skills, and coresidence status as well as the mother's education, marital status, and number of children with disabilities were entered as covariates. Models were estimated based on the maximum likelihood method in the Mplus program.

Second, the differential strength of executive dysfunction as a mediator in mothers of children with fragile X syndrome versus mothers of children with autism was explored. For this second step, separate path analyses were conducted for the fragile X syndrome and autism samples. All models included the same set of covariates (i.e., son or daughter's age, daily living skills, and coresidence status as well as mother's education, marital status, and number of children with disabilities).

Standardized coefficients were reported for the effect of child behavioral symptoms on maternal depressive symptoms, and for the indirect effects of executive functioning limitations. Since conventional significance tests of indirect effects may violate the assumption of normal distribution, bootstrapping techniques with confidence intervals were conducted and reported to clarify the significance level of all indirect effects (Geiser, 2013; MacKinnon, 2008). To explore group differences, we conducted another set of multi-group analyses to identify which particular path may differ across groups. Corresponding effect sizes (standardized indirect effects) and confidence intervals were provided instead of using a Bonferroni correction for multiple comparisons (e.g., Garamszegi, 2006; Nakagawa, 2004).

Models were adjusted with all covariates. Since results using cases with complete data ($N = 225$) were similar to those of cases with missingness, the following analyses handled missing data with full information maximum likelihood (FIML) by default in the Mplus program.

Existing research suggests that depressive symptoms as measured by the CES-D are quite stable over time (e.g., Carter, Martínez-Pedraza, & Gray, 2009), with stability coefficients ranging from $r_s = .61$ to $.68$ across waves from the current study ($p_s < .001$). Therefore, an alternative model was tested to rule out the possibility that the reverse direction of effects

accounted for the processes being examined here. To clarify the directionality of the mediation model, maternal depressive symptoms at Time 2 was modeled as the mediator and maternal executive functioning at Time 3 was modeled as the outcome in the alternative model. If the indirect effect was not significant in the alternative model, then the conclusion that maternal executive functioning mediated the association between child behavioral symptoms and maternal depressive symptoms could be strengthened.

Results

Descriptive Data

Descriptive statistics and correlations are presented in Table 1. Mean scores of the major study variables were well below of the threshold of clinical significance. Only a quarter of the adolescents and adults in the full sample (24%) demonstrated behavioral symptoms at clinically significant levels (i.e., T-score ≥ 64). Less than one-fifth of the sample of mothers (17.8%) were above the level of clinical significance indicating executive functioning problems (i.e., T-score ≥ 65). One-quarter (29.4%) of the sample of mothers reported clinically significant depressive symptoms. Thus, the majority of the sample was within the normal range with regard to the behavioral symptoms of the son or daughter, and the executive functioning and level of depression of the mothers.

Group differences on demographic characteristics were found. Individuals with fragile X syndrome were significantly younger than those with autism ($t(306) = 9.11, p < .001$) and their mothers were more highly educated ($t(279.18) = 3.93, p < .001$). The sons and daughters with fragile X syndrome were more independent in their daily living skills than their counterparts with autism ($t(267.80) = 4.31, p < .001$). There were also group differences with respect to coresidence, with those who had fragile X more likely to live at home than those with autism ($\chi^2(1) = 45.44, p < .001$). However, no group differences were found for child behavioral symptoms, maternal executive functioning, or depressive symptoms after controlling for demographic characteristics ($F_s \leq 1.88, ns$).

As expected, child behavioral symptoms at Time 1 were associated on a bivariate level with subsequent maternal executive functioning limitations and maternal depressive symptoms. Greater levels of behavioral symptoms were associated with higher levels of maternal executive functioning limitations and higher levels of depressive symptoms (refer to Table 1; $r_s \geq .19, p_s < .05$).

Full Sample Multivariate Model

As shown in Figure 1a, the relationship between child behavioral symptoms and maternal depressive symptoms was examined in the full sample (i.e., fragile X syndrome and autism combined), after adjusting for covariates (maternal marital status and educational level, number of children with disabilities in the family, and the age, coresidence status, and daily living skills of the son or daughter). Consistent with our hypotheses, greater levels of child behavioral symptoms at Time 1 predicted more maternal depressive symptoms at Time 3 ($\beta = .29, p < .001$). After introducing executive functioning limitations into the model, child behavioral symptoms were associated with executive functioning limitations which also

predicted maternal depressive symptoms, suggesting that exposure to child behavioral symptoms predicted depressive symptoms in mothers via increasing the risk for executive dysfunction. The indirect effect was significant, unstandardized estimates = .16, 95% CI [.09, .25]; standardized estimates = .13, 95% CI [.07, .20]. The direct effect of child behavioral symptoms on maternal depressive symptoms was reduced ($\beta = .15, p = .010$), providing support to our hypothesis that executive functioning limitations partially mediated the relationship between child behavioral symptoms and maternal depressive symptoms over time.

Diagnostic Group Differences

Next, we calculated indirect effects separately for the two groups of mothers. As expected, the indirect effects remained significant for both samples (refer to Figure 1b for the fragile X syndrome sample: unstandardized estimates = .26, 95% CI [.06, .49]; standardized estimates = .20, 95% CI [.04, .37]; refer to Figure 1c for the autism sample: unstandardized estimates = .11, 95% CI [.02, .21]; standardized estimates = .09, 95% CI [.02, .17]). Executive functioning limitations fully mediated the effects of child behavioral symptoms on maternal depressive symptoms among mothers of individuals with fragile X syndrome, but it only partially mediated these effects among mothers whose adolescent or adult children had autism.

For exploratory purposes, we queried whether specific paths contributed differentially to these indirect effects in the two samples. We conducted another set of multi-group analyses by imposing equality constraints on one path at a time (i.e., child behavioral symptoms $\rightarrow$ executive functioning limitations versus executive functioning limitations $\rightarrow$ maternal depression). Results of multi-group analyses showed that child behavioral symptoms had a stronger effect on executive functioning limitations in mothers of children with fragile X syndrome (Figure 1b; $\beta = .56, p < .001$) than in mothers of children with autism (Figure 1c; $\beta = .18, p = .009$), Wald test = 13.11, $df = 1, p < .001$. However, the effect of executive functioning limitations on maternal depression remained similar across samples (Figure 1b; $\beta = .36, p = .003$; Figure 1c; $\beta = .48, p < .001$; Wald test = 1.24, $df = 1, ns$). These findings suggested that there may be greater cognitive vulnerability triggered by exposure to the stress of child behavioral symptoms among midlife and aging mothers whose son or daughter had fragile X syndrome than their counterparts whose children had autism, but that the effect of executive functioning limitations on depression was similar across groups.

The Alternative Model

To examine an alternative direction of effects, we treated maternal depressive symptoms at Time 2 as the mediator and executive functioning limitations at Time 3 as the outcome. However, the indirect effects via maternal depressive symptoms at Time 2 were not significant in any sample (unstandardized estimates = .00, 95% CIs [-.09, .16]; standardized estimates = .00, 95% CIs [-.06, .09]). Thus, child behavioral symptoms did not predict subsequent maternal executive functioning limitations through maternal depressive symptoms. The mediating role of limitations in maternal executive functioning was further supported.

Discussion

Guided by the well-established linkage between child behavioral symptoms and maternal depression in families of individuals with developmental disabilities (e.g., Bailey et al., 2007; Baker et al., 2012), the present study aimed at understanding of mechanisms of this association. Using a longitudinal approach with three time points, we examined the mechanism by which child behavioral symptoms contributed to maternal depression over time. We found that executive functioning limitations mediated this effect. This mediating process was evident in both mothers of individuals with fragile X and mothers of individuals with autism. The findings are consistent with the general literature on impacts of caregiving stress on cognitive functioning (Pinquart & Sorensen, 2003; Vitaliano et al., 2011).

One of the novel contributions of the present study is that it parsed individual differences in this general process, particularly those associated with a specific genotype present in one sample. Although both groups of caregiving mothers were exposed to similar levels of stress due to their adolescent or adult child's behavioral symptoms, premutation carrier mothers appeared to be more vulnerable to this source of stress than mothers of sons or daughters with autism spectrum disorders. Because there were no mean-level differences in child behavioral symptoms, a possible contributing factor to this differential effect is the genetic vulnerability of premutation carrier mothers of children with fragile X syndrome. Given prevalence of the premutation (approximately 1 in 148–209 females; Maenner, Baker, et al., 2013; Seltzer, Baker, et al., 2012; Tassone et al., 2012), understanding how premutation carriers may be affected by stress is of public health significance.

Indeed, past research suggested that fragile X premutation carriers may have some neurocognitive difficulties (e.g., Mailick et al., 2014). In contrast to mothers of individuals with autism, premutation carrier mothers of individuals with fragile X syndrome have been shown to have greater levels of linguistic dysfluency than mothers of individuals with autism, and that this dysfluency worsened with age only among the premutation carriers (Sterling et al., 2013). The present study, along with findings from Sterling and colleagues, suggests that the neurocognitive functioning of fragile X premutation carrier mothers may be particularly vulnerable to the presence of behavioral problems in their children with fragile X syndrome.

Contrary to our expectation, there were no mean differences on executive functioning limitations between premutation carrier mothers of individuals with fragile X syndrome versus mothers whose children had autism. It is possible that our sample of premutation carrier mothers was too young to detect cognitive declines. In the general population, measurable changes in executive functioning may not be detectable until the later stages of the life course. The average age of the mothers with fragile X syndrome in our sample was approximately 51 years old. An important question is whether premutation carrier mothers experience cognitive aging at a faster rate than their counterparts who have children with autism spectrum disorders. Although in this study we did not find evidence that this was the case, future research is needed on older premutation carriers to more definitively answer this question.

Some methodological limitations of the present study must be acknowledged. First, all measures were based on maternal report that was inevitably subject to shared method variance. Second, the sample size of premutation carrier mothers of children with fragile X syndrome ($n = 95$) was small relative to their counterparts with autism, which may have limited statistical power. Additionally, although the design was longitudinal, the total span of time elapsing between Times 1 and 3 was relatively short (approximately 3 – 4 years). Future studies should extend the period of study in order to better evaluate lasting effects of child psychopathology on the well-being of their aging parents.

Juxtaposed against these limitations are multiple strengths of the current study, including its longitudinal design, the moderated mediational analysis, and the parallel measures administered to two distinct groups of mothers of individuals with developmental disabilities, one of which was defined by genotype. Our findings suggest that stress-induced cognitive erosion could in part account for the path between exposure to child behavioral symptoms and maternal depression over time for both groups of mothers, which is consistent with the extant literature on the impacts of stress on cognitive functioning of caregivers more generally (e.g., Mackenzie, Smith, Hasher, Leach, & Behl, 2007; Peavy et al., 2009). Our results also corroborate the findings of past research showing that the cognitive abilities in mothers of young children with special health care needs are negatively affected (e.g., Caicedo, 2014). More important, the findings suggest that fragile X premutation carriers may have been doubly affected, in part because of caregiving stress and also possibly due to their own genetic vulnerability.

Our findings may also inform interventions and services for both family caregivers and people with developmental disabilities. Child behavioral symptoms not only can undermine the cognitive functioning and mental health of parent caregivers, but also often result in physical injury to the parents and the subsequent need for their medical care (Bailey et al., 2012). Thus, services for managing behavior problems in adolescents and adults with developmental disabilities are needed to minimize the rippling effects of these problems across generations and over time. Such services could be particularly beneficial to individuals with fragile X syndrome and their mothers who themselves may have heightened vulnerabilities.

In conclusion, this longitudinal examination highlights the long-lasting impacts of caregiving for an adolescent or adult child with developmental disabilities. Having a child with autism or fragile X syndrome takes a toll on maternal psychological well-being via challenges to cognitive functioning. Stress emanating from child behavioral symptoms took a greater toll on executive functioning for fragile X premutation carrier mothers of children with fragile X syndrome than mothers of children with autism. The combination of genetic susceptibility and caregiving burden warrants special attention in future research and from clinicians.

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Figure 1.

The mediational models for child behavioral symptoms, maternal executive functioning limitations, and maternal depressive symptoms. Standardized coefficients (β) are reported. Values in parentheses represent the estimates between child behavioral symptoms and maternal depressive symptoms before adding maternal executive functioning limitations to the model. Each variable has identical set of covariates (i.e., maternal educational level and marital status, number of children with disabilities in the family, and the age, coresidence status, and daily living skills of the son or daughter with developmental disabilities) which are not illustrated in the figure for simplicity.

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$.

Table 1

Correlations matrices across waves (N = 308)

Variables	1	2	3	4	5	6	7	8	9	M	SD	n
1. Child behavioral symptoms at T1	–	.19*	.24**	.05	.04	.18*	-.01	.00	-.17*	59.00	8.00	213
2. Maternal executive functioning limitations at T2	.52***	–	.49***	-.19**	-.08	.11	-.04	.09	-.05	52.85	10.69	182
3. Maternal depressive symptoms at T3	.44***	.49***	–	-.07	-.01	-.08	-.12	.00	-.15†	11.71	9.93	160
4. Maternal education	.05	-.05	-.30**	–	.06	.03	-.07	-.09	.09	2.75	1.15	213
5. Maternal marital status at T1 ¹	-.14	-.28*	-.15	.01	–	.07	-.31***	.13†	.01	.72	–	210
6. Number of children with disabilities	.10	.06	.07	-.08	.08	–	-.07	.12	.09	1.64	1.08	181
7. Child's age at T1	-.19†	.14	-.10	-.00	-.01	-.03	–	-.29***	-.12†	31.16	8.87	213
8. Coresidence status at T1 ²	.12	-.09	.06	-.23*	.06	.12	-.35**	–	.10	.46	–	213
9. Daily living skills at T1	-.20†	.07	-.27*	.17	-.00	-.10	.38***	-.14	–	20.65	8.15	209
M	58.08	54.52	12.41	3.17	.81	1.73	21.61	.87	24.01	–	–	–
SD	7.43	11.63	9.92	.69	–	.74	7.63	–	5.24	–	–	–
n	95	82	78	95	95	95	95	95	95	–	–	–

Notes. T1 = Time 1. T2 = Time 2. T3 = Time 3.

¹ 1 = married, 0 = other.² 1 = living together, 0 = other.

Values below the diagonal represent the fragile X syndrome sample. Values above the diagonal represent the autism sample.

† $p < .10$.* $p < .05$.** $p < .01$.*** $p < .001$.