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## Executive function and somatic problems in adolescents with above target glycemic control

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### Abstract

Adolescents with type 1 diabetes may be at elevated risk for somatic problems. This study used cross-sectional, baseline data from an intervention to examine if problems with executive function (EF) were associated with greater somatic problems independent of poor adherence and disease severity in adolescents with type 1 diabetes and above target glycemic control. In addition, it examined whether certain types of EF skills, i.e., metacognitive and behavior regulation, accounted for variance in somatic problems. Ninety-three adolescents completed a glycated hemoglobin (HbA1c) blood test and parents completed adherence, somatic problems, and EF questionnaires, which measured metacognitive, behavior regulation, and global EF. Greater somatic problems had significant bivariate associations with greater global ( $r = .42, p < .01$ ), metacognitive ( $r = .43, p < .01$ ), and behavior regulation EF problems ( $r = .31, p < .01$ ), worse adherence ( $r = -.39, p < .01$ ), and poorer metabolic control ( $r = .26, p < .05$ ). However, when adherence, metabolic control, and EF subscales were examined together in the same model, only greater global EF problems ( $b = .15, p < .01$ ) and metacognitive EF problems ( $b = .16, p < .01$ ) were independently associated with greater somatic problems; behavior regulation EF problems were not independently associated with greater somatic problems when controlling for adherence. Metacognitive EF problems may predict somatic problems in adolescents with above target glycemic control above and beyond physical symptoms related to disease management, underscoring the importance of proper assessment and treatment of these distinct somatic problems.

### Keywords

executive function; somatic problems; adolescence; metabolic control; diabetes management

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## Introduction

Somatic problems are perceived physical ailments (e.g., aches, pains, nausea, and vision problems) that may or may not be explained by an identifiable physical illness, and these problems predict worse health-related quality of life (HRQoL) across numerous chronic diseases and conditions (1,2). In addition, somatic problems are associated with high utilization of and high expenditures for medical services, with research showing average healthcare costs of patients with somatic problems to be as much as 14 times higher than those of average patients (3). Youths with type 1 diabetes have been shown to be at greater risk for somatic problems than youth without type 1 diabetes (4–6), but the mechanism through which this increased risk occurs remains unclear.

Type 1 diabetes is a chronic autoimmune disease that destroys cells in the pancreas that produce insulin—a hormone that converts blood sugar into energy. Individuals with type 1 diabetes must adhere to an intensive daily medical regimen to maintain healthy glucose levels. In adolescents with type 1 diabetes, increased somatic problems have been associated with worse disease severity (i.e., poorer metabolic control) in at least one study (5). An explanation for this finding is that poor adherence leads to increased disease severity, which exacerbates physical symptoms associated with type 1 diabetes, causing increased somatic problems. However, research in other chronic illness populations (7,8) has suggested that not only disease severity, but also problems with executive function, may contribute to increased somatic problems, possibly through decreasing one's ability to appropriately interpret and cope with bodily signals (9,10). For example, impairments in executive function have been linked with increased likelihood of bodily pain in survivors of childhood cancer (7) as well as with increased physical impact of epilepsy among children, even independent of disease severity (8). These findings are important, suggesting that for youth with type 1 diabetes and above target glycemic control, greater problems with executive function may be an important predictor of increased somatic problems, independent of metabolic control and its proximal cause, non-adherence.

The existing literature is mixed as how to interpret the association between diabetes and somatic problems in adolescents with type 1 diabetes (11,12). Studies have consistently found that youth with type 1 diabetes experience greater somatic problems than do those without type 1 diabetes (4,5,11). This finding remains even when adjusting for possible inflation of somatic symptom scores due to type 1 diabetes symptoms (11), indicating that pathways outside of poor adherence and metabolic control (which contribute to those type 1 diabetes symptoms) might also influence somatic symptoms. Further, research directly linking poor metabolic control to increased somatic problems has produced more varied findings. For example, one study found adolescents with poorer metabolic control experienced greater somatic problems than did those with better metabolic control (5), whereas another found no significant difference in somatic problems between youth with higher glycated hemoglobin percentages (HbA1c, an indicator of metabolic control) and lower HbA1c (12). So, although Perrin, Stein, and Drotar (13) have advised researchers to use caution when examining somatic problems in populations with chronic illness—suggesting that reported aches and pains may be inappropriately inflated as a product of the illness rather than as indicators of behavioral or psychological problems—it remains unclear

if poor adherence and disease severity are the primary processes associated with increased somatic problems in youth with type 1 diabetes. This highlights the importance of examining other factors in addition to adherence and disease severity, such as problems with executive function, that might contribute to the link between type 1 diabetes and somatic problems in youth with above target glycemic control.

Executive function is comprised of several cognitive self-regulatory processes—including the ability to plan, inhibit impulses, shift attention, self-monitor, organize, initiate activity, and utilize working memory (14). Although research has not yet examined the associations of executive function and somatic problems in youth with type 1 diabetes, there is evidence supporting an association between greater somatic problems and greater impairments with executive function in other populations with chronic health issues. Problems with executive function have been associated with somatic problems in survivors of childhood cancer (7), children with epilepsy (8), adult patients with multiple functional somatic symptoms—operationally defined as individuals exhibiting six or more clinically significant somatic problems (10)—and adults with somatic symptom disorder (15). One theorized pathway to explain the relation between executive function and somatic problems is that executive function impairments affect the development of somatic problems by influencing how a person construes and copes with bodily cues (9,10). For example, more perseverative errors on assessment- and computer-based executive function tests have been associated with somatic problems (16,17), implying that perseveration on a physical symptom (e.g., stomach pain) may be a pathway through which executive function deficits contribute to somatic problems.

More specifically, evidence suggests that somatic problems may be more strongly linked with metacognitive executive skills as opposed to other sets of executive skills, such as behavior regulation skills (18,19). Metacognitive executive skills are theorized to capture cognitive regulatory skills needed to plan, coordinate activity, and engage in active problem-solving, whereas behavior regulation executive skills index capability to implement behavioral actions toward goals. Bailey and Wells (18) provide compelling evidence to suggest dysfunctional metacognitive processes such as worrying, rumination, and perseverative thinking—i.e., heightened attentional focus on threatening stimuli—may help explain the relation between metacognitive impairments and somatic problems (20,21). For example, a lack of cognitive flexibility or ability to shift attention has been related to increased somatic problems (16,17), suggesting that an inability to interrupt or modify thoughts related to perceived physical symptoms may contribute to somatic problems. Moreover, some evidence supports that interventions bolstering metacognitive skills (e.g., worry postponement) lessen somatic problems (19). Given that adolescents with type 1 diabetes are a population at risk for somatic problems (4,5) and somatic problems are associated with high utilization of and high expenditures for medical services, it may be especially important to understand if perceived somatic problems are uniquely linked with executive function impairments over and above disease severity and poor adherence.

This was the first study to investigate if problems with executive function were associated with greater somatic problems independent of poor adherence and metabolic control in a sample of adolescents with type 1 diabetes and above target glycemic control. First, it was

hypothesized that there would be a bivariate association of greater executive function problems (at the global, metacognitive, and behavior regulation level) with poorer metabolic control, worse adherence, and greater somatic problems. Second, it was hypothesized that even when controlling for the associations between metabolic control and adherence with somatic problems, greater problems with executive function would be independently associated with greater somatic problems. Last, it was hypothesized that of the two subsets of executive skills—metacognition and behavior regulation—metacognitive executive skills would account for greater variance in explaining somatic problems.

## Methods

### Participants

Participants for this study included ninety-three adolescents (43% female, 96% white, 62% pump,  $M$  age = 15.12 years,  $SD$  = 1.40, average length of diagnosis = 6.15 years,  $SD$  = 3.46) recruited from two clinical sites affiliated with a children's hospital. All were recruited to participate in research intervention trials targeting non-adherence in adolescents with above target glycemic control through web-delivered counseling and incentives. This study utilizes cross-sectional data collected at intake prior to intervention participation. The sample size was powered according to the interventions for which participants were recruited, not for this study specifically. Inclusion criteria were the following: adolescents ages 13–17, type 1 diabetes diagnosis more than 15 months prior, average HbA1c  $\geq$  8% or 64 mmol/mol for the past 6 months and most recent HbA1c  $\geq$  8% or 64 mmol/mol, as well as broadband internet and a computer at home (to allow participation in web-delivered treatment). On average, families were middle class based on the Hollingshead 9-step scale for parental occupation ( $M$  = 5.44,  $SD$  = 2.49). Exclusion criteria included pregnancy and severe medical or psychiatric illness. This study was conducted in compliance with the college's Institutional Review Board. There is no missing data to report.

### Procedures

Intake assessments were conducted in the pediatric endocrinology department or a study office centrally located in the region. The study was explained to parents/guardians and adolescents, and parental/guardian consent and adolescent assent were obtained for all participants. Both the adolescent and parents completed a series of tasks and/or questionnaires. In this study, we used parent-reported youth somatic problems, parent-reported youth executive function, parent-reported adherence, and a glycosylated hemoglobin blood test for the youth.

### Measures

#### **Child Behavior Checklist (CBCL) – DSM-oriented Somatic Problems subscale.**

—The CBCL (22) is a parent-report measure used to identify behavioral and emotional problems across eight empirically derived syndrome scales and six DSM-oriented scales, including somatic problems. The DSM-oriented scales were created by asking experienced and culturally diverse child psychiatrists and psychologists to rate how consistent CBCL items were with specific DSM diagnostic categories. Items rated as very consistent by at least 64% of the raters were included in the DSM-oriented scales. The DSM-oriented

Somatic Problems subscale was selected for use in this study to index somatic problems as traditionally classified in the DSM that have been previously linked to clinically important outcomes of diminished HRQoL (4,23,24). The DSM-oriented subscale excludes items from the CBCL Somatic Complaints syndrome subscale less relevant to disease burden and HRQoL (e.g., nightmares). The DSM-oriented Somatic Problems subscale includes 7 items to assess somatization: aches and pains, headaches, nausea, eye problems, rashes and skin problems, stomachaches, and vomiting. The CBCL previously has been used to measure behavioral, somatic, and emotional problems in youth with type 1 diabetes (11,25–27).

**Behavioral Rating of Executive Function (BRIEF).**—The BRIEF Parent Form (28) is a parent-report scale containing 86 items. Parents rated whether their child engaged in specific behaviors using a scale of never, sometimes, or often. The BRIEF contains eight subscales (Initiate, Working Memory, Plan/Organize, Organization of Materials, Monitor, Inhibit, Shift, and Emotional Control) as well as two composite indices, the Metacognition Index (MI) and the Behavior Regulation Index (BRI), and a global index, the Global Executive Composite (GEC). The MI encompasses the Initiate, Working Memory, Plan/Organize, Organization of Materials, and Monitor subscales, and the BRI encompasses the Inhibit, Shift, and Emotional Control subscales.

Higher ratings on the BRIEF indicate greater problems with executive function. This measure has been validated in other samples of adolescent with type 1 diabetes (29). In addition, consistent with previous research conducted on youth with type 1 diabetes, we used parent-reported executive function (30–32). Gender- and age-adjusted t-scores of the BRIEF parent-report scale were used in analyses.

**Self-Care Inventory (SCI).**—Patient adherence was measured using the parent-reported SCI (33), a 14-item scale designed to measure four domains of adherence behaviors to the type 1 diabetes regimen, including self-monitoring of blood glucose, insulin dosing, diet, and exercise. Parents report on a 5-point Likert scale, with 1 representing “never do it” and 5 representing “always do it,” such that lower scores on each item represent worse adherence. To assess overall adherence to diabetes behaviors, an average of the 14 SCI items was computed. The SCI has shown validity and adequate reliability ( $\alpha$ 's > .78) in other samples of adolescents with type 1 diabetes (34–36).

The use of parent-reported adherence in the context of this study was supported by evidence to suggest parent- and youth-reported adherence are differentially linked with overall diabetes management and HbA1c (35). Specifically, youth reports of adherence have been found to correlate closely with one element of adherence, frequency of daily self-monitoring of blood glucose, but less so with HbA1c, whereas primary caregiver reports of adherence correlate more closely with HbA1c across time, potentially representing a more comprehensive assessment of overall diabetes management (35). This discrepancy informed the decision to use parent-reported adherence in the context of this study, as opposed to an average or a latent variable that combined parent- and youth-report, which may have obscured important variability in predicting HbA1c.

**Metabolic control.**—HbA1c from a blood sample at study intake was used as the measure of metabolic control. Because these data are combined from two iterations of the same intervention, HbA1c was measured via a venous blood test for 76 youth (with all samples analyzed in the same central laboratory), and a point of care glycosylated hemoglobin blood test was conducted for 17 youth. HbA1c serves as an indicator of metabolic control over the previous three months.

## Analyses

All analyses were conducted in SPSS v.25. First, a t-test was conducted to assess whether average HbA1c differed based on type of test (venous blood draw or point of care finger stick). Next, descriptive statistics and Pearson correlations between demographic variables, somatic problems, adherence, HbA1c, and executive function at the global and index levels were computed. Third, analogous multiple linear regressions were conducted to test if GEC, BRI, and MI were each significant predictors of somatic problems independent of adherence and metabolic control. Each model controlled for pump status and duration of diagnosis in Step 1. In addition, type of HbA1c test (venous blood draw or point of care finger stick) was also entered as a covariate in Step 1 to further account for any variance in somatic problems that might be explained by the different testing modalities. In Step 2, only HbA1c was entered to evaluate its association with somatic problems. In Step 3, adherence was entered to assess whether it explained unique variance in somatic problems independent of HbA1c. Steps 1, 2, and 3 were repeated for all models; in Step 4, each executive function predictor (GEC, BRI, or MI) was separately entered into a distinct model. This step examined the link between each executive function variable and somatic problems while accounting for the relation between somatic problems and both HbA1c and adherence. Thus, Step 4 assessed whether each executive function predictor explained a significant amount of variance ( $R^2$ ) in somatic problems above and beyond HbA1c and adherence.

## Results

There was no significant difference in HbA1c based on the type of test (venous blood draw versus a point of care glycosylated hemoglobin blood test) conducted ( $t(20) = -.49, p = .63$ ). Nevertheless, type of HbA1c test was still included in the model to control for systematic variance that could be introduced due to differences in HbA1c test. In addition, there were no differences in executive function or somatic problems based on baseline demographic variables (i.e., gender, age, pump status, date of diagnosis, and socioeconomic status), so these demographic variables were not included in the model. Descriptive statistics and correlations are provided in Table 1 for somatic problems, adherence, HbA1c, GEC, BRI, and MI. Greater somatic problems were associated with greater executive function problems on the GEC, BRI, and MI indices (see Table 1). In addition, greater somatic problems, the GEC, and the BRI were significantly associated with higher (worse) HbA1c; however, MI was not associated with HbA1c. Parent-reported adherence was associated with HbA1c, somatic problems, and greater executive function problems on all three subscales (i.e., the GEC, BRI, and MI indices). This suggests that executive function, adherence, and HbA1c were all linked with somatic problems.

To examine the association between executive function and somatic problems while accounting for the relation between somatic problems and both adherence and HbA1c, multiple linear regressions were conducted. In Step 1, pump status, but not duration of disease or type of HbA1c test, was significantly associated with somatic problems. In Step 2, HbA1c, but not pump status, duration of disease, or type of HbA1c test, was significantly correlated with somatic problems. In Step 3, adherence was added to the model and only adherence, but none of the other predictors, including HbA1c, was significantly correlated with somatic problems. In Step 4, three different models were examined, first using the GEC as a predictor, then the BRI, and last the MI. In the model with the GEC added in Step 4 (see Table 2), the full model was significant. Higher scores on the GEC, indicating greater problems with executive function, were significantly associated with somatic problems and accounted for 22% more variance explained in somatic problems. In addition, worse adherence, but not HbA1c, was still associated with somatic problems after controlling for GEC. Next, in the model with BRI added in Step 4 (see Table 2), the full model was significant. Worse adherence remained statistically significantly associated with somatic problems after controlling for BRI, but higher scores on BRI were not statistically significantly associated with greater somatic problems, despite accounting for 17% more variance explained in somatic problems. Last, in the model with MI added in Step 4 (see Table 2), the full model was significant, and higher scores on MI were associated with greater somatic problems, accounting for 24% more variance explained in somatic problems.

In addition, we conducted exploratory post-hoc analyses to assess whether any specific subscales within the MI index were more associated with somatic problems than were others, finding that no one subscale of MI independently accounted for the relationship between MI and somatic problems; rather, shared variance across the subscales explained the relation.

In summary, in all Step 4 models, HbA1c was not a significant predictor of somatic problems after controlling for adherence and executive functions. Although adherence remained a significant predictor of somatic problems in each Step 4 model, the GEC and MI executive function subscales were associated with somatic problems independent of the link between adherence and somatic problems. This finding supports the hypothesis that executive function and specifically metacognitive executive function problems would be associated with somatic problems independent of both HbA1c and adherence.

## Discussion

This study examined whether problems with executive function were associated with greater somatic problems independent of poor adherence and metabolic control in a sample of adolescents with type 1 diabetes and above target glycemic control. The results supported hypotheses that: (1) greater executive function problems (measured via the GEC, MI, and BRI), worse adherence, and poorer metabolic control were significantly associated with greater somatic problems; and (2) greater problems with the GEC and MI were independently associated with greater somatic problems, even after accounting for the association between both adherence and metabolic control and somatic problems. These findings are consistent with literature in other clinical populations, specifically that problems

with executive function are associated with somatic symptoms in survivors of childhood cancer (7) and in children with epilepsy, even when controlling for disease severity (8), which in the context of type 1 diabetes, is closely linked to poor adherence. Thus, these findings suggest that in addition to poor adherence and metabolic control, problems in executive function, and metacognitive executive function in particular, may result in ruminative thoughts, catastrophizing, and cognitive inflexibility, which may uniquely contribute to somatic problems in adolescents with type 1 diabetes and above target glycemic control.

An interesting finding from this study is that adherence predicted somatic problems above and beyond metabolic control. Thus, it may be that non-adherence contributed more to momentary or day-to-day type 1 diabetes symptoms that are not reflected in average HbA1c, thereby capturing more variance in the somatic problems that may be related to the disease process. Nevertheless, despite the relation between non-adherence and somatic problems, executive function predicted variance in somatic problems uniquely from adherence, suggesting problems with executive function are related to somatic problems through a different pathway.

This unique pathway between problems with executive function and somatic problems was elucidated in part through our investigation into which subtype of executive function was most associated with somatic problems. The results indicated that deficits in metacognitive executive function remained a robust predictor of somatic problems even after controlling for metabolic control and adherence, whereas deficits in behavioral regulation executive function failed to be a significant predictor of somatic problems after controlling for adherence. This is consistent with other literature finding an association between deficits in metacognition or metacognitive executive skills and somatic problems (18,37). More specifically, this finding supports that difficulties with metacognitive executive function, such as heightened focus on threatening stimuli, perseverative thinking, and inability to shift attention away from these thoughts, may be related to fixation and rumination on, as well as catastrophizing of physical symptoms, contributing to somatic problems (18). For example, problems with metacognitive executive function, such as excessive worrying about a physical symptom, may serve to strengthen and perpetuate health-related concerns through prolonged activation and reinforcement of cognitive pathways related to the somatic problems (38). Brosschot and Thayer (38) propose that these overactive cognitive networks related to somatic problems may distort information processing by enhancing detection of illness-relevant stimuli and interpretation of bodily signals as threatening, thereby inflating and maintaining the reporting of physical symptoms. Multiple studies have shown support for this proposed pathway, providing evidence to imply that problems with metacognition, specifically perseveration and worrying, represent pathogenic factors of somatic problems (19,21).

These findings should be interpreted in the context of some limitations. White adolescents made up the majority of this sample. As such, these findings may not generalize to racial or ethnic minority adolescents, and replication in a larger, more diverse sample is needed. Also, given that the sample consisted of adolescents with above target glycemic control (HbA1c 8 or 64 mmol/mol), the restriction of HbA1c range may have reduced our ability to see a



greater association of HbA1c with somatic problems and executive function and limited the generalizability of these findings. In addition, we recognize that those with higher HbA1c may present with more physical problems as a result of poor metabolic control; nevertheless, the purpose of this study was to examine how physical symptoms may exist in excess of poor adherence or HbA1c level, i.e., there may be other contributing factors to consider, such as poor executive function and its relation to somatic problems.

In addition, analyses were limited to cross-sectional hypotheses due to the nature of the clinical trial following this data collection. However, longitudinal examinations of changes in adolescent executive function over time and how it relates to somatic problems are needed to address potential links between executive function and the diabetes disease processes. Research indicates that episodes of low blood sugar (hypoglycemia) and early onset of diabetes can impair central nervous system development, affecting learning, processing speed, and executive function (39,40). These findings suggest irregular levels of blood glucose damage cognitive functioning gradually, and these accumulating insults may not be reversible (e.g., improving metabolic control may not mitigate the cognitive impairments inflicted by early onset diabetes or severe hypoglycemic events). Thus, previous poor diabetes control might impair executive function, which could contribute to increased somatic problems that persist even after diabetes management and disease severity have improved. At the same time, evidence supports more immediate and bidirectional relations between metabolic control and executive function. Specifically, research shows high blood glucose levels negatively affect real-time mental efficiency and performance (41) and that problems with executive function (e.g., inability to plan) are linked to poor medical adherence and metabolic control (29,30,42). As such, longitudinal examinations are needed to elucidate the complex and bidirectional relation between executive function and both disease management and severity across time.

Finally, apart from HbA1c, the other measures—executive function, adherence, and somatic problems—were parent reported. This is particularly relevant to somatic problems, as some investigators have suggested that parents conflate physiologic symptoms associated with above target glycemic control and somatic problems (or physical symptoms “without medical cause”) when reporting on adolescents’ somatic problems (13). However, research has indicated that physiological items relevant to diabetes (e.g., dizziness and headaches) captured by the CBCL somatic subscales do *not* artificially inflate somatic problem scores of youth with type 1 diabetes (11). Nevertheless, additional research utilizing teen, parent, and health care provider reports as well as performance-based measures of executive function are needed to better understand the associations found in this study. In particular, future research should examine if problems with metacognition in early adolescence or middle childhood indicate risk of somatic problems among adolescents with type 1 diabetes and further investigate how metacognitive executive skills could be targeted to improve somatic problems in these youths. Future research may also examine how commonly comorbid psychopathology with somatic problems, such as depression and anxiety, might contribute to the links between executive function and somatic problems in youths with type 1 diabetes.

Given that somatic problems are associated with worse HRQoL at the individual level (1,2) and exorbitant health care costs and utilization at the societal level (43), these findings are

clinically and practically significant. There is preliminary evidence suggesting metacognitive therapy (MCT) may reduce somatic problems through attention training, e.g., diverting attention from heightened threat monitoring (18,44,45). Further, there is evidence to support that among youth with executive function deficits, executive function training focused on metacognitive executive skills has resulted in substantial gains in executive function generally, in the BRIEF-measured MI specifically, and in youths' overall ability to regulate thoughts (46). Thus, when a pediatric endocrinologist sees a patient with type 1 diabetes exhibiting significant physical symptoms that are not consistent with the patients' level of adherence or disease severity, it is worth considering that these symptoms may be somatic problems related to problems with metacognitive executive function. These types of encounters represent key moments to assess for somatic complaints or refer patients to psychosocial services that may be able to better identify and target executive function and somatic problems through the treatments described above, whereas more traditional medical treatments addressing changes in metabolic control or adherence may be insufficient. While such interventions may not necessarily improve HbA1c—given that metacognitive executive function skills and HbA1c were not associated in this sample—they may help adolescents with comorbid type 1 diabetes and somatic problems increase adherence, but perhaps equally importantly, reduce somatic problems and their associated health care costs and health care utilization. Thus, for those adolescents struggling with both above target glycemic control and somatic problems, there are accessible methods available that may boost their metacognitive executive skills, lessen their somatic problems, and ultimately improve their HRQoL.

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## References

1. Hyphantis T, Tomenson B, Paika V, et al. Somatization is associated with physical health-related quality of life independent of anxiety and depression in cancer, glaucoma and rheumatological disorders. *Qual Life Res.* 2009;18(8):1029–1042. [PubMed: 19701696]
2. Mahrer NE, Montañó Z, Gold JI. Relations between anxiety sensitivity, somatization, and health-related quality of life in children with chronic pain. *J Pediatr Psychol.* 2012;37(7):808–816. [PubMed: 22493024]
3. Smith G, Monson R, Ray DC. Patients with multiple unexplained symptoms. *Arch Intern Med.* 1986;146:69–72. [PubMed: 3942467]
4. Nardi L, Zucchini S, D'Alberton F, et al. Quality of life, psychological adjustment and metabolic control in youths with type 1 diabetes: a study with self- and parent-report questionnaires. *Pediatr Diabetes.* 2008;9(5):496–503. [PubMed: 18507786]
5. Ohmann S, Popow C, Rami B, et al. Cognitive functions and glycemic control in children and adolescents with type 1 diabetes. *Psychol Med.* 2010;40(1):95–103. [PubMed: 19400976]
6. Stewart SM, Simmons A, White PC. Somatic items in the assessment of depressive symptoms in pediatric patients with diabetes. *J Behav Med.* 2011;34(2):112–119. [PubMed: 20857189]
7. Ness KK, Gurney JG, Zeltzer LK, et al. The impact of limitations in physical, executive, and emotional function on health-related quality of life among adult survivors of childhood cancer: a report from the Childhood Cancer Survivor Study. *Arch Phys Med Rehabil.* 2008;89(1):128–136. [PubMed: 18164342]

8. Sherman E, Slick DJ, Eylr KL. Executive dysfunction is a significant predictor of poor quality of life in children with epilepsy. *Epilepsia*. 2006;47(11):1936–1942. [PubMed: 17116035]
9. Campbell LK, Scaduto M, Van Slyke D, Niarhos F, Whitlock JA, Compas BE. Executive function, coping, and behavior in survivors of childhood acute lymphocytic leukemia. *J Pediatr Psychol*. 2008;34(3):317–327. [PubMed: 18667478]
10. Hall NM, Kuzminskyte R, Pedersen AD, Ørnbøl E, Fink P. The relationship between cognitive functions, somatization and behavioural coping in patients with multiple functional somatic symptoms. *Nordic journal of psychiatry*. 2011;65(3):216–224. [PubMed: 21062124]
11. Holmes CS, Respass D, Greer T, Frentz J. Behavior problems in children with diabetes: Disentangling possible scoring confounds on the Child Behavior Checklist. *J Pediatr Psychol*. 1998;23(3):179–185. [PubMed: 9640897]
12. Leonard BJ, Jang Y-P, Savik K, Plumbo PM, Christensen R. Psychosocial factors associated with levels of metabolic control in youth with type 1 diabetes. *Journal of Pediatric Nursing: Nursing Care of Children and Families*. 2002;17(1):28–37.
13. Perrin EC, Stein RE, Drotar D. Cautions in using the Child Behavior Checklist: Observations based on research about children with a chronic illness. *J Pediatr Psychol*. 1991;16(4):411–421. [PubMed: 1941423]
14. Jurado MB, Rosselli M. The elusive nature of executive functions: a review of our current understanding. *Neuropsychol Rev*. 2007;17(3):213–233. [PubMed: 17786559]
15. Inamura K, Shinagawa S, Nagata T, Tagai K, Nukariya K, Nakayama K. Cognitive dysfunction in patients with late-life somatic symptom disorder: a comparison according to disease severity. *Psychosomatics*. 2015;56(5):486–494. [PubMed: 25596020]
16. Trivedi JK, Sharma S, Singh AP, Sinha PK, Tandon R. Neurocognition in somatisation disorder. *Hong Kong Journal of Psychiatry*. 2005;15(3):97.
17. Inamura K, Shinagawa S, Nagata T, Tagai K, Nukariya K, Nakayama K. Executive dysfunction correlated with 2-year treatment response in patients with late-life undifferentiated somatoform disorders. *Psychosomatics*. 2016;57(4):378–389. [PubMed: 27044513]
18. Bailey R, Wells A. Metacognitive therapy in the treatment of hypochondriasis: A systematic case series. *Cognit Ther Res*. 2014;38(5):541–550.
19. Brosschot JF, Van Der Doef M. Daily worrying and somatic health complaints: Testing the effectiveness of a simple worry reduction intervention. *Psychology and Health*. 2006;21(1):19–31.
20. Brosschot JF, Gerin W, Thayer JF. The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *J Psychosom Res*. 2006;60(2):113–124. [PubMed: 16439263]
21. Brosschot JF, Pieper S, Thayer JF. Expanding stress theory: Prolonged activation and perseverative cognition. *Psychoneuroendocrinology*. 2005;30(10):1043–1049. [PubMed: 15939546]
22. Achenbach TM, Rescorla L. ASEBA school-age forms & profiles. In: Aseba Burlington, VT; 2001.
23. Dufton LM, Dunn MJ, Compas BE. Anxiety and somatic complaints in children with recurrent abdominal pain and anxiety disorders. *J Pediatr Psychol*. 2008;34(2):176–186. [PubMed: 18577541]
24. Nakamura BJ, Ebesutani C, Bernstein A, Chorpita BF. A psychometric analysis of the child behavior checklist DSM-oriented scales. *Journal of Psychopathology and Behavioral Assessment*. 2009;31(3):178–189.
25. Cohen DM, Lumley MA, Naar-King S, Partridge T, Cakan N. Child behavior problems and family functioning as predictors of adherence and glycemic control in economically disadvantaged children with type 1 diabetes: a prospective study. *J Pediatr Psychol*. 2004;29(3):171–184. [PubMed: 15131135]
26. Duke DC, Geffken GR, Lewin AB, Williams LB, Storch EA, Silverstein JH. Glycemic control in youth with type 1 diabetes: Family predictors and mediators. *J Pediatr Psychol*. 2008;33(7):719–727. [PubMed: 18296726]
27. Strudwick SK, Carne C, Gardiner J, Foster JK, Davis EA, Jones TW. Cognitive functioning in children with early onset type 1 diabetes and severe hypoglycemia. *The Journal of pediatrics*. 2005;147(5):680–685. [PubMed: 16291363]

28. Gioia GA, Isquith PK, Guy SC, Kenworthy L. Test review behavior rating inventory of executive function. *Child Neuropsychology*. 2000;6(3):235–238. [PubMed: 11419452]
29. Duke DC, Harris MA. Executive function, adherence, and glycemic control in adolescents with type 1 diabetes: a literature review. *Current diabetes reports*. 2014;14(10):532. [PubMed: 25142717]
30. McNally K, Rohan J, Pendley JS, Delamater A, Drotar D. Executive functioning, treatment adherence, and glycemic control in children with type 1 diabetes. *Diabetes Care*. 2010;33(6):1159–1162. [PubMed: 20215458]
31. Graziano PA, Geffken GR, Williams LB, et al. Gender differences in the relationship between parental report of self-regulation skills and adolescents' management of type 1 diabetes. *Pediatr Diabetes*. 2011;12(4pt2):410–418. [PubMed: 21392190]
32. Smith LB, Kugler BB, Lewin AB, Duke DC, Storch EA, Geffken GR. Executive functioning, parenting stress, and family factors as predictors of diabetes management in pediatric patients with type 1 diabetes using intensive regimens. *Child Health Care*. 2014;43(3):234–252.
33. Lewin AB, LaGreca AM, Geffken GR, et al. Validity and reliability of an adolescent and parent rating scale of type 1 diabetes adherence behaviors: The Self-Care Inventory (SCI). *Journal of pediatric psychology*. 2009;34(9):999–1007. [PubMed: 19423660]
34. Berg CA, Butler JM, Osborn P, et al. Role of parental monitoring in understanding the benefits of parental acceptance on adolescent adherence and metabolic control of type 1 diabetes. *Diabetes Care*. 2008;31(4):678–683. [PubMed: 18202244]
35. Berg CA, Butner JE, Turner SL, Lansing AH, King P, Wiebe DJ. Adolescents', mothers', and fathers' reports of adherence across adolescence and their relation to HbA1c and daily blood glucose. *Journal of behavioral medicine*. 2016;39(6):1009–1019. [PubMed: 27501733]
36. Helgeson VS, Reynolds KA, Siminerio L, Escobar O, Becker D. Parent and adolescent distribution of responsibility for diabetes self-care: Links to health outcomes. *J Pediatr Psychol*. 2007;33(5):497–508. [PubMed: 17848390]
37. Bouman TK, Meijer KJ. A preliminary study of worry and metacognitions in hypochondriasis. *Clin Psychol Psychother*. 1999;6(2):96–101.
38. Brosschot JF, Thayer JF. Worry, perseverative thinking and health. Emotional expression and health: Advances in theory, assessment and clinical applications. 2004:99–114.
39. Gaudieri PA, Chen R, Greer TF, Holmes CS. Cognitive function in children with type 1 diabetes: a meta-analysis. *Diabetes Care*. 2008;31(9):1892–1897. [PubMed: 18753668]
40. Northam EA, Anderson PJ, Jacobs R, Hughes M, Warne GL, Werther GA. Neuropsychological profiles of children with type 1 diabetes 6 years after disease onset. *Diabetes Care*. 2001;24(9):1541–1546. [PubMed: 11522696]
41. Gonder-Frederick LA, Zrebiec JF, Bauchowitz AU, et al. Cognitive Function Is Disrupted by Both Hypo- and Hyperglycemia in School-Aged Children With Type 1 Diabetes: A Field Study. *Diabetes Care*. 2009;32(6):1001–1006. [PubMed: 19324943]
42. Suchy Y, Turner SL, Queen TL, et al. The relation of questionnaire and performance-based measures of executive functioning with Type 1 diabetes outcomes among late adolescents. *Health Psychol*. 2016;35(7):661.
43. Barsky AJ, Orav EJ, Bates DW. Somatization increases medical utilization and costs independent of psychiatric and medical comorbidity. *Arch Gen Psychiatry*. 2005;62(8):903–910. [PubMed: 16061768]
44. Papageorgiou C, Wells A. Effects of attention training on hypochondriasis: A brief case series. *Psychol Med*. 1998;28(1):193–200. [PubMed: 9483696]
45. Weck F, Neng JM, Stangier U. The effects of attention training on the perception of bodily sensations in patients with hypochondriasis: A randomized controlled pilot trial. *Cognit Ther Res*. 2013;37(3):514–520.
46. Tamm L, Nakonezny PA, Hughes CW. An open trial of a metacognitive executive function training for young children with ADHD. *Journal of Attention Disorders*. 2014;18(6):551–559. [PubMed: 22647287]

**Table 1.**

Descriptive statistics and correlations of somatic problems, parent-reported adherence (SCI), HbA1c, and parent-reported executive function GEC, BRI and MI scales (T-scores)

	<b>Somatic problems</b>	<b>SCI</b>	<b>HbA1c</b>	<b>GEC</b>	<b>BRI</b>	<b>MI</b>
Mean	53.48	3.25	9.24	57.42	53.84	58.12
SD	5.98	0.54	1.11	11.74	11.45	11.94
Range	50–77	2.14–4.43	8.0–12.7	37–92	37–99	37–84
Somatic problems	1	-.39**	.26*	.42**	.31**	.43**
SCI		1	-.30**	-.37**	-.32**	-.36**
HbA1c			1	.21*	.23*	.18
GEC				1	.85**	.95**
BRI					1	.66**
MI						1

Abbreviations: BRI, Behavior Regulation Index; GEC, global executive composite; MI, Metacognition Index.

\*  $p < .05$

\*\*  $p < .01$

\*\*\*  $p < .001$

**Table 2.**

Multiple linear regression analyses predicting change in somatic problems using HbA1c, adherence, GEC, BRI, and MI as predictors<sup>1</sup>

Criterion: Somatic Problems	F	R <sup>2</sup>	R <sup>2</sup>	B	95% CI
Step 1	1.38	.05			
Pump				-2.85*	[-5.63, -.06]
Duration of disease				.18	[-.22, .57]
Type of HbA1c test				.25	[-2.93, 3.44]
Step 2	2.22	.09	.05*		
Pump				-1.77	[-4.67, 1.14]
Duration of disease				.24	[-.15, .63]
Type of HbA1c test				.36	[-2.76, 3.48]
HbA1c				1.31*	[.09, 2.53]
Step 3	4.24**	.20	.15**		
Pump				-1.77	[-4.52, .97]
Duration of disease				.17	[-.20, .54]
Type of HbA1c test				.90	[-2.07, 3.87]
HbA1c				.71	[-.49, 1.92]
Adherence				-3.81**	[-6.07, -1.55]
Step 4	5.38***	.28	.22**		
Pump				-1.54	[-4.17, 1.10]
Duration of disease				.13	[-.23, .48]
Type of HbA1c test				1.40	[-1.46, 4.26]
HbA1c				.53	[-.63, 1.69]
Adherence				-2.73*	[-5.00, -.45]
GEC				.15**	[.05, .26]
Step 4	4.12**	.23	.17		
Pump				-1.58	[-4.30, 1.15]
Duration of disease				.14	[-.23, .51]
Type of HbA1c test				1.26	[-1.71, 4.23]
HbA1c				.59	[-.62, 1.79]
Adherence				-3.30**	[-5.61, -.99]
BRI				.09	[-.01, .20]
Step 4	5.66***	.29	.24**		
Pump				-1.64	[-4.25, .97]
Duration of disease				.14	[-.21, .49]
Type of HbA1c test				1.47	[-1.37, 4.32]
HbA1c				.58	[-.57, 1.73]

Criterion: Somatic Problems	F	R <sup>2</sup>	R <sup>2</sup>	B	95% CI
Adherence				-2.66*	[-4.92, -.40]
MI				.16**	[.06, .26]

Abbreviations: BRI, Behavior Regulation Index; GEC, global executive composite; MI, Metacognition Index.

\*  
 $p < .05$

\*\*  
 $p < .01$

\*\*\*  
 $p < .001$

<sup>1</sup>Please note Steps 1, 2, and 3 were the same for all three models, with Step 4 adding each executive function variable individually into the model.

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