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Fear of hypoglycemia, parenting stress, and metabolic control for children with type 1 diabetes and their parents

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Abstract

This study sets out to extend current knowledge of parenting stress and fear of hypoglycemia (FoH) in parents of children with type 1 diabetes mellitus (T1DM). We examined if the relationship between parental and children's FoH and metabolic control, as reflected by HbA1c, is mediated by parenting stress. A total of 63 parents and children with T1DM were recruited during their routine physician's appointment. Parents completed questionnaires on parenting stress and FoH. Children eight years and older also completed a questionnaire on FoH. HbA1c values were obtained from all children. Mediation analysis revealed an indirect association between parental FoH and HbA1c values through parenting stress (Sobel's $z = 2.42$, $p = .02$), but no indirect association between children's FoH and HbA1c. We concluded that parental FOH has an indirect association with the child's metabolic control that is mediated by parenting stress. More simply, fear of hypoglycemia predicts parent stress, which in turn, predicts metabolic control.

Key words

fear of hypoglycemia, parenting stress, metabolic control, type 1 diabetes mellitus

Introduction

In the past several years, many countries have reported a rapidly increasing incidence of childhood type 1 diabetes mellitus (T1DM; Craig, Jefferies, Dabelea, Blade, Seth, & Donaghue, 2014; Patterson, Dahlquist, Gyürüs, Green, Soltész, & the EURODIAB <study group, 2009). Furthermore, the age of onset also appears to have shifted: more children are being diagnosed with T1DM at a younger age and are undergoing treatment (Berhan, Waernbaum, Lind, Möllsten, & Dahlquist, 2011). This treatment, however, is not without risks. The Diabetes Complications and Controls Trial (DCCT Research Group, 1991) concluded that intensive treatment of T1DM is accompanied by a threefold increase in severe hypoglycemia compared with conventional therapy. This relationship between better glycemic control and increased hypoglycemia holds a significant challenge for children and their parents attempting to achieve optimal glycemic control (Gonder-Frederick, Nyer, Shepard, Vajda, & Clarke, 2011).

Hypoglycemia is usually defined as a blood glucose level < 65 mg/dL. However, in clinical practice, a glucose value < 70 mg/dL is used as the threshold value for initiating treatment for hypoglycemia (Ly, Maahs, Rewers, Dunger, Oduwole, & Jones, 2014). Hypoglycemia is the most common acute complication in patients with T1DM (Becker & Ryan, 2000), which can be described as:

> … a common adverse event associated with insulin treatment in type 1 and type 2 diabetes. It can occur suddenly and is characterized by unpleasant physical and psychological symptoms such as shaking, sweating, drowsiness, nausea, poor motor coordination, mental confusion, negative mood and unconsciousness (Wild, von Maltzahn, Brohan, Christensen, Clauson, & Gonder-Frederick, 2007, p. 10).

Furthermore, hypoglycemia can result in morbidity and sometimes in death. At the very least, symptoms are uncomfortable and carry the fear of loss of control, or loss of consciousness (Becker & Ryan, 2000).

Because the symptoms of hypoglycemia are aversive and also possibly dangerous, they could result in fear (Irvine, Cox, & Gonder-Frederick, 1992) and/or lead to the development of phobic avoidance behaviors (Cox, Irvine, Gonder-Frederick, Nowacek, & Butterfield, 1987). This specific type of fear is known as fear of hypoglycemia (FoH). Such high fear might be adaptive in the presence of high risk of hypoglycemia, as it promotes monitoring of appropriate behaviors to avoid hypoglycemic episodes, whereas low fear or denial could be maladaptive when risk of hypoglycemia is high (Irvine, Cox, & Gonder-Frederick, 1994). However, excessive fear in situations of low risk may lead to inappropriate responses. Patients may engage in preventive attempts to control hypoglycemia, e.g. by keeping blood glucose levels elevated or engaging in overtreatment of low blood glucose levels (Cox et al., 1987). Irvine et al. (1992) noticed that patients with higher daily mean blood glucose levels showed lower fear. This may have important implications for adherence and for metabolic control because hypoglycemia is one of the major barriers to achieve and maintain near normal blood glucose levels in

T1DM (Beléndez & Hernández-Mijares, 2009; Cryer, Davis, & Qhamoon, 2003). This barrier is a challenge for the children, but also for their parents (Gonder-Frederick et al., 2011). The unpleasant symptoms related to T1DM might not only frighten a child, but also the child's parents. Indeed, there is some evidence for the association between FoH and worse glycemic control (Hawkes, McDarby, & Cody, 2014; Freckleton, Sharpe, & Mullan, 2014; Patton, Dolan, Henry, & Powers, 2007). For some individuals FoH might therefore be considered as maladaptive, as it can have significant implications for diabetes management, glycemic control, quality of life, and emotional wellbeing (Cox et al., 1987; Gonder-Frederick et al., 2006; Wild et al., 2007).

Managing the diabetes of a child with T1DM induces high levels of self-reported anxiety in most parents (Barnard, Thomas, Royle, Noyes, & Waugh, 2010). There are a number of factors that influence whether parents will experienceFoH. Firstly, parents who believe that their child is able to cope with a hypoglycemic episode, e.g., by consuming glucose, are likely to have lower FoH. Secondly, FoH does not appear to be related to the frequency of hypoglycemia, but rather to the severity of past hypoglycemic events, especially if the child had convulsions (Barnard et al., 2010). Similar findings were also reported by Clarke, Gonder-Frederick, Snyder, and Cox (1998): FoH in mothers did not appear to be related to the number of hypoglycemic episodes in the past year, but mothers whose child passed out during a hypoglycemic episode (an indication of higher severity) had higher scores on the Parents Hypoglycemia Fear Survey (PHFS), compared to mothers whose child never passed out. Thirdly, the diagnosis of childhood T1DM (and the related FoH) is likely to increase the stressfulness of family-life, and parents' anxiety. Parents of children with T1DM do have higher parenting stress compared to healthy control subjects (Powers, Byars, Mitchell, Patton, Standiford, & Dolan, 2002). Illness-related variations in parenting stress influences the whole family, both parents and children (Streisand, Braniecki, Tercyak, & Kazak, 2001), and the resulting increase in family life stress can in turn affect the treatment of the child with diabetes. For example, poor family relations are associated with poorer adherence (Hanson, De Guire, Schinkel, & Kolterman, 1995).

Previous research has already focused on the relationship between children's glycemic control and parental FoH and emotional distress. More specifically, a study conducted by Haugstvedt, Wentzel-Larsen, Graue, Sovik, and Rokne (2010) in Norway found an association between a higher level of parental FoH and emotional distress and poorer glycemic control in children.

The current study set out to extend existing evidence by providing additional cross-cultural data from a Belgian sample, and by further mapping the complex interplay between parental stress, FoH, and glycemic control. More specifically, we explored whether there is an association between FoH and metabolic control and whether this relationship is mediated by parenting stress. We hypothesized that when either parents or children have higher FoH, parents will also experience more parenting stress. In

turn, this stressful parenting climate will be associated with children's having increased HbA1C levels (i.e., decreased glycemic control).

Methods

Study population

Families who participated in this study were recruited from the Childhood Diabetes Centre at Jessa Ziekenhuis, Hasselt, Belgium. They were eligible for participation if: they were Dutch-speaking; their child was between 2 and 18 years of age and had a confirmed diagnosis of T1DM for at least six months; if the child received intensive insulin treatment (e.g., insulin pump or multiple daily injections); and if one parent was willing to complete a number of questionnaires. The minimal duration of six months since diagnosis was set to assure that participating families had ample time to adjust to both the diagnosis and the treatment. Out of 99 families, a total of 85 were eligible for participation. Sixty-three families consented to participate in the study, reflecting a recruitment rate of 74%. All children who were eight years or older (*n* = 56) completed the questionnaire. The primary reason for declining participation was that the child showed no interest in participating (50%).

Procedure

Participants were recruited from the outpatient pediatric endocrinology clinic. Children and their parents were informed by their physician, during a routine diabetes clinic appointment, about the possibility of participating in a study on hypoglycemia. Informed consent was obtained from the parents and from patients older than 12 years, who received an adapted consent form for approval. In the consent, both children (above the age of eight) and parents agreed to fill in self-report questionnaires. They also agreed that the results of the glycosylated HbA1c analysis could be used in the study. If they did not have time to participate at the day of their appointment, they completed the questionnaires at home and sent them back using a prestamped envelope. Participants who had not returned their questionnaires several weeks later were contacted by phone and encouraged to still complete and return them. Both the study protocol and the informed consent forms were approved by the ethics committee of the Jessa Ziekenhuis facility.

Instruments

Parenting Stress-Index (NPSI-S)

The Nijmegen Parenting Stress Index-Short form (NPSI-S, De Brock, Vermulst, Gerris, & Abidin, 1992) was used to assess parenting stress. This official Dutch translation and adaptation of the Parenting Stress Index (PSI; Abidin et al., 1983 in De Brock et al., 1992) is a short version developed for screening parenting stress. This version consists of 25 items divided into a "parent domain" (11 items) and a "child domain" (14 items). The "parent domain" refers to perceived stress regarding family factors (e.g., "Parenting this child is more difficult for me than I expected" or "I often have the feeling that I cannot handle things very well"). The "child domain" refers to stress evoked by the child's behavior and emotions (e.g., "It is difficult for me to accept my child as it is" or "My child makes more demands on me than most children"). The items are scored on a six-point Likert scale ranging from 1 = *totally disagree* to 6 = *totally agree*. The total score is computed by summing up all item scores; total scores range from 25 to 150. Higher scores indicate greater parenting stress. The questionnaire shows good criterion validity (De Brock et al., 1992), and high internal consistency was found in this study (α = .96).

Fear of Hypoglycemia

The Parent's fear of hypoglycaemia scale (HFS-P; Clarke et al., 1998) is a modified version of the Hypoglycemia Fear Survey (HFS; Cox et al., 1987) for use with parents. We developed a Dutch version with back-translation by an independent bilingual translator. The HFS consists of 25 items to measure parents' worries (15 items) and behaviors used to avoid hypoglycemia (10 items). The "worry" subscale was designed to measure anxieties concerning negative consequences of hypoglycemia (e.g., "I am worried that my child does not recognize/realize he/she is having a reaction" or "I am worried that no one is around to help my child during a reaction"). Higher scores on the "worry" subscale indicate greater worry. The "behavior" subscale was designed to measure specific behaviors which might be taken to avoid hypoglycemia (e.g., "Have your child eat a large snack at bedtime" or "Have your child carry fast-acting sugar"). Higher scores on the "behavior" subscale are taken as an indicator of greater fear of hypoglycemic episodes. The original HFS-P has been revised and now includes an additional item on the behavior-subscale about parents checking on their child while the child is asleep (K. Vajda, personal communication, November 27, 2012). Items were rated on a 5-point Likert scale (1 = *never* to 5 = *very often*). By summing up all items, a total HFS-P score and a score on the worry and behavior subscales can be obtained (Gonder-Frederick, Fisher,Ritterband, Cox, Hou, DasGupta, & Clarke, 2006). The total HFS-P score was used in all subsequent analyses in which parental FoH was a predictor. In the current study, the mean of items was used as the total score, i.e., the respondent's score on each of the 25 items were summed and then divided by 25 to yield a total score that could range from a minimum

of 1 to a maximum of 5. Using this average avoided the artificial inflation of the sum of item scores that would have resulted due to the recent addition of a new item. In this study, the survey demonstrated good internal consistency (α = .86).

The Children's fear of hypoglycemia scale (HFS-C; Gonder-Frederick et al., 2006; Gonder-Frederick et al., 2011) is another adaptation of the HFS, which has been made appropriate for children. Similarly as with the HFS-P, a Dutch version was developed using back-translation by an independent bilingual translator. This survey has the same structure as the adult version, with two subscales: a worry (15 items) and behavior subscale (10 items). The "worry" subscale measures anxieties concerning negative consequences of hypoglycemia (e.g., "Getting in trouble at school because of something that happens when my sugar is low" or "Making a mistake or having an accident at school"). The behavior subscale measures specific behaviors which might be taken to avoid hypoglycemia (e.g., "Keep blood sugars a little high to be on the safe side" or "Eat large snacks at bedtime"). HFS-C items are also rated on a 5-point Likert scale, ranging from 1 = *never* to 5 = *always*. As with the Parent version of the HFS, items for the "worry" subscale and the "behavior" subscale were combined to form a single composite measure of the child's fear of hypoglycemia. This combined index was used in all subsequent analyses in which Child FoH was a predictor. For this survey we also used overall mean of the 25 items. Reliability analysis showed an average internal consistency (α =.68).

HbA1c values

HbA1c values were obtained from medical records and the time point of measurement nearest to the date of participation was used. These values reflect the average blood glucose control of the past several months, with poorer control showing in higher levels of HbA1c values (Beléndez & Hernández-Mijares, 2009). All blood samples were analyzed in the hospital laboratory using a Siemens ADVIA 120/2120/2120i. The recommended value of HbA1c is < 7.5% for children and adolescents with T1DM (Rewers, Pillay, de Beaufort, Graig, Hanas, Acerini, & Maahs, 2014).

Statistical Analysis

All data were analyzed with SPSS (SPSS 20.0, IBM) and a freely-available macro for SPSS: PROCESS (Hayes, 2013). Preliminary analyses were carried out to compute descriptive statistics such as means and standard deviations. Pearson's correlation coefficients were calculated to examine relationships among the study variables (parenting stress index, parental FoH, childrens FoH, and HbA1c values). We used multivariate analysis of covariance (MANCOVA) to assess the impact of some group factors (gender, age and time in years since diagnosis) on the study variables. Using the PROCESS tool, we conducted separate mediation analyses (i.e., PROCESS model 4) to examine the direct effects of children's FoH and parental FoH on the outcome variable, HbA1c, and the indirect effect of parenting

stress on the FoH/HbA1c relationship. To estimate the indirect effects and construct confidence intervals (CI), we used a non-parametric resampling procedure: bootstrapping. This test can be applied to small samples with more confidence and has the advantage that it makes no assumptions about the shape of the distribution and decreases the chance of both Type I and Type II errors (Preacher & Hayes, 2004). Each analysis used 5,000 bootstrapped samples. A p-value of <.05 was considered as significant and the mediation effect is considered significant if the 95% confidence interval (CI) does not include zero.

Results

Participant profile

Out of the 85 families that were contacted, 22 declined participation. The mean age of children of families that declined participation (M = 14.36, SD = 3.81) was higher than the mean age of those children who participated, M = 12.36, SD = 3.90, $t(83) = 2.09$, p = .04. The mean number of years since diagnosis was also longer for families that participated (M = 5.86, SD = 3.81) compared to those families that did not, M = 4.07, SD = 2.61, $t(83) = 2.44$, p = .017. No other socio-demographic variables from the refusing participants were available for analysis. An overview of the demographic variables of participating children and parents can be found in Table 1.

Preliminary analyses

Pearson correlation coefficients of study variables are presented in Table 2. A significant correlation was found between parenting stress and parental FoH, and between parenting stress and HbA1c values of the child. The correlations between parental FoH and HbA1c, and between children's FoH and HbA1c, were not significant.

Parental FoH

A multivariate test showed that age, $F(3,53) = 1.49$, p = .23, time since diagnosis, $F(3,53) = 1.37$, p = .26, child gender, $F(3,53) = 0.60$, p = .62, and parent gender, $F(3,53) = 1.93$, p = .14, were unrelated to parental FoH, parenting stress, or HbA1c, and so these were not entered as covariates in the structural model.

Mediation analysis

The upper half of Table 3 and Figure 1A present results for the regression tests of the mediation analysis for parent FoH. The total effect, i.e., the association between parental FoH and HbA1c without taking parenting stress into account (path c), was not significant. There was also no significant direct association between parental FoH on HbA1c, with parenting stress taken into account (path c').

However, the path between parental FoH and parenting stress (path *a*), and the path between parenting stress and HbA1c (path *b*), were both significant and positive. Moreover, the indirect association between parental FoH and HbA1c through parenting stress (path *ab*), also was significant according to a Sobel's test, Sobel's $z = 2.42$, $p = .02$.

Children's FoH

A multivariate test showed that: age, $F(3,47) = 1.32$, $p = .28$; time since diagnosis, $F(3,47) = 1.63$, $p = .20$; child gender, $F(3,47) = 0.43$, $p = .73$; and parental gender, $F(3,47) = 1.38$, $p = .26$, had no significant effect on children's FoH, parenting stress, and HbA1c. Consequently, these variables were not entered as covariates in the structural model. To further determine whether covariates should be added to the model, univariate tests were conducted. With one exception, these tests found no significant associations between potential covariates and children's FoH, parenting stress, and HbA1c. However, time since diagnosis and children's FoH were significantly associated, $F(1,49) = 4.65$, $p = .036$. Therefore, the mediation analysis for children was redone using a modified version of the children's FoH variable that controlled for time since diagnosis. Results from this additional mediation analysis were consistent with those reported below.

Mediation analysis

A visual representation of the child data is shown in Figure 1B. The lower half of Table 3 presents results for the regression tests of the mediation analysis. The total effect, i.e., the association between children's FoH and HbA1c without taking parenting stress into account (path *c*), was not significant. The association between children's FoH and HbA1c, with parenting stress taken into account (path *c'*), also was not significant. The association between children's FoH and parenting stress (path *a*) also was not significant, but there was a significant association between parenting stress and HbA1c (path *b*). The indirect effect from children's FoH to HbA1c through parenting stress (path *ab*) was not significant, Sobel's z = .05, $p = .57$.

Discussion

The goal of this study was to extend current knowledge about the influence of parenting stress on the relationship of parental FoH, and child FoH, to metabolic control in children with T1DM. Results show that parental FoH was not directly related to metabolic control. However, an indirect relationship was found in which parental FoH was associated with HbA1c through parenting stress; greater parental FoH was associated with more parenting stress, which in turn was associated with increased HbA1c. As for children's FoH and glycemic control, no relationships were found, neither direct nor mediated

through parenting stress. The latter is not surprising as only parenting stress (and not children's stress) was measured. The lack of a direct relationship between FoH and poor hypoglycemic control also seems to provide some evidence for the hypothesis that FoH may to some extent be adaptive, as it encourages frequent monitoring (Irvine, Cox, & Gonder-Frederick, 1994). In any case, this study puts an additional focus on parental stress as the mechanism of interest through which parental FoH, but not child FoH, results in poor glycemic control.

There are a number of methodological limitations to this study. First, due to privacy restrictions we were able to collect only limited demographic information about non-participating families, and therefore could not conduct more extensive comparisons between families who participated and those who declined to participate. A second limitation relates to the measured constructs: we made use of self-report measures, which are prone to bias and we did not include a measure for general trait anxiety or social fear. Other research suggested that adherence of adolescents with T1DM might be influenced by social anxiety (Di Battista, Hart, Greco, & Gloizer, 2009). Gonder-Frederick et al. (2006) reported that trait anxiety is a predictor of FoH in adolescents, but not of parental FoH. Furthermore, these authors found a relationship between parental trait anxiety and child trait anxiety. However, we opted not to include such a measure, not only because the relationship with FoH is rather complex (Wild, von Maltzahn, Brohan, Christensen, Clauson, & Gonder-Frederick, 2007), but because we also wanted to limit the scope of the study. A third limitation is our relatively small sample, which might hamper the validity of our findings. A fourth and final limitation is the cross-sectional nature of our design, which does not allow us to determine the direction of causality in the relationship between parenting stress and glycemic control.

Several recommendations for future research can therefore be made. First, the subjective experience of both parental and children's FoH might not only have a psychological component – which has been measured through self-report questionnaires – but also a behavioral one. Future studies could expand the scope of self-report measures (e.g., by also measuring children's stress) and also explore FoH using external observations, e.g., by relying on semi-structured interviews for the psychological component and observations for the behavioral one. The complex interplay between the different variables in our mediation model could furthermore be explored using a longitudinal study design, which would shed light on the direction of the reported relationships. Second, although our limited sample was able to produce significant results, future studies would benefit from larger samples in order to reliably replicate our findings. Third, based on this study, some recommendations can also be made related to measures used for exploring FoH and parenting stress. As for behavioral measures, a more direct measurement of treatment adherence would allow one to explore whether and how FoH could be somewhat adaptive, leading to improved diabetes management (e.g., by an increased frequency of

checking blood glucose levels). As for biochemical measures, HbA1c might lack the sensitivity to fully detect the complex patterns of glucose variability, data which could perhaps be supplemented and captured more adequately by also including blood glucose levels and/or continuous glucose monitoring data.

The findings of the current study have important implications for clinical practice. The positive relationship between parenting stress and glycemic control points to the importance of psychological support for parents of children with T1DM, not only at the time of diagnosis, but also over the entire span of their child's treatment. Addressing factors that contribute to the parent-child relationship may also foster more positive interactions and help youth become more independent in their diabetes care (Sweenie, Mackey &, Streisand, 2014). A practical intervention for parents would be to help them manage difficult child behavior and cope with parenting stress, with the goal of improving parental management of the child's T1DM. Although it is hard to find a tailored approach for this specific group of parents, Guthrie, Sargent, Speelman, and Leland (1990) suggested the importance of assessing the stress levels and psycho-education on stress in families with a child with T1DM because the diagnosis has an effect on the whole family system. Our findings also suggest that clinical providers should exercise caution in efforts to reduce parental FoH because FoH may be adaptive to some extent. FoH may function as a warning that keeps parents focused on the importance of maintaining their child's HbA1c in a safe range. Yet, such potential benefits of FoH may be outweighed by the negative consequences of FoH, especially for parents who are already experiencing high levels of parental stress.

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Compliance with Ethical Standards Conflict of Interest Ann-Sofie Viaene, Tom Van Daele, Dries Bleys, Kelly Faust, and Guy G. Massa declare that they have no conflict of interest.

Human and animal rights and informed consent All procedures performed in studies involving human participants were in accordance with the ethical standards of the ethics committee of the Jessa Ziekenhuis facility and with the 1964 Helsinki declaration, as revised in 2000. Informed consent was obtained from all patients for being included in the study.

References

Barnard, K., Thomas, S., Royle, P., Noyes, K., & Waugh, N. (2010). Fear of hypoglycamia in parents of young children with type 1 diabetes: a systematic review. *BMC Pediatrics, 10*, 1471-2431.

Becker, D. J., Ryan, C.M. (2000). Hypoglycemia: A complication of diabetes therapy in children. *Trends in endocrinology and metabolism, 11*(5), 198-201.

Beléndez, M. & Hernández-Mijares, A. (2009). Beliefs about insulin as a predictor of fear of hypoglycaemia. *Chronic Illness, 5*(4), 250-256.

Berhan, Y., Waernbaum, I., Lind, T., Möllsten, A., & Dahlquist, G. (2011). Thirty years of prospective nationwide incidence of childhood type 1 diabetes the accelerating increase by time tends to level off in Sweden. *Diabetes, 60*(2), 577-581.

Clarke, W. L., Gonder-Frederick, L. A., Snyder, A. L., & Cox, D. J. (1998). Maternal fear of hypoglycaemia in their children with insulin dependent diabetes mellitus. *Journal of Pediatric Endocrinology & Metabolism, 11,* 189-194.

Cox, J. D., Irvine, A., Gonder-Frederick, L., Nowacek, G., & Buttefield, J. (1987). Fear of hypoglycemia: Quantification, validation and utilization. *Diabetes Care, 10*(5), 617-621.

Craig, M. E., Jefferies, C., Dabelea, D., Balde, N., Seth, A., & Donaghue, K. C. (2014). Definition, epidemiology, and classification of diabetes in children and adolescents. *Pediatric Diabetes, 15*(20), 4-17.

Cryer, P. E., Davis, S. N., & Qhamoon, H. (2003). Hypoglycemia in diabetes. *Diabetes Care, 26*(6), 1902-1912.

DCCT Research Group. (1991). Epidemiology of severe hypoglycemia in the diabetes control and complications trial. *The American Journal of Medicine, 90*(4), 450-459.

De Brock, A. J. L. L., Vermulst, A. A., Gerris, J. R. M., & Abidin, R. R. (1992). *Nijmeegse ouderlijke stress index, Handleiding experimentele versie.* Lisse: Swets en Zeitlinger.

Di Battista, A. M., Hart, T. A., Greco, L., & Gloizer, J. (2009). Type 1 diabetes among adolescents reduced diabetes self-care caused by social fear and fear of hypoglycemia. *The Diabetes Educator, 35*(3), 465-475.

Freckleton, E., Sharpe, L., & Mullan, B. (2014). The relationship between maternal fear of hypoglycaemia and adherence in children with type-1 diabetes. *International Journal of Behavioral Medicine, 21*(5), 804-810.

Gonder-Frederick, L. A., Fisher, C. D., Ritterband, L.M., Cox, D. J., Hou, L., DasGupta, A. A., & Clarke, W.L. (2006). Predictors of fear of hypoglycemia in adolescents with type 1 diabetes and their parents. *Pediatric Diabetes, 7*(4), 215-222.

Gonder-Frederick, L., Nyer, M., Shepard, J. A., Vajda, K., & Clarke, W. (2011). Assessing fear of hypoglycemia in children with type 1 diabetes and their parents. *Diabetes Management, 1(*6), 627-639.

Guthrie, D. W., Sargent, L., Speelman, D., & Parks, L. (1990). Effects of parental relaxation training on glycosylated hemoglobin of children with diabetes. *Patient Education and Counseling, 16*(3), 247-253.

Hanson, C. L., De Guire, M. J., Schinkel, A. M., & Kolterman, O. G. (1995). Empirical validation for a family-centered model of care. *Diabetes Care, 18*(10), 1347-1356.

Hawkes, C. P., McDarby, V., & Cody, D. (2014). Fear of hypoglycemia in parents of children with type 1 diabetes. *Journal of Paediatrics and Child Health, 50*(8), 639-642.

Haugstvedt, A., Wentzel-Larsen, T., Graue, M., Søvik, O., & Rokne, B. (2010). Fear of hypoglycaemia in mothers and fathers of children with Type 1 diabetes is associated with poor glycaemic control and parental emotional distress: A population-based study. Diabetic Medicine, 27(1), 72-78.

Hayes, A. F. (2013). *Introduction to mediation, moderation, and conditional process analysis.* New York: Guilford Publications, Inc.

Irvine, A. A., Cox, D., & Gonder-Frederick, L. (1992). Fear of hypoglycemia: Relationship to physicaland psychological symptoms in patients with insulin-dependent diabetes mellitus. *Health Psychology, 11*(2), 135-138.

Irvine, A. A., Cox, D., & Gonder-Frederick, L. (1994). The fear of hypoglycaemia scale. In C. Bradley (Ed.), *Handbook of psychology and diabetes: A guide to psychological measurement in diabetes research & practice (pp. 133-155*). Hove, UK: Psychology Press.

Ly, T.T., Maahs, D.M., Rewers, A., Dunger, D., Oduwole, A., & Jones, T.W. (2014). Assessment and management of hypoglycemia in children and adolescents with diabetes. *Pediatric Diabetes, 15*(Suppl. 20), 180-192

Patterson, C. C., Dahlquist G. G., Gyürüs E., Green A., Soltész G., & the EURODIAB study group (2009). Incidence trends for childhood type 1 diabetes in Europe during 1989-2003 and predicted new cases 2005-20: A multicentre prospective registration study. *The Lancet, 373*(9680), 2027-2033.

Patton, S. R., Dolan, L. M., Henry, R., & Powers, S. W. (2007). Parental fear of hypoglycemia: Young children treatedwith continuous subcutaneous insulin infusion. *Pediatric Diabetes, 8*(6), 362-368.

Patton, S.R., Dolan, L.M., Henry, R., & Powers, S.W. (2008). Fear of hypoglycemia in parents of young children with type 1 diabetes mellitus. *Journal of Clinical Psychology in Medical Settings, 15*(3), 252-259.

Patton, S. R., Dolan, L. M., Smith, L. B., Thomas, I. H., & Powers, S. W. (2011). Pediatric parenting stress and its relation to depressive symptoms and fear of hypoglycemia in parents of young children with type 1 diabetes mellitus. *Journal of Clinical Psychology in Medical Settings,*

18(4), 345-352.

Powers, S. W., Byars, K. C., Mitchell, M. J., Patton, S. R., Standiford, D. A., & Dolan, L. M. (2002). Parent report of mealtime behavior and parenting stress in young children with type 1 diabetes and in healthy control subjects. *Diabetes Care, 25*(2), 313-318.

Preacher, K.J., & Hayes, A.F. (2004). SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods, Instruments, & Computers, 36*(4), 717-731.

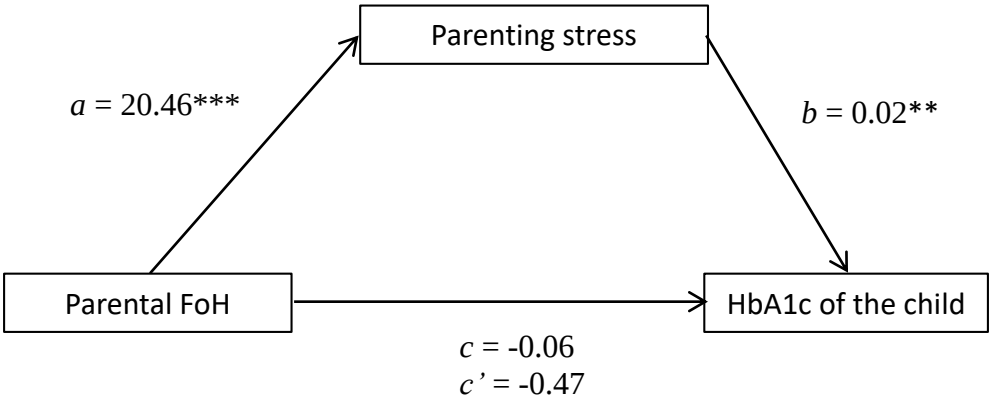
Rewers, M. J., Pillay, K., de Beaufort, C., Craig, M. E., Hanas, R., Acerini, C. L., & Maahs, D. M. (2014). Assessment and monitoring of glycemic control in children and adolescents with diabetes. *Pediatric Diabetes, 15*(20), 102-114.

Streisand, R., Swift, E., Wickmark, T., Chen, R., Holmes, C. S. (2005). Pediatric parenting stress among parents of children with type 1 diabetes: The role of self-efficacy, responsibility, and fear. *Journal of Pediatric Psychology, 30*(6), 513-521.

Sweenie, R., Mackey, E. R., & Streisand, R. (2014). Parent–child relationships in type 1 diabetes: Associations among child behavior, parenting behavior, and pediatric parenting stress. *Families, Systems, and Health, 32*(1), 31-42.

Wild, D., von Maltzahn, E., Brohan, E., Christensen, T., Clauson, P., & Gonder-Frederick, L. (2007). A critical review of the literature on fear of hypoglycemia in diabetes: implications for diabetes management and patient education. *Patient Education and Counseling, 68,* 10-15.

MODEL 1A



MODEL 1B

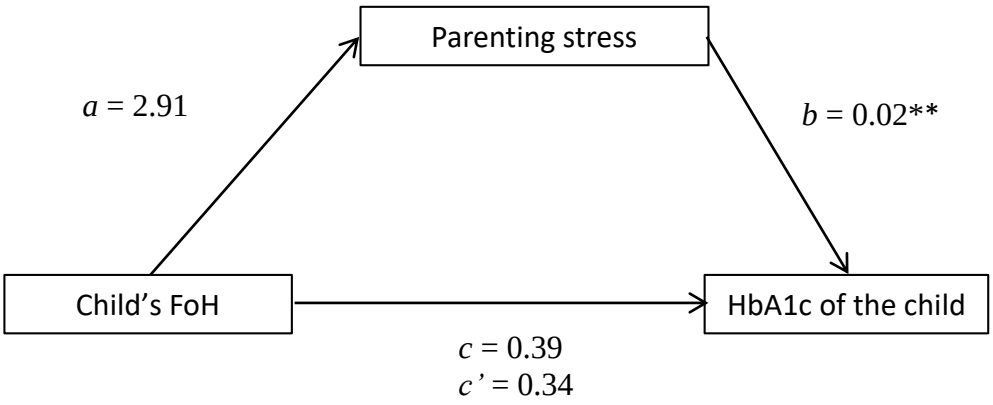


Figure 1. Model of parental FoH as a predictor of HbA1c values mediated by parenting stress (1a), and model of child's FoH as a predictor of HbA1c values mediated by parenting stress (1b). *a*, *b*, *c* (total effect), and *c'* (direct effect) are unstandardized regression coefficients. * $p < .05$, ** $p < .01$, *** $p < .001$

Table 1

Profile of participating children (N = 63) and parents (N = 63)

Variable	*M*	*SD*	*n (%)*
Children			
Child gender (female)			28 (44.48)
Age (years)	12.36	3.90	
Age at diagnosis (years)	8.29	3.89	
Time since diagnosis (years)	4.07	2.61	
Blood glucose (HbA1c)	8.28	1.07	
Child FoH (HFS-C)	1.41	.71	
Parents			
Parent gender (female)			53 (84.1)
Marital status (married)			48 (76.20)
Parenting FoH (HFS-P)	1.65	.51	
Parenting Stress (NPSI-S)	52.23	24.27	

Table 2

Pearson correlation coefficients among study variables

Measure		1	2	3	4
1. Parenting Stress	(NPSI-S)	-	.43**	.08	.36**
2. Parenting FoH	(HFS-P)	-	-	.10	-.03
3. Child FoH	(HFS-C)	-	-	-	.25
4. Blood glucose	(HbA1c)	-	-	-	-

Note. **$p < .01$

Table 3

Regression results of the mediation analyses

Variable	*b*	*SE*	*t*	*p*	95% CI
Parents					
Path *a*: Parental FoH -> Parenting stress	20.462	5.648	3.623	< .001	[9.156, 31.767]
Path *b*: Parenting stress -> HbA1c of the child	0.020	0.006	3.364	.001	[0.008, 0.032]
Path *c*: Parental FoH -> HbA1c of the child	- 0.061	0.277	- 0.220	.827	[-0.616, 0.494]
Path *c'*: Parental FoH -> HbA1c of the child	- 0.470	0.283	- 1.660	.102	[-1.036, 0.097]
Children					
Path *a*: Child's FoH -> Parenting stress	2.910	4.783	0.608	.546	[-6.688, 12.508]
Path *b*: Parenting stress -> HbA1c of the child	0.017	0.006	2.961	.005	[0.005, 0.028]
Path *c*: Child's FoH -> HbA1c of the child	0.387	0.210	1.841	.071	[-0.035, 0.809]
Path *c'*: Child's FoH -> HbA1c of the child	0.338	0.197	1.718	.092	[-0.057, 0.733]

Note. Paths *a* and *b* are the mediation analyses paths. Path *c* is the total effect without including the mediator in the model. Path *c'* is the direct effect when the mediator is included in the model.