



Eating Disorders in Girls and Women With Type 1 Diabetes: A Longitudinal Study of Prevalence, Onset, Remission, and Recurrence

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OBJECTIVE

Girls and women with type 1 diabetes are at increased risk for developing eating disorders (EDs), and these disorders are associated with serious diabetes-related medical complications. This study describes the longitudinal course of disturbed eating behavior (DEB) and EDs in a cohort with type 1 diabetes.

RESEARCH DESIGN AND METHODS

A total of 126 girls with type 1 diabetes receiving care for diabetes at The Hospital for Sick Children in Toronto participated in a series of seven interview-based assessments of ED behavior and psychopathology over a 14-year period, beginning in late childhood. Survival analysis was used.

RESULTS

Mean age was 11.8 ± 1.5 years at time 1 and 23.7 ± 2.1 years at time 7. At time 7, 32.4% (23/71) met the criteria for a current ED, and an additional 8.5% (6/71) had a subthreshold ED. Mean age at ED onset (full syndrome or below the threshold) was 22.6 years (95% CI 21.6–23.5), and the cumulative probability of onset was 60% by age 25 years. The average time between onset of ED and subsequent ED remission was 4.3 years (95% CI 3.1–5.5), and the cumulative probability of remission was 79% by 6 years after onset. The average time between remission of ED and subsequent recurrence was 6.5 years (95% CI 4.4–8.6), and the cumulative probability of recurrence was 53% by 6 years after remission.

CONCLUSIONS

In this longitudinal study, EDs were common and persistent, and new onset of ED was documented well into adulthood. Further research regarding prevention and treatment for this vulnerable group is urgently needed.

The prevalence, clinical characteristics, and medical consequences of disturbed eating behavior (DEB) and eating disorders (EDs) in individuals with type 1 diabetes has received increasing attention since case reports of this dangerous combination were first published in the 1980s (1,2). Although the specificity of this association was initially unclear, systematic research has demonstrated that teenage girls and women with type 1 diabetes are at significantly increased risk of DEB compared with their nondiabetic peers (3). Such DEB includes dieting, fasting, binge-eating, and a range of compensatory and purging behaviors that can directly interfere with optimal diabetes management.

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Deliberately underdosing or omitting insulin to induce hyperglycemia and loss of glucose in the urine, and thereby control weight, is a unique purging behavior to control weight that is available to individuals with type 1 diabetes (4). This is an important mediator of the association of DEB and EDs with poorer metabolic control (5,6) and contributes to an increased risk of a range of short-term and long-term diabetes-related medical complications. These include abnormal lipid profiles (7), diabetic ketoacidosis (6), retinopathy (8), neuropathy (9), and nephropathy (10), as well as higher than expected mortality (11).

Several longitudinal studies of DEB and EDs in individuals with type 1 diabetes have been conducted. Pollock et al. (12) assessed eating problems in a group of 79 boys and girls, 8–13 years of age, at type 1 diabetes onset, over a period of up to 14 years. They found eating problems to be highly associated with psychiatric disorders and with pervasive noncompliance with medical treatment. Rydall et al. (8) found that DEB at the study baseline among a cohort of adolescent girls with type 1 diabetes predicted a tripled risk of retinopathy 4 years later. In a third study, Bryden et al. (13) assessed a group of individuals with type 1 diabetes in adolescence and then again in early adulthood. They found that rates of overweight, concern over weight and shape, and heightened dietary restraint all increased from adolescence to adulthood. DEB was common among the female participants, though it did not necessarily meet diagnostic thresholds for anorexia nervosa or bulimia nervosa, as did insulin underdosing for weight control. They found EDs or other significant eating problems in 26% of participants, as well as significant associations between eating problems, insulin misuse, and microvascular complications (14). Goebel-Fabbri et al. (15) assessed 234 adult women with type 1 diabetes twice over an 11-year period. They found insulin omission for weight control to be very common (reported by 30% at baseline). Insulin omission frequently persisted over the lengthy follow-up period and was associated with higher rates of diabetes-related medical complications and tripled risk of mortality.

Finally, in earlier stages of the study (study baseline and 1- and 5-year follow-up [16–18]) described in this report, DEB

was usually mild but frequent and persistent among girls with type 1 diabetes during late childhood and adolescence. Earlier in the follow-up of this cohort, DEB was associated with higher BMI but not with poorer metabolic control. This report describes the prevalence, onset, remission, and recurrence of DEB and EDs in a cohort of girls with type 1 diabetes recruited in late childhood and followed into early adulthood. This 14-year, longitudinal study encompasses the peak age range of DEB and ED onset. It adds to the existing longitudinal literature in this high-risk group by using up to 7 assessments over the study period to calculate detailed estimates of the course of these disturbances during this crucial developmental period.

RESEARCH DESIGN AND METHODS

This study was reviewed and approved by the research ethics boards at The Hospital for Sick Children and University Health Network, Toronto, Canada. Participants were initially recruited from the Diabetes Clinic at The Hospital for Sick Children in Toronto during 1998 to 2001. All girls 9–13 years of age with a type 1 diabetes duration >6 months at the study baseline (time 1) and fluency in English were invited to participate. Participants were assessed at time 1 (study baseline) as well as on six subsequent occasions. These occurred 1 (time 2), 2 (time 3), 3 (time 4), 5 (time 5), 8–10 (time 6), and 10–14 (time 7) years after time 1. At each assessment point, participants completed a semistructured diagnostic interview, which was administered by a trained interviewer. At times 1–4, the Children's Eating Disorder Examination (cEDE) (19) was used, and at times 5–7 the Eating Disorder Examination (EDE) (20) was administered. The EDE is a well-validated interview tool (21) widely used to identify and rate the psychological and behavioral disturbances that occur in individuals with EDs and is considered the gold standard for interview assessment of EDs. The cEDE is psychometrically identical to the EDE, but the language has been slightly modified for use in children 7–14 years of age. The period of inquiry for the cEDE and EDE was the past 3 months, with greatest detail for the 28 days before the interview. This assessment schedule resulted in seven “snapshots” over time rather than continuous information

about ED symptoms over the 14-year follow-up period.

DEB, EDs, and subthreshold EDs were identified during the cEDE/EDE interview both at study inception and throughout the follow-up period using criteria from Jones et al. (22). DEB was defined as reporting any of the following during the 28 days before the EDE interview: dieting; objective binge-eating episodes; self-induced vomiting; abuse of laxatives, diuretics, or diet pills; insulin omission or underdosing for weight control; and intense, excessive exercise for weight control (defined as high-intensity exercise for weight and shape control for at least 30 min at least 5 days/week (16,22)). Anorexia nervosa and bulimia nervosa were diagnosed throughout the study using DSM-IV-TR diagnostic criteria (23). The operational diagnostic criteria used for categorization of DEB, ED not otherwise specified (EDNOS), and subthreshold EDs in this study, along with further details of study methodology, are published elsewhere (16,22). Subthreshold EDs were defined as the following: 1) occasional binge-eating and/or purging over the past 3 months (three or more times); 2) self-evaluation unduly influenced by shape or weight, and extreme dietary restraint (<500 kcal/day); and 3) self-evaluation unduly influenced by shape or weight, and intense, excessive exercise for the purpose of weight control five or more times weekly over the past 3 months. Height, weight, and HbA_{1c} were recorded at each assessment point. Participants were classified as underweight, normal weight, overweight, or obese using the International Obesity Task Force values (24) until age 18 years. At age 18 years and older, participants' weight status was classified using the National Institutes of Health clinical guidelines (25). From times 1–5, all HbA_{1c} laboratory assessments were carried out at The Hospital for Sick Children using the Bio-Rad variant method (26). HbA_{1c} values at time 6 and time 7 were collected from The Hospital for Sick Children if the participant continued to receive diabetes care at that center or were requested from their endocrinologist or family doctor.

Statistical Approach

Analyses were carried out using SPSS software version 21. According to plotting recommendations (27), survival

plots are presented upward, and the x-axis of plots extends until the proportion of the remaining sample is unreasonably small (i.e., less than 10% of the initial sample). Plots include SE as a measure of statistical uncertainty.

RESULTS

Descriptive Results

As previously reported (18), 126 girls participated at time 1, 106 at time 2, 88 at time 3, 76 at time 4, and 98 at time 5. Newly reported here, 81 girls (64.3% participation) took part at time 6 and 71 (56.4% participation) at time 7. Girls who participated at time 6 and time 7 were not significantly more likely than nonparticipants to have reported DEB at time 1 (time 6: $\chi^2 = 2.1$, $df = 1$, $P = 0.20$; time 7: $\chi^2 = 1.3$, $df = 1$, $P = 0.32$). Mean age was 11.8 ± 1.5 years at time 1 and 23.7 ± 2.1 years at time 7. Mean BMI was 20.1 ± 3.2 kg/m^2 at time 1 and 25.4 ± 4.3 kg/m^2 at time 7. At time 7, 4.2% (3/71) of participants were classified as underweight, 46.5% (33/71) as normal weight, 38.0% (27/71) as overweight, and 11.3% (8/71) as obese.

At time 6, 19.8% (16/81) suffered from a full-syndrome or subthreshold ED; there were 2 cases of anorexia nervosa, 1 of bulimia nervosa, 2 of EDNOS, and 11 of a subthreshold ED. At time 7, 40.8% (29/71) had a full-syndrome or subthreshold ED, of whom 2 met criteria for anorexia nervosa, 1 for bulimia nervosa, 20 for EDNOS, and 6 for a subthreshold ED.

At time 6, participant age ranged from 17.3 to 23.2 years, and DEB was reported by 33.3% (27/81) of participants. At time 7, age ranged from 19.2 to 27.8 years, and DEB was reported by 59.2% (42/71) of participants. The rates of current (i.e., in the past 28 days) specific DEB were reported during the EDE interview: dieting, 24.7% (20/81) at time 6 and 33.8% (24/71) at time 7; objective binge episodes, 3.7% (3/81) at time 6 and 16.9% (12/71) at time 7; self-induced vomiting, 4.9% (4/81) at time 6 and 8.5% (6/71) at time 7; insulin omission for weight loss, 6.2% (5/81) at time 6 and 26.8% (19/71) at time 7; laxative abuse, 0% at time 6 and 4.2% (3/71) at time 7; diet pill abuse, 1.2% (1/81) at time 6 and 0% at time 7; and excessive exercise for weight control, 8.6% (7/81) at time 6 and 25.4% (18/71) at time 7. Rates of DEB overall, excluding insulin omission, were 30.8% (25/81) at time 6 and 35.2% (25/71) at time 7.

Mean HbA_{1c} was $8.3 \pm 1.1\%$ (67 ± 10 mmol/mol) at time 1 ($n = 126$), $8.9 \pm 1.7\%$ (74 ± 16 mmol/mol) at time 6 ($n = 54$), and $8.5 \pm 1.5\%$ (69 ± 14 mmol/mol) at time 7 ($n = 58$). HbA_{1c} was significantly higher overall at time 6 compared with time 1 ($t = -2.5$, $df = 53$, $P = 0.02$, $d = 0.42$), with a trend for higher HbA_{1c} at time 7 compared with time 1 ($t = -1.9$, $df = 57$, $P = 0.06$, $d = 0.15$). At time 6, HbA_{1c} was significantly higher among women with a current ED than in those without ($10.0 \pm 1.5\%$ [86 ± 14 mmol/mol] vs. $8.7 \pm 1.6\%$ [72 ± 15 mmol/mol]; $t = 2.2$, $df = 52$, $P = 0.04$, $d = 0.83$), with a trend for higher HbA_{1c} among those with an ED at time 7 than those without ($9.0 \pm 1.8\%$ [75 ± 17 mmol/mol] vs. $8.2 \pm 1.3\%$ [66 ± 12 mmol/mol]; $t = 1.9$, $df = 51$, $P = 0.07$, $d = 0.51$).

Risk of First Onset and Point Prevalence

Life tables were used to determine the risk of first onset (i.e., hazard rates) of detected DEB and ED onset by age (Table 1). Estimated rates of full-syndrome ED (anorexia nervosa, bulimia nervosa, and EDNOS) and subthreshold ED by age are presented in combined format. Point prevalence of detected DEB and EDs also are presented in Table 1, integrating all assessments at times 1–7 and

organized by age in years. Point prevalence was calculated using the following formula: (all “cases” of DEB or ED in the pool of participants at a specific age)/(total number of participants assessed at that age).

Onset, Recovery, and Recurrence: Observed Incidence and Cumulative Probability

A flowchart of the observed incidence of onset, remission, and recurrence of detected DEB and EDs is presented in Fig. 1. Remission was defined as the first point at which participants no longer reported DEB or met criteria for an ED subsequent to the first observed onset. Recurrence was defined as the first point at which participants reported DEB or met criteria for an ED subsequent to the first observed remission.

To address the limitations related to right-censoring of data (loss of follow-up as a result of study attrition or termination) in longitudinal research, Kaplan-Meier survival analysis was used to estimate the cumulative probability of first detection of DEB and ED (full-syndrome and subthreshold EDs combined), again organized by age. The cumulative probability of experiencing the first onset of DEB or an ED is plotted in Fig. 2. The average age at DEB and ED onset was 18.3 years

Table 1—Risk of first onset and point prevalence of DEB and full-syndrome or subthreshold ED by age in years

Age (years)	DEB		Full-syndrome or subthreshold ED	
	Risk of onset HR (SE)	Point prevalence*, % (cases/total)	Risk of onset HR (SE)	Point prevalence*, % (cases/total)
9	0.01 (0.01)	5.3 (1/19)	0.00 (0.00)	0.0 (0/19)
10	0.04 (0.02)	13.9 (5/36)	0.01 (0.01)	2.8 (1/36)
11	0.04 (0.02)	11.1 (6/54)	0.02 (0.01)	3.7 (2/54)
12	0.02 (0.01)	11.0 (8/73)	0.01 (0.01)	2.7 (2/73)
13	0.17 (0.04)	30.7 (23/75)	0.06 (0.02)	10.7 (8/75)
14	0.15 (0.04)	22.7 (17/75)	0.01 (0.01)	1.3 (1/75)
15	0.06 (0.03)	29.8 (17/57)	0.02 (0.01)	8.8 (5/57)
16	0.10 (0.04)	31.9 (15/47)	0.01 (0.01)	4.3 (2/47)
17	0.09 (0.04)	36.0 (9/25)	0.03 (0.02)	12.0 (3/25)
18	0.13 (0.05)	19.4 (7/36)	0.02 (0.02)	8.3 (3/36)
19	0.13 (0.06)	32.1 (9/28)	0.08 (0.03)	32.1 (9/28)
20	0.13 (0.07)	54.5 (12/22)	0.10 (0.04)	36.3 (8/22)
21	0.08 (0.06)	41.2 (7/17)	0.16 (0.04)	29.4 (5/17)
22	0.29 (0.13)	56.7 (17/30)	0.08 (0.07)	30.0 (9/30)
23	0.10 (0.10)	46.7 (7/15)	0.11 (0.06)	40.0 (6/15)
24	0.13 (0.10)	60.0 (6/10)	0.09 (0.08)	40.0 (4/10)
25–27	0.13 (0.09)	50.0 (11/22)	0.09 (0.05)	27.3 (6/22)

HR, hazard rate. *Point prevalence is calculated as follows: ([all “cases” of DEB or ED among participants of a particular age]/[total number of participants assessed at that age]).

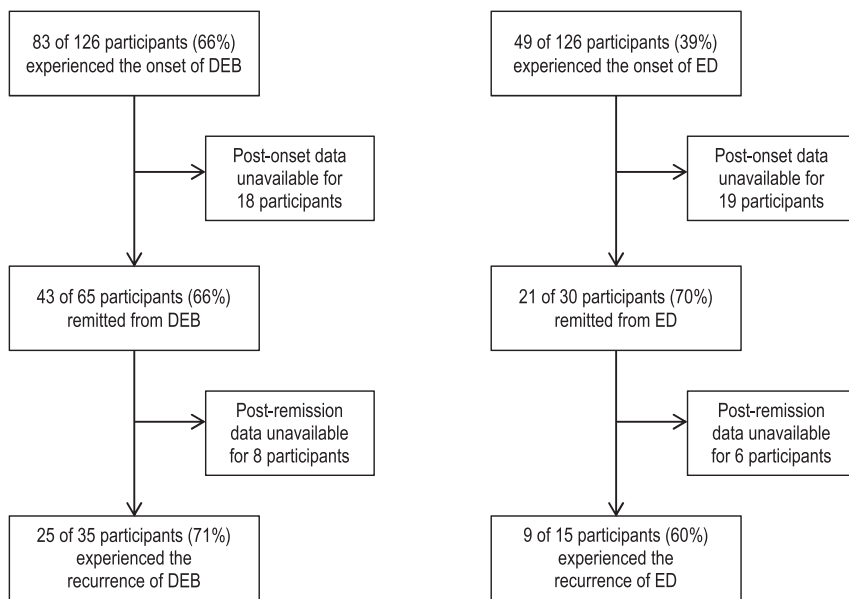


Figure 1—Flowchart of observations of participants who experienced the onset, remission, and recurrence of DEB (left panel) and full-syndrome ED or subthreshold ED (right panel). Postonset or postremission data were unavailable for participants who either dropped out of the study or experienced onset and/or remission at the final wave of data collection.

(95% CI 17.3–19.3) and 22.6 years (95% CI 21.6–23.5), respectively. Estimates of the cumulative probability of DEB onset remained reliable up to age 23 years; 79% experienced onset of DEB by this point. Estimates of the cumulative probability of ED onset remained reliable up to age 25 years; 60% experienced onset of an ED by this point.

Survival analysis then was used to estimate the cumulative probability of detected remission (“detected” because the exact time of remission occurred at some time before the assessment point) from DEB and an ED, organized by duration of time since first detection. The cumulative probability of detected remission from DEB or ED is plotted in

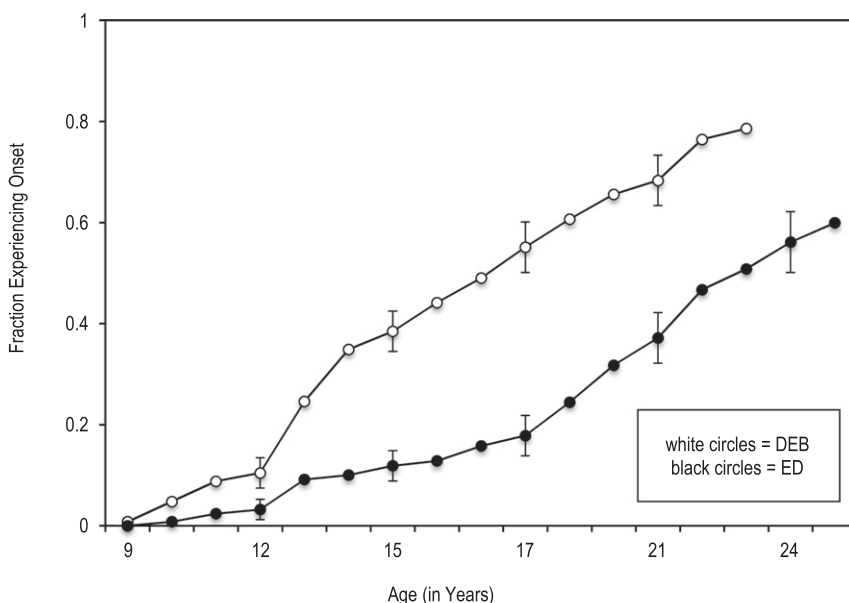


Figure 2—Cumulative probability of experiencing the onset of DEB and full-syndrome ED or subthreshold ED. The x-axis of plots extends until the proportion of the remaining sample is unreasonably small (i.e., <10% of the initial sample).

Fig. 3. The average time period between onset and subsequent detected remission of DEB and ED was 6.0 years (95% CI 4.7–7.2) and 4.3 years (95% CI 3.1–5.5), respectively. Estimates of the cumulative probability of DEB remission remained reliable until 9 years after DEB onset; 57% experienced remission by this point. Estimates of the cumulative probability of ED remission remained reliable until 6 years after detected ED onset; 79% experienced remission by this point.

Survival analysis also was used to estimate the cumulative probability of recurrent detected onset of DEB and an ED, organized by duration of time since first detected remission. The average time period between detected remission and subsequent recurrent onset of DEB and an ED was 4.7 years (95% CI 3.5–5.8) and 6.5 years (95% CI 4.4–8.6), respectively. Estimates of the cumulative probability of DEB recurrence remained stable until 6 years after DEB remission; 79% experienced recurrence by this point. Estimates of the cumulative probability of ED recurrence also remained stable until 6 years after ED remission; 53% experienced recurrence by this time.

CONCLUSIONS

The most striking finding in this prospective study with a 14-year follow-up is the high incidence of DEB and EDs in a group that is particularly vulnerable to their serious adverse health consequences. Accounting for loss to follow-up and study termination, the probability of developing DEB or an ED over the course of the study was estimated to be 79% and 60%, respectively.

These rates provide evidence that disordered eating is a common and serious concern among girls and young women with type 1 diabetes. Although adolescent and adult women in the general population also frequently report dieting, rates of more extreme weight loss behaviors and clinical eating disorders tend to be lower than those that occurred in this study (22,28–30). Comparisons to the general population must be cautious because there is no comparison group included in this study and because insulin omission, a behavior unique to those with type 1 diabetes, is not a purging behavior available to those without diabetes.

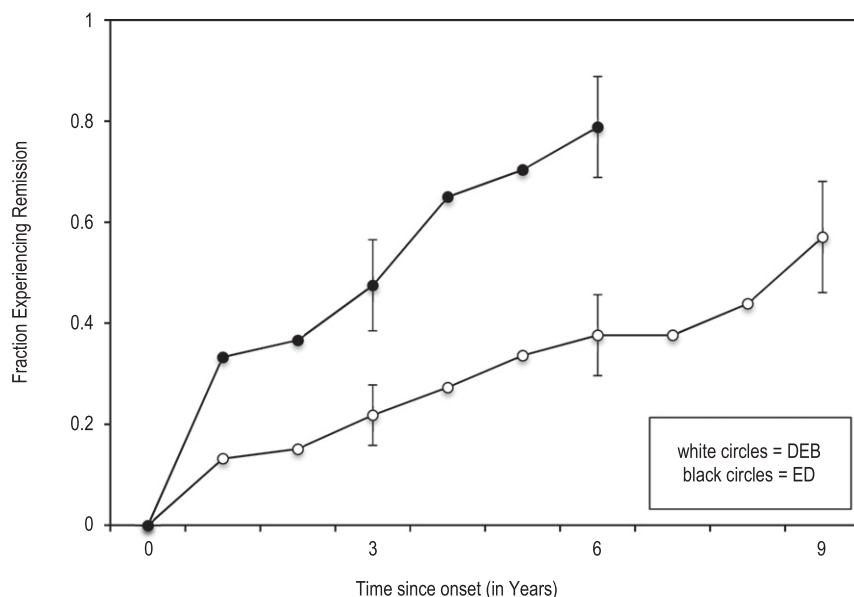


Figure 3—Cumulative probability of experiencing remission from DEB and full-syndrome ED or subthreshold ED. The x-axis of the plots extends until the proportion of the remaining sample is unreasonably small (i.e., <10% of the initial sample).

The point prevalence for DEB and ED continued to increase across the study, largely because of marked increases in reported insulin omission for weight loss. Of particular concern, insulin omission as a weight control method was reported by 27% of participants at time 7. This dangerous method of purging directly compromises metabolic control and confers both short-term and long-term medical risk. Other researchers found it to be highly persistent among adult women with type 1 diabetes and associated with increased morbidity and mortality (10,15).

Results regarding the longitudinal course of eating disturbances among the general population vary depending on sample, age range, and methodology. Bulimic symptoms most commonly emerge during mid-adolescence (31), with peak bulimia nervosa onset in late adolescence (30,32). Rates of dieting and other DEB generally increase from adolescence into early adulthood (28,29), but decrease by mid-adulthood (33). Of concern, in this study the incidence of DEB and EDs remained high, even during follow-up well into young adulthood, and the expected decrease in the incidence and frequency of eating disturbances has not yet emerged in this cohort. According to the hazard rates calculated in this study, the risk of onset of DEB and EDs remains substantial into

adulthood, even among individuals who have successfully avoided DEB and EDs throughout their adolescence.

In this study, both DEB and EDs tended to be persistent, with a mean time from observed onset to detected remission of 6.0 and 4.3 years, respectively, and significant estimated risk of recurrence among those whose eating disturbances initially remitted. The persistent nature of DEB was apparent even at early stages in this longitudinal study: 92% of girls who reported DEB early in the study continued to report it at the 5-year follow-up (18). Consistent with both naturalistic and posttreatment studies of the general population showing persistence but fluctuating severity of eating problems over time (32,34–38), substantial rates of remission and recurrent onset were observed across the study period. The course of EDs among the general population, as well as the high prevalence and persistence thus far in this cohort, suggest that clinicians should anticipate that eating disturbances in this population may often be persistent and recurring, rather than mild and transient.

The high prevalence of DEB and EDs among women with type 1 diabetes, in addition to high incidence of new ED cases continuing into the young adult years, suggests that sustained efforts at prevention, detection, and treatment of eating disturbances are needed

across the adolescent and young adult years among women with type 1 diabetes. This may not regularly occur because young adults with chronic medical and mental health conditions are particularly vulnerable to disrupted medical care during the years of transition from pediatric to adult care (39,40). This study suggests that mental health and eating disturbances in particular should be an important clinical focus of effective transition and continuity of care for young women with type 1 diabetes. The strong association of DEB/EDs with long-term medical complications among those with type 1 diabetes justifies giving significant attention to screening and treatment efforts. Findings regarding longitudinal risk and protective factors for DEB/EDs in this population should be incorporated into the design of clinical interventions.

Study limitations include the diminished sample size over time as a result of both loss to follow-up and study termination. Thus the survival curves provide only an estimate of the percentage of participants experiencing DEB/ED onset, remission, and recurrence. Significant information regarding the natural history of these disturbances remains unknown because in this study the mean age at ED onset was 23 years, and a significant number of participants had no or short follow-up after this initial onset. Given the uneven spacing of assessment points, the exact time of these events is unknown and can only be approximated from the assessment times. Nevertheless, the study also had a number of strengths, including the prospective design, lengthy follow-up period, reasonable participation rates, and the use of a well-validated diagnostic interview to assess ED symptoms. Future research should focus on the development and testing of strategies for the prevention and treatment of DEB and EDs in this high-risk group.

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