Eating Habits, Body Weight, and Insulin Misuse

A longitudinal study of teenagers and young adults with type 1 diabetes

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OBJECTIVE — To examine disordered eating, insulin misuse, weight change, and their relationships with glycemic control and diabetic complications in adolescents with type 1 diabetes followed up over eight years.

RESEARCH DESIGN AND METHODS — Of 76 adolescents (43 male, 33 female) with type 1 diabetes aged 11–18 years at the first assessment, 65 were interviewed as young adults (aged 20–28 years). Eating habits were assessed using a standardized Eating Disorder Examination. Height and weight were determined and BMI calculated. Three consecutive urine specimens were collected for measurement of albumin/creatinine ratio and other significant diabetic complications were recorded. Glycemic control was assessed by glycated hemoglobin.

RESULTS — Weight and BMI increased from adolescence to young adulthood. Females were overweight as adolescents and both sexes were overweight as young adults. Concern over weight and shape increased significantly for both sexes from adolescence to young adulthood. This increase in concern was reflected in increased levels of dietary restraint. Features of disordered eating were apparent in females at both assessments, but no patients met the criteria for anorexia nervosa or bulimia nervosa at either assessment. A total of 10 (30%) females, but none of the males admitted underusing insulin to control weight. Five (45%) females with microvascular complications had intentionally misused insulin to prevent weight gain.

CONCLUSIONS — An increase in BMI from adolescence to adulthood was associated with higher levels of concern over shape and weight and more intense dietary restraint, especially among females. Overt eating disorders were no more prevalent in these patients than in the general population, but milder forms of disordered eating were common and had implications for diabetes management. Insulin omission for weight control was frequent among females and may contribute to poor glycemic control and to risk of complications.

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Several previous studies have reported an increased incidence of disordered eating in young adult patients with type 1 diabetes (1–5). This in turn has been related to poor glycemic control, noncompliance with treatment, and the possible increased risk of developing diabetic complications (1,3,6–8). However, in a cross-sectional study of a representative sample of adolescents aged 11–18 years (9) we reported that there was no significant excess in eating disorders when compared with carefully matched nondiabetic controls. We also found that adolescent females with type 1 diabetes were significantly heavier than nondiabetic controls. They were more concerned about their weight than nondiabetic subjects and took more excessive dietary measures to control their shape and weight. Intentional undertreatment with insulin among adolescent females was common (9).

The longer-term outcome of adolescent diabetes in terms of weight, eating disorder, and glycemic control is uncertain. To explore this subject further, we followed these adolescent patients and reassessed them at ~23 years of age (8 years after the original study). Our aims were *1*) to record the subjects' change in body weight, *2*) to assess if concerns over shape and weight were still significant, *3*) to examine if disordered eating and misuse of insulin were more apparent in the subjects now that they are young adults, and *4*) to explore the relationship between disordered eating/insulin misuse and glycemic control and the presence of diabetic complications.

RESEARCH DESIGN AND METHODS

Subjects

The index group consisted of all identifiable surviving members of a cohort of 76 adolescents first assessed in 1989–1990 for the prevalence of eating disorders (9). All the adolescent subjects, at that time, lived within the catchment area of the John Radcliffe Hospital, Oxford, U.K., and were eligible for inclusion if they were between the ages of 11 and 18 years and had been diagnosed as having diabetes at least one year before the start of the study.

The follow-up assessment took place in 1997–1998 and consisted of 65 of the original subjects (86%). Among those not re-interviewed, two subjects had died; one death was unrelated to diabetes, the other followed diabetic ketoacidosis. Another subject was too mentally handicapped to

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Abbreviations: EDE, eating disorder examination; EDNOS, eating disorder not otherwise specified; SDS, standard deviation scores.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

participate after a severe hypoglycemic episode and one subject was omitted because of concurrent illness. A further three subjects refused to participate; three subjects could not be traced; and the general practitioner refused permission for us to contact one subject.

The majority of subjects who took part in the follow-up were still attending diabetic clinics in Oxford. Subjects who had moved outside the district were traced through families, general practitioners, or the district health authority health service computerized records. Some clinic information was available on the subjects whom we were unable to interview.

Procedures

Subjects still attending Oxford clinics were invited to participate in the study by the research nurse (K.S.B.) at a routine clinic visit. The study was explained to them and they were given at least 24 h to consider their response before signed informed consent was sought. The research assessment was conducted by the research nurse in the homes of participants. Nonattenders and subjects who lived outside the area were contacted by letter and telephone. The study was approved by the Central Oxford Research Ethics Committee.

Measures

Height and weight of the subjects were recorded either from data obtained from the clinic visit nearest to the interview date or from measurements made in the subject's home using electronic weighing scales (calibrated to the weighing scales used in clinic) and a Leicester stadiometer. Height, weight, and BMI, defined as weight (kilograms) divided by height (meters) squared, were then transformed to standard deviation scores (SDS) using the current U.K. national standards (10,11) by the formula [(observed measurement − population mean)/population SD] appropriate for sex and age of the subject.

The baseline and follow-up assessment of features of eating disorders were made using the Eating Disorder Examination (EDE) (12). This is a standardized investigator-based research interview of established reliability and validity (13–15), which assesses the core features of eating disorders, enabling operationally defined diagnoses of eating disorders to be made. Subscales cover concerns about shape and weight and dietary restraint, and the frequency of key behaviors such as bulimic episodes and self-induced vomiting is appraised. Each subscale has a possible range of 0–6 for each subject. Subscale scores are expressed as a mean at each assessment. The interview was adapted to allow distinction to be made between behavior necessary for the treatment of diabetes, such as the avoidance of sugary foods for the purpose of glycemic control, and that attributable to an eating disorder, such as extreme dietary restraint for the purpose of shape and weight control. At each assessment the subjects were asked if they ever reduced or omitted their insulin in order to lose weight. At the follow-up assessment, both past and present misuse was ascertained and the likely duration of misuse. The research nurse was trained in accordance with the Oxford Eating Disorder Research Group under the supervision of Christopher Fairburn. All interviews were taped and random scores were regularly subjected to independent review by an experienced trained interviewer.

Glycemic control was assessed by the measurement of HbA_{1c}. For those subjects attending Oxford clinics, the HbA_{1c} measured nearest to the time of the assessment interview was used as an indicator of glycemic control. For all other subjects, a blood sample was taken at the time of interview and brought back to the Oxford laboratory. Only samples taken to this laboratory were included in the analyses to ensure reliability. Medical notes were studied to obtain data concerning serial measurements of HbA_{1c} between baseline and follow-up (minimum 4 years for each subject, average of 20–25 results/subject).

Assessment of renal function was made by collection of three consecutive early morning or overnight urine specimens for urinary albumin/creatinine ratio (16). Evidence of other serious diabetic microvascular complications was obtained from the clinical case notes. These complications were defined as laser-treated proliferative retinopathy; evidence of incipient nephropathy-microalbuminuria or proteinuria (16), and hypertension where this was reported in subjects' medical records and they were receiving antihypertensive therapy.

HbA_1 was measured from 1987–1991 by a Glytrac electroendosmosis method (Ciba Corning Diagnostics, Halstead, U.K.). From 1991 onwards, HbA_{1c} has been measured by a high-performance liquid chromatography method (17) with a between-batch coefficient of variation of 3.5% at a level of 5.6 and 2.2% at a level of 10.1% and a normal range of 4.3–6.1% using a Bio-Rad Diamat automated glycosylated hemoglobin analyser (Bio-Rad Laboratories, Hemel Hempstead, Hertfordshire, U.K.). The changeover between the two methods was carefully documented and appropriate regression equations were available for conversion of the earlier data to the current method and have been documented in detail elsewhere (16).

Albumin was measured by a double-antibody ELISA method (16). The interassay coefficient of variation was 12% at 1.5 mg/l and 10% at 16 mg/l. The interassay coefficient of variation was 12.1% at 1.9 mg/l, 3.2% at 7.3 mg/l, and 3.6% at 20.8 mg/l, giving a mean of 6.3%. Creatinine was measured using the modified Jaffe method (Unimate 7, on a Cobas Mira [Roche Diagnostic Systems, Basel]) automated spectrophotometer. The coefficient of variation was 2% at 2.2 mmol/l.

Statistical methods

SPSS 6.0 for Windows (SPSS, Chicago) was used for statistical analysis. BMI SD scores were compared with normal population data by one-sample *t* tests. EDE subscale scores from both assessments were compared using the Wilcoxon matched pairs signed ranks test. Correlation of change in BMI SDS and the EDE subscale scores was assessed using Spearman's rank correlation. Normally distributed data were analyzed using *t* tests and nonparametric data using Mann-Whitney U tests. Categorical data were analyzed using χ^2 tests or Fisher's exact test. Data are presented as means ± SD.

RESULTS — Demographic details of the study groups at both assessments are shown in Table 1.

There was no difference between the ages and duration of diabetes of those subjects who participated in the study and those subjects not interviewed. At the baseline assessment, 53% of subjects were on twice-daily insulin injections, 3% on a three times daily, and 45% on a four times daily insulin regimen. At the follow-up assessment, 20% of subjects were on twice-daily injections, 2% on three times daily, and 78% on four insulin injections a day.

Weight

The height, weight, and BMI of the subjects at each assessment are summarized in Table 1. Males did not have significantly greater BMI SDS compared with the nor-

Table 1—*Characteristics of the study group*

	Males		Females	
	Baseline	Follow-up	Baseline	Follow-up
n	43	39	33	26
Age (years)	15.2 ± 2.2	23.7 ± 2.1	15.3 ± 1.9	23.9 ± 2.0
Duration of diabetes (years)	8.0 ± 3.7	16.3 ± 3.5	7.1 ± 2.9	15.7 ± 2.9
Height (m)	1.66 ± 0.1	1.79 ± 0.1	1.61 ± 0.1	1.65 ± 0.1
Height SDS	0.05 ± 1.0	0.21 ± 0.8	0.04 ± 1.50	0.15 ± 1.3
Weight (kg)	56.5 ± 12.8	76.8 ± 8.9	58.9 ± 12.6	67.3 ± 11.6
Weight SDS	0.16 ± 0.9	0.55 ± 0.9	0.63 ± 1.1	0.90 ± 1.0
BMI (kg/m^2)	20.1 ± 2.4	24.0 ± 2.5	22.7 ± 3.7	24.7 ± 2.6
BMI SDS	0.21 ± 0.85	0.29 ± 0.86	0.77 ± 0.75	0.79 ± 0.75
BMI SDS versus normal data	NS	$P < 0.05$	$P < 0.001$	$P < 0.001$
HbA_{1c}	9.4 ± 2.5	9.6 ± 1.8	9.7 ± 1.8	9.3 ± 1.9

Data are means ± SD.

mal population at baseline but did by follow-up, whereas females had significantly greater BMI SDS compared with the normal population data at both assessments.

Concern about weight and shape

The mean subscale scores for both sexes at each of the assessments for dietary restraint and shape and weight concern are shown in Table 2. Women were significantly more concerned about weight and shape at follow-up than they had been as teenagers. This was especially so for dietary restraint. Of women who expressed greater concern about weight at follow-up, 72% had a higher BMI SDS than at baseline. Men showed a similar pattern of increased concern at follow-up, although at much lower levels than females at both assessments.

Change in weight in individual men, but not women, was correlated with change in level of dietary restraint (r_s = 0.42, P = 0.008).

Eating disorders

Table 3 shows the prevalence and severity of the features of eating disorders for the diabetic subjects from both the baseline assessment as adolescents and the follow-up assessment as young adults. None of the subjects (male or female) at either assessment met the *Diagnostic and Statistical Manual of Mental Disorders (3rd ed., revised)* (DSM-IIIR) criteria for a diagnosis of anorexia nervosa or bulimia nervosa. Six subjects (one male and five females) from the baseline (n = 3) or the follow-up interview (n = 3) met the criteria for eating disorder not otherwise specified (EDNOS) with either markedly abnormal behavior (n = 3), such as recurrent self-induced vomiting or laxative use, or abnormal importance placed on shape and weight (n = 3). One female was classified with EDNOS on both assessments; she was diagnosed with bulimia nervosa between completing baseline and follow-up interviews and has been receiving treatment for several years. None of the other five subjects had been formally diagnosed as having an eating disorder and none had received treatment.

Misuse of insulin

At the adolescent assessment, five females gave a history of deliberate underuse or omission of insulin to control their weight. At the follow-up assessment, five different women admitted that they had also been omitting/underusing insulin around the time of the baseline interview. Thus, it appeared that at least 10 females (30%) had intentionally reduced or omitted their insulin dosage to control their weight during adolescence or young adulthood. The duration of misuse varied greatly, the minimum length of time being 3 months with the average duration being 2 years. One girl omitted her insulin for up to 2 weeks when she was aged 15–16 years; another admitted to only giving herself a maximum of two injections per week for a period of 7 years. Four females who had misused their insulin were among the six subjects classified as having a clinical eating disorder (EDNOS). No women admitted to current misuse at the follow-up assessment. No males admitted to deliberate insulin misuse at either assessment.

Glycemic control

HbA_{1c} for both sexes at each of the assessments is summarized in Table 1.

There was no relationship between those subjects with EDNOS and glycemic control, nor was there a difference between the glycemic control of those EDNOS subjects with abnormal behavior (vomiting, laxative use) and those with abnormal psychiatric attitudes. Mean glycated hemoglobin of the 10 females who admitted intentional insulin misuse was worse than that of the rest of the females at both baseline, 10.3 ± 1.1 vs. 9.5 ± 2.0, and follow-up, 9.7 ± 1.8 vs. 9.2 ± 1.9, although neither of the differences were statistically significant.

Diabetic complications

Table 4 shows the prevalence of diabetic complications. Of females who had developed microvascular complications, five (46%) had deliberately misused their insulin (one was diagnosed with EDNOS at baseline, another was diagnosed with EDNOS at follow-up). Two females had laser-treated proliferative retinopathy, two had nephropathy, and one had both laser-treated proliferative retinopathy and nephropathy. There were no significant relationships between either disordered eating at baseline or insulin misuse and the development of diabetic complications.

An overall mean HbA_{1c} for the 8-year period between baseline and follow-up demonstrates significantly worse long-term glycemic control for those subjects

Table 2—*EDE subscales*

	Males	Females
n	39	26
Restraint subscale		
Baseline	0.2 ± 0.5	0.7 ± 0.8
Follow-up	0.4 ± 1.0	1.6 ± 1.3
P	NS	<0.01
Shape concern subscale		
Baseline	0.3 ± 0.4	0.9 ± 0.8
Follow-up	0.5 ± 0.4	1.3 ± 0.9
P	<0.05	<0.02
Weight concern subscale		
Baseline	0.3 ± 0.3	1.0 ± 0.9
Follow-up	0.5 ± 0.3	1.8 ± 1.2
P	<0.01	<0.01

Data are means ± SD for the subjects who completed both assessments.

Table 3—*Features of clinical eating disorders*

EDE	Males		Females	
	Baseline	Follow-up	Baseline	Follow-up
Objective Bulimic Episodes				
Current	0	0	0	0
Past	—	0	—	1
Objective Overeating				
Any currently	1	4	1	2
At least 4/month	1	0	1	0
Vomiting				
Current	0	0	1	2
Past	—	0	—	1
Laxative Misuse				
Current	0	0	0	0
Past	—	0	—	2
Insulin Omission or Misuse				
Current	0	0	4	0
Past	0	0	1	5*
EDNOS				
Current	0	1	3	3†

*Different subjects from those admitting to misuse at baseline; †one of these subjects is the same as at baseline.

with complications. Duration of diabetes did not predict risk of complications.

CONCLUSIONS — This study provides the first comprehensive view of long-term outcome of childhood and adolescent diabetes by describing eight-year outcomes for a representative sample. It has the additional advantages that the subjects were identified as all those known to specialist diabetic services within a defined geographical area and that a high proportion of subjects (86%) completed both the initial and follow-up interviews. The data collected represented a thorough assessment of eating habits of adolescents and young adults, together with longitudinal glycemic control and body mass.

We found males were only marginally overweight at baseline but significantly so at follow-up, while females were significantly overweight at both times. The increases in BMI from adolescence to young adulthood were accompanied by marked increases in body shape concern, weight concern, and dietary restraint scores in the eating disorder interview, particularly in females.

The mechanism responsible for weight gain during adolescence and early adulthood in diabetes is unclear. Our recent study suggested that this increase could be associated with relative leptin resistance—with females being more resistant and gaining greater fat mass than males (18), consistent with the greater BMI in females in this study. Insulin resistance during puberty is restricted to peripheral glucose metabolism (19) and therefore another possibility is that weight gain may relate to hyperinsulinemia due to excess peripheral rather than portal levels of insulin. Hyperinsulinemia may be particularly marked during multiple injection therapy and some researchers have implicated the inflexibility of this regimen in the development of obesity (20). Of our subjects, 48% were on multiple injections in 1989–1990, whereas the number had increased to 80% in 1997–1998.

Certainly patients perceive an association between insulin dose and obesity risk. The diabetic clinic strives to increase insulin dose to cope with insulin resistance at puberty, but there is a risk that higher doses persist when insulin sensitivity is improving at the end of puberty. It seems that at least 30% of females are omitting insulin to suppress weight gain. This finding is consistent with our earlier study in young adults (6) and with other reports (21,22). Generally, adolescents may have a lower adherence to insulin treatment than subjects aged >20 years (23). None of the subjects confessed to current omission at follow-up, although underreporting is likely and five admitted to past omission. As might be expected, these females all had higher mean glycated hemoglobin than those not omitting insulin.

We found no cases of anorexia nervosa or bulimia nervosa at either assessment, although we did find cases of EDNOS. This is a similar finding to our previous cross-sectional reports of adolescents and young adults, in which age- and social class–matched controls were included (6,9). This finding contrasts with some other studies that have found bulimia nervosa to be more prevalent in females with type 1 diabetes (2,3,5). However, these studies used rating scales such as the Eating Attitudes Test or the Eating Disorders Inventory. These self-reporting measures tend to provide higher estimates of prevalence of eating pathology (24); formal eating disorder diagnosis cannot be made because the scales do not elicit the necessary specific criteria. Interview-based measures such as the

Table 4—*Microvascular complications and the relationship with glycemic control and insulin misuse*

	Subjects with complications		Subjects without complications		
	Males	Females	Males	Females	*P*
n	10	11	30	18	—
Complications					
Severe retinopathy*	1	2	—	—	—
Nephropathy†	6	5	—	—	—
Hypertension‡	2	1	—	—	—
Multiple§	1	3	—	—	—
HbA_{1c} (%)	11.3 ± 1.4		9.4 ± 1.7		<0.001
Duration of diabetes (years)	16.3 ± 2.6		15.9 ± 3.6		NS
Number with insulin misuse	0 (0)	5 (46)	0 (0)	5 (28)	NS

Data are *n*, means ± SD, or *n* (%). Complications given as measured over the 8-year period between baseline and follow-up. *Laser-treated proliferative retinopathy; †microalbuminuria or proteinuria; ‡antihypertensive therapy (with no nephropathy); §severe retinopathy and nephropathy. HbA_{1c} is given as measured over the 8-year period between baseline assessment and follow-up.

EDE with its criterion-based diagnoses, as used in this present study, have been recognized as the preferred instrument for diagnosing eating disorders (24).

Overall, the association between glycemic control and features of eating disorders was unclear. Disordered eating behavior alone in adolescence was not related to increased risk of complications, but deliberate insulin misuse appears to be more important, although not statistically significant in our relatively small sample size. At least two other studies (1,7) have reported significant relationships between disordered eating and glycemic control when discussing the increased risk of complications. However, one of these studies (1) recorded much higher HbA_{1c} values in their eating-disordered group than the subjects in our study, and both included insulin omission as a feature of an eating disorder. If we also included those subjects who misused insulin in our eating disorder group, 39% of females would have met this criterion, a similiar figure to one of the previous studies (7), but the proportion who developed significant complications would remain at 46%.

In conclusion, this longitudinal study did not demonstrate evidence of increase in the prevalence of bulimia nervosa, but it did reveal continuing and increasing concerns about weight and shape that correspond with the actual increase in weight gain, especially in females. It is possible that these data reflect a general increase in concern about shape and weight gain with age in the normal population, but there are no data to support this possibility. Nevertheless, the increased concerns in the subjects with diabetes are likely to reflect their greater weight gain and were associated with a worryingly high occurrence of insulin omission to control weight. This practice could be contributing to worse glycemic control and increased risk of complications, particularly in females during adolescence and young adult life. Weight gain may in part relate to intensification of therapy; thus, as well as being aware of the need to increase doses to cope with the insulin resistance of puberty, we need to remember the patient's concerns that this may lead to weight gain. The use of intensified insulin regimens can reduce HbA_{1c} but they need to be refined to reduce the risk of hyperinsulinemia and weight gain.

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