Blood Glucose Awareness Training (BGAT-2)

Long-term benefits

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OBJECTIVE — Blood glucose awareness training (BGAT) has been shown to improve awareness of blood glucose (BG) fluctuations among adults with type 1 diabetes. This study investigates the long-term (12-month) benefits of BGAT-2.

RESEARCH DESIGN AND METHODS — A total of 73 adults with type 1 diabetes participated in a 6-month repeated baseline design with a 12-month follow-up. At 6 months and 1 month before BGAT-2 and at 1, 6, and 12 months after BGAT-2, subjects used a handheld computer for 50 trials and completed psychological tests. Throughout assessment, subjects completed diaries, recording occurrences of diabetic ketoacidosis, severe hypoglycemia, and motor vehicle violations. During follow-up, 50% of the subjects received booster training.

RESULTS — During the first and last halves of both the baseline period and the follow-up period, dependent variables were generally stable. However, from baseline to follow-up, BGAT-2 led to *1*) improved detection of hypoglycemia and hyperglycemia; *2*) improved judgment regarding when to lower high BG, raise low BG, and not drive while hypoglycemic; *3*) reduction in occurrence of diabetic ketoacidosis, severe hypoglycemia, and motor vehicle violations; and *4*) improvement in terms of worry about hypoglycemia, quality of life, and diabetes knowledge. Reduction in severe hypoglycemia was not associated with a worsening of metabolic control (HbA$_1$). The presence or absence of booster training did not differentially affect these benefits.

CONCLUSION — BGAT has sustained broad-ranging benefits, independent of booster intervention.

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Self-monitoring of blood glucose (SMBG) by the patient offers specific information about BG levels. Due to financial or other constraints, patients are often unable to perform SMBG frequently to use actual BG levels to make moment-to-moment treatment decisions. Consequently, individuals with type 1 diabetes often rely on symptoms and estimates of their BG levels (1) to decide whether to eat, exercise vigorously, perform SMBG, or even drive and/or perform other potentially dangerous activities.

Blood glucose awareness training (BGAT) is a psychoeducational programmatic intervention designed to improve the accuracy of patients' detection and interpretation of relevant BG symptoms and other cues. BGAT has been shown to improve accuracy of overall recognition of current BG level as well as to specifically improve detection of hypoglycemia and hyperglycemia (2,3). Its efficacy has been demonstrated to be greatest among patients who are the least accurate in detecting current BG levels (e.g., individuals with reduced hypoglycemic awareness) (4). When patients receiving BGAT training were compared with patients receiving dietary nutritional education, the BGAT group was found to have better-preserved hormonal counterregulation while undergoing successful intensive insulin therapy (5).

To date, there has been only one long-term follow-up study of BGAT (5). We followed 28 BGAT-1 subjects and 12 control subjects for 4.9 years. Of the BGAT subjects, 50% received booster training consisting of completion of BGAT diaries for 2 weeks before assessment. At follow-up, BGAT subjects who received booster training were significantly more accurate at estimating their BG levels. There was a trend ($P < 0.07$) in BGAT subjects who received no booster training to be more accurate in estimating BG levels as compared with control subjects. BGAT subjects were also involved in fewer motor vehicle accidents than control subjects; 42% of the control subjects versus only 15% of the BGAT subjects had collisions.

The current study presents prospective 12-month follow-up data of BGAT-2 (4) in terms of metabolic, behavioral, and psychological parameters. BGAT-2 differs from BGAT-1 in several aspects. BGAT-2 added separate chapters on how insulin, food, and exercise influence BG to anticipate changes in BG. Additionally, the concluding chapter of BGAT-2 specifically focuses on relapse prevention. The short-term effects of BGAT-2 on patients with either normal or reduced hypoglycemia awareness have been previously reported (4). These two groups were drawn from three diverse clinical settings (University of Virginia, Vanderbilt University, and the Joslin Diabetes Center), and four instructors (D.J.C., L.G.F., W.P., and D.S.) delivered the training. Detection of high BG was improved for both groups of subjects; detection of low BG improved
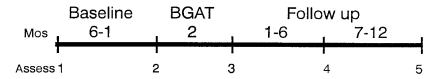
for only the subjects with reduced hypoglycemic awareness. During follow-up, 50% of the BGAT-2 subjects received booster training.

The following hypotheses were tested: *1*) BGAT would lead to sustained, improved awareness of BG fluctuations as reflected in detection of both hypoglycemia and hyperglycemia and overall BG estimation accuracy; *2*) improved awareness would lead to better judgments concerning extreme BG levels in terms of better judgment regarding when to treat high and low BG levels and when not to drive a motor vehicle because of low BG; *3*) better awareness and judgment would lead to fewer extreme BG levels and fewer negative sequelae of extreme BG, such as diabetic ketoacidosis (DKA), severe hypoglycemia, and motor vehicle violations; *4*) less uncertainty and fewer negative events would lead to better psychological functioning in terms of less worry about hypoglycemia, improved quality of life, less depression, and reduced marital conflict over diabetes; and *5*) these effects would be greater for subjects receiving booster training.

RESEARCH DESIGN AND METHODS

All sites recruited subjects through newsletters, diabetes clinic notices, and physician referrals. All subjects had to *1*) have diabetes for ≥ 2 years, *2*) have taken insulin since the time of diagnosis, *3*) routinely measure BG ≥ 2/day, and *4*) no history of severe depression (psychiatric hospitalization or a Beck Depression Inventory [BDI] score >24) or substance abuse (CAGE score >1). One applicant was excluded because of depression. A total of 36, 29, and 35 subjects were recruited and trained at the University of Virginia, the Joslin Diabetes Center, and Vanderbilt University, respectively. Of the 78 subjects who completed BGAT-2, 76 completed 6 months of follow-up assessments and 73 completed 12 months of follow-up assessments. At 12-month follow-up, there were 25 male subjects and 48 female subjects: mean age $= 38.3 \pm 9.1$ years, duration of disease $= 19.5 \pm 10.5$ years, insulin U/day $= 38.9 \pm 16.5$, and $HbA_1 = 10.2 \pm 2.1\%$. In our laboratory, the upper limit of normal HbA_1 is 6.9%. An HbA_1 of 9% is equivalent to the mean HbA_{1c} of the Diabetes Control and Complications Trial intensive treatment group.

Figure 1—*Repeated baseline design.*

Dependent variables

Subjects were given handheld computers that prompted them to estimate their BG level; they then recorded whether they would raise or lower their BG level and whether they would or would not drive. The subjects then measured and recorded their actual BG levels. Subjects repeated this process 50 times over a 3-week period, just before the routine SMBG and whenever they believed their BG levels were high or low. The computer recorded the time and date of each entry and the elapsed time between the prompt "Measure your BG" and when SMBG reading was entered. Any entries made in <45 s after the prompt were considered unreliable. Unreliable readings and entries that occurred within 1 hour of the previous entry were not considered for data analysis. From this data set, we calculated the following: *1*) percentage of low (<3.8 mmol/l) and high (>10 mmol/l) BG levels detected; *2*) the overall percentage of accurate BG estimates ("A" zones of the error grid analysis [EGA]); *3*) the percentage of time when BG level was low and the subject judged not to drive a motor vehicle and judged to treat the low BG level; and *4*) the BG risk index that logarithmically weights progressively extreme BG levels, where a BG of 6.25 mmol is weighted 0 and BG levels of 1.1 mmol (20 mg/dl) and 33.3 mmol (600 mg/dl) are weighted 100 (the sum of these weighted values is then divided by the number of BG readings to calculate a BG risk index) (9,10).

Subjects completed a series of psychometric instruments. The Hypoglycemic Fear Survey—Worry (11,12) determined whether BGAT led to a reduction in participants' concerns over the possibility of experiencing hypoglycemia. The Quality of Life Scale (13) was administered to determine whether greater awareness of BG fluctuations, or less uncertainty, would improve quality of life. The BDI (14) was used to determine whether BGAT improved mood. The Dyadic Adjustment Scale—Diabetes Conflict (15) was used to determine whether greater awareness of BG fluctuations led to reduced marital conflict surrounding diabetes issues. We developed a diabetes knowledge scale, which assessed basic information derived from BGAT. This was administered to determine subjects' mastery of information learned from BGAT.

Subjects completed and mailed in monthly diaries that chronicled occurrence of DKA, severe hypoglycemia (stupor or unconsciousness necessitating assistance in the treatment of low BG) (16), and citations for motor vehicle violations.

Procedure

Because placebo conditions have not been shown to affect accuracy of BG estimation (2,3), and because it was judged to be difficult and inappropriate to keep subjects in a placebo condition for 20 months, we used a repeated baseline design to establish stability of the measures (see Fig. 1). For 6 months before and 12 months after BGAT-2, monthly diaries were completed. At 6 months and 1 month before BGAT-2 and at 1, 6, and 12 months after BGAT-2, subjects completed the psychometric instruments, used handheld computers for 3 weeks, and had their blood drawn for measurement of HbA_1.

Table 1—*Reliability and stability of primary dependent variables across the 6- and 1-month pre-BGAT-2 repeated baseline*

Variable	6- and 1-month pre-BGAT	Correlations	Contrasts
Improved recognition of BG levels*			
% Detection of low BG	36 ± 32; 34 ± 31	$F = 0.64, P < 0.001$	$t = 0.9$, NS
% Detection of high BG	52 ± 25; 49 ± 26	$F = 0.65, P < 0.001$	$t = 1.1$, NS
% Accurate estimates	39 ± 13; 38 ± 13	$F = 0.72, P < 0.001$	$t = 0.1$, NS
Reduced extreme BG fluctuations†			
BG risk index	14.1 ± 5.1; 13.7 ± 4.9	$F = 0.55, P < 0.001$	$t = 0.7$, NS
HbA$_1$	10.2 ± 2.1; 10.2 ± 2.0	$F = 0.85, P < 0.001$	$t = 0.5$, NS
Improved judgement‡			
% Decision to treat when low	49 ± 30; 55 ± 33	$F = 0.34, P < 0.003$	$t = 1.3$, NS
% Decision not to drive when low	52 ± 38; 47 ± 38	$F = 0.50, P < 0.002$	$t = 0.8$, NS
Reduction of negative consequences§			
DKA (total no.)	4; 3	—	—
Severe hypoglycemia	1.4 ± 2.1; 1.8 ± 1.9	$F = 0.77, P < 0.001$	$t = 1.7$, NS
Motor vehicle violations	0.1 ± 0.3; 0.08 ± 0.2	$F = 0.45, P < 0.001$	$t = 0.2$, NS
Change in psychological parameters‖			
Hypoglycemia fear survey–worry	23.7 ± 10.3; 20.2 ± 10.1	$F = 0.76, P < 0.001$	$t = 4.3, P < 0.01$
DQOL–impact	46.7 ± 10.5; 45.8 ± 9.0	$F = 0.57, P < 0.001$	$t = 0.9$, NS
DQOL–worry	19.4 ± 8.6; 17.1 ± 8.1	$F = 0.69, P < 0.001$	$t = 3.1, P < 0.01$
BDI–total	6.1 ± 5.4; 7.7 ± 6.8	$F = 0.67, P < 0.001$	$t = 2.8, P < 0.01$
DAS–diabetes conflict	19.8 ± 11.2; 18.4 ± 8.7	$F = 0.53, P < 0.001$	$t = 1.3$, NS
Knowledge	NA; 43.2 ± 4.2	—	—

Data are means ± SD unless otherwise indicated. *$F = 0.77, P = 0.52$, MANOVA; †no MANOVA performed because only one variable, BG risk index, was hypothesized to change; ‡$F = 2.4, P = 0.1$, MANOVA; §$F = 0.87, P = 0.46$, MANOVA; ‖$F = 5.6, P < 0.005$, MANOVA. DAS, Dyadic Adjustment Scale; DQOL, Daily Quality of Life; NA, not available.

BGAT-2 was delivered to groups of 5–15 subjects in 8 weekly sessions that focused on teaching patients how to identify their best internal cues of extreme levels of BG and how to anticipate extreme levels of BG based on information concerning insulin, food, and exercise (4). At posttreatment, subjects were matched based on their ability to detect low BG levels and then randomized to either booster or no-booster training. Subjects randomized to booster training received the following: *1*) prompts to look for BG cues and anticipate high and low BG levels, along with key concept summary pages from the BGAT-2 manual at months 3 and 9; *2*) a summary report concerning their handheld computer results at months 4 and 10; and *3*) BGAT-2 diaries to complete daily for 1 week at months 5 and 11.

Data analysis

Table 1 presents the correlations and results of Student's *t* tests for handheld computer, psychometric, and diary data for the first and second halves of the baseline period. This stability of measures during 6 months of pretreatment monitoring demonstrated that these variables did not change as a function of time or repeated testing, which allowed us to average the 6 months of baseline data for comparison with follow-up data. To test hypotheses concerning long-term effects of BGAT-2, 1×3 (6- to 1-month pretreatment, 1- to 6-month and 7- to 12-month follow-up) multiple analyses of variance (MANOVAs) were first performed for the separate clusters of dependent variables (BG estimation accuracy, judgment, negative clinical sequelae, and psychological parameters). To assess the impact of BGAT-2 on individual variables, across-subject repeated-measure analyses of variance (ANOVAs) were performed. When significant ($P < 0.01$) time effects were identified, two contrasts were performed. Contrast 1 compared the 6-month baseline with the 6- and 12-month follow-up data to determine whether there was a long-term benefit of BGAT-2. Contrast 2 compared posttreatment (assessment 3, Fig. 1) with 6- and 12-month follow-up data (assessments 4 and 5) to assess the stability of the effect. To assess the effects of booster training, 2 (booster versus no-booster) $\times$ 2 (assessment 3 versus assessment 5) ANOVAs were performed.

RESULTS

Accuracy of BG estimation

The ability of the subjects to estimate BG levels was significantly improved (MANOVA $F = 4.0, P < 0.01$) by BGAT-2. BGAT-2 led to a significant improvement in the detection of low and high BG levels from baseline through 12 months of follow-up (Table 2, Contrast 1, $P < 0.003$), and this was stable across 6- and 12-month follow-up (Contrast 2). Similarly, there was a significant improvement in clinically accurate estimates ("A" zones of EGA) from baseline through 12 months of follow-up ($P < 0.001$), and this was stable across 6- and 12-month follow-up ($P = 0.24$).

BG fluctuations

As shown in Table 2 (BG risk index), BGAT-2 led to a significant reduction in extreme BG levels from baseline through 12 months of follow-up ($P = 0.001$), and was stable across 6- and 12-month follow-up ($P = 0.32$).

Judgment

Determination of when to treat high BG levels, when to treat low BG levels, and whether to drive a motor vehicle with low BG levels was significantly improved by

Table 2—*Comparison of mean scores for primary dependent variables across baseline and follow-up periods*

Variable	Baseline	6-month follow-up	12-month follow-up	Time P levels	Contrast 1* P levels	Contrast 2† P levels
Improved recognition of BG levels‡						
% Detection low BG	34 ± 29	44 ± 30	44 ± 27	$F = 3.5$ $P < 0.005$	$t = 2.4$ $P = 0.002$	$t = 0.5$ NS
% Detection high BG	51 ± 24	55 ± 26	53 ± 27	$F = 3.1$ $P < 0.001$	$t = 1.7$ $P < 0.05$	$t = 0.9$ NS
Accurate estimates	38 ± 11	45 ± 15	46 ± 15	$F = 13.6$ $P < 0.001$	$t = 4.3$ $P < 0.001$	$t = 0.6$ NS
Reduced extreme BG fluctuations§						
BG risk index	13.9 ± 4.4	13.3 ± 6.0	13.0 ± 5.2	$F = 2.1$ $P = 0.002$	$t = 3.7$ $P = 0.001$	$t = 0.01$ NS
HbA$_{1c}$	10.2 ± 2.0	10.2 ± 2.0	10.2 ± 1.9	$F = 0.1$ NS	$t = 0.0$ NS	$t = 0.5$ NS
Improved judgment‖						
% Decision to raise low BG	50 ± 27	59 ± 34	58 ± 30	$F = 3.6$ $P < 0.005$	$t = 2.6$ $P = 0.01$	$t = 2.2$ $P < 0.5$
% Decision to lower high BG	53 ± 26	54 ± 30	60 ± 28	$F = 5.2$ $P < 0.001$	$t = 3.3$ $P < 0.001$	$t = 2.2$ $P < 0.05$
% Decision not to drive when low	48 ± 33	50 ± 36	51 ± 31	$F = 2.0$ $P < 0.01$	$t = 2.7$ $P < 0.005$	$t = 0.3$ NS
Reduction of negative consequences¶						
DKA (total no.)	7	0	0	—	—	—
Severe hypoglycemia (mean episodes/month)	1.6 ± 2.0	1.2 ± 1.9	1.1 ± 2.0	$F = 3.9$ $P < 0.002$	$t = 2.3$ $P = 0.002$	$t = 0.8$ NS
Motor vehicle violations (mean violations/month)	0.09 ± 0.27	0.03 ± 0.09	0.03 ± 0.15	$F = 5.4$ $P < 0.001$	$t = 2.8$ $P = 0.001$	$t = 0.4$ NS
Improvement in psychologial parameters#						
Hypoglycemia fear survey–worry	22 ± 9.6	17.5 ± 10.7	17.4 ± 9.9	$F = 21.2$ $P < 0.001$	$t = 5.2$ $P < 0.002$	$t = 0.8$ NS
DQOL–impact	46.3 ± 8.7	44.0 ± 7.7	43.8 ± 8.3	$F = 6.7$ $P < 0.005$	$t = 3.1$ $P < 0.005$	$t = 1.0$ NS
DQOL–worry	18.3 ± 7.6	16.5 ± 8.7	16.2 ± 8.5	$F = 11.7$ $P < 0.001$	$t = 4.3$ $P < 0.001$	$t = 0.8$ NS
BDI–total	6.9 ± 5.6	5.8 ± 5.7	6.1 ± 6.2	$F = 2.4$ $P = 0.09$	$t = 1.6$ $P = 0.11$	$t = 0.6$ NS
DAS–diabetes conflict	19.1 ± 8.7	18.5 ± 8.3	18.9 ± 8.7	$F = 0.5$ NS	$t = 0.5$ NS	$t = 0.7$ NS
Knowledge	43.2 ± 4.2	46.8 ± 3.3	46.3 ± 3.5	$F = 61.7$ $P < 0.001$	$T = 8.2$ $P < 0.001$	$t = 1.4$ NS

Data are means ± SD unless otherwise indicated. *Contrast 1 compared the 6-month baseline with the 6- and 12-month follow-up data to determine whether there was a long-term benefit of BGAT-2; †contrast 2 compared posttreatment (assessment 3, Fig. 1); ‡$F = 4.0$, $P < 0.01$, MANOVA; §no MANOVA was performed because only one variable, BG risk index, was hypothesized to change; ‖$F = 2.7$, $P = 0.05$, MANOVA; ¶$F = 4.5$, $P < 0.005$, MANOVA; #$F = 14.9$, $P < 0.0001$, MANOVA.

BGAT-2 (MANOVA $F = 2.7$, $P < 0.05$). BGAT-2 led to a significant increase in decision to treat (raise/lower) when the BG level was low/high from baseline through 12 months of follow-up ($P = 0.001$), and was stable across 6- and 12-month follow-up ($P'g > 0.5$). Similarly, decisions concerning avoiding driving a motor vehicle when BG levels were low significantly increased from baseline through 12 months of follow-up ($P = 0.004$), and was stable from 6 to 12 months of follow-up ($P = 0.74$).

Sequelae

Negative sequelae of extreme BG levels were significantly reduced by BGAT-2 (MANOVA $F = 4.5$, $P < 0.005$). During baseline assessment, there were six episodes of DKA among our subjects: one event occurred during BGAT-2 training, and none occurred during the 12 months after BGAT-2. Severe hypoglycemia was reduced by a third across the first and last 6 months of follow-up ($P < 0.002$) (Table 2). BGAT-2 led to a significant reduction in motor vehicle violations (by ~67%) from

baseline through 12 months of follow-up ($P = 0.001$), and was stable from 6 to 12 months of follow-up ($P = 0.74$).

Psychological impact

BGAT-2 significantly improved psychological functioning (MANOVA $F = 14.9$, $P < 0.0001$). Specifically, worry scores concerning hypoglycemia were reduced from baseline through 12 months of follow-up ($P < 0.001$), and was stable across 6 and 12 months of follow-up ($P = 0.80$). Diabetes quality of life, for both the im-

pact and worry subscales, was significantly improved from baseline through 12 months of follow-up ($P < 0.001$), and was stable across 6 and 12 months of follow-up ($P > 0.40$). Knowledge about diabetes was improved from baseline through 12 months of follow-up ($P < 0.001$), and was stable across 6 and 12 months of follow-up ($P = 0.17$). Neither depression nor dyadic conflict was improved, partly because baseline scores were within normal limits (14,15). However, when we examined the 21 subjects who had BDI scores >9, indicating the presence of at least mild depression, these individuals did demonstrate a significant ($P = 0.05$) reduction in symptoms from a mean baseline score of 14.5 to 6- and 12-month mean scores of 11.4 and 11.2, respectively.

Booster training effects

There were no significant two-way interaction effects for booster versus no-booster and posttreatment versus 12-month follow-up. There were no three-way interaction terms involving booster, awareness, and time.

Post-hoc analyses

Subsequent analyses were performed to help understand mechanisms underlying the reduction in occurrences of severe hypoglycemia, motor vehicle violations, and DKA. It was hypothesized that reduction in severe hypoglycemia, for those who had such episodes at baseline, would be associated with improved detection of hypoglycemia, reduction of exposure to low BG levels (low BG index), and improved decision to self-treat when the BG level was low. A regression analysis was not significant, with improvement in self-treatment decisions only marginally correlated with improvement in severe hypoglycemia ($r = 0.19$, $P = 0.086$).

It was hypothesized that reduction in motor vehicle violations would be associated with improved detection of hypoglycemia, reduced exposure to low BG levels (low BG index), and improved judgment concerning not driving a motor vehicle when BG was low. A regression analysis predicting reduction of motor vehicle yielded an $R^2 = 0.47$ ($P = 0.03$), with only improvement in judgment not to drive entering into the equation (partial correlation $= 0.67$).

Because there were no episodes of DKA during follow-up and the seven episodes of DKA before BGAT were accounted for by only five subjects, statistical analyses were not appropriate. However, it is of interest to note that reduction of DKA was associated with slightly higher HbA_1 (11.4–11.8%) and an improvement in detection of high BG levels (>10 mmol, 49–63%) in these subjects.

CONCLUSIONS — These data indicate that BGAT-2, a programmatic training program directed at teaching adults with type 1 diabetes to better recognize and anticipate BG fluctuations, has significant, sustained, and broad-ranging benefits. Although no placebo group was used, for both practical and ethical reasons, the fact that dependent variables generally did not change as a function of time or monitoring, during either the 6 months of baseline or 12 months of follow-up, demonstrates the robust effects of BGAT-2. However, whereas improvement in BG estimation was statistically significant, the improvement in detection of hypoglycemia and hyperglycemia was relatively modest. Furthermore, improvement in detection did not correlate with reduction of severe hypoglycemia or motor vehicle violations, indicating that the beneficial effects of BGAT-2 are not solely a function of better recognition of hypoglycemia and hyperglycemia.

However, in BGAT, patients are also taught to use the feedback provided by BG estimation and SMBG more appropriately. This is reflected in both the significant improvements in decisions to treat and not to drive a motor vehicle when the BG level was low, suggesting that changes in decision-making and attitude may be just as important as improvements in BG detection. In fact, improved decisions not to drive a motor vehicle when the BG level was low significantly predicted reduction of motor vehicle violations ($R^2 = 0.47$). The importance of this fact is underlined by recent field (17) and laboratory studies (18) demonstrating that drivers with type 1 diabetes often do decide they can drive while hypoglycemic. The positive impact of BGAT on driving mishaps was confirmed by our previous long-term follow-up study (6) and is the only intervention shown to reduce such life-threatening events.

BGAT also teaches subjects to better anticipate future extreme BG levels. Anticipation and prevention of extreme BG levels may account for both the reductions in DKA and severe hypoglycemia. Although we have no data to directly test this speculation, post-hoc analysis of the current data demonstrated that the significant reduction in severe hypoglycemia was due to reduction in nocturnal hypoglycemia. It seems likely that this reduction in nocturnal severe hypoglycemia is not due to improved detection of hypoglycemic symptoms but rather the improved ability to anticipate and prevent low BG levels during sleep.

In addition to being the first experimental study to demonstrate the reduction in severe hypoglycemia and DKA without manipulating diabetic regimen or metabolic control, this is the first study demonstrating the improvement in both fear of hypoglycemia and quality of life. This may be very important, because concerns about hypoglycemia are suggested to be the major barrier to intensive insulin therapy (19). In addition, BGAT seemed to reduce depression in subjects who were mildly depressed. In conclusion, BGAT produced persistent broad-ranging effects relevant to both hypoglycemia and hyperglycemia among adults with type 1 diabetes. This intervention may be particularly beneficial to patients who are attempting intensive insulin therapy, are bothered with frequent DKA, have had severe hypoglycemia- or diabetes-related car accidents, or experience wide fluctuations in BG levels or impaired hypoglycemia awareness. To date, BGAT has not been tested either with children or with adults who have type 2 diabetes.

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