change of action is generally achieved based on the perception and reflection of self action (6–8). This graph helped patients understand both the treatment goal and long-term variations of diabetes control level, which effectively enabled them to adhere to the appropriate therapeutic regimens by modifying their behavior.

The significant improvement of glycemic control was observed irrespective of age, sex, duration of diabetes, BMI, complications, or treatment regimens, indicating the evident impact of the graph among all patient groups. The Wilcoxon rank-sum test revealed that the impact of the HbA_{1c} graph was stronger in younger patients and in patients with neuropathy. A greater improvement in HbA_{1c} was also observed in poorly controlled patients, which suggests that poorly controlled patients are more highly motivated to improve their control levels. In addition, fairly well-controlled people were able to maintain their even control, which also suggests that this graph is useful in maintaining a good control level.

We should be careful, however, when advising patients with advanced retinal complications, because a rapid lowering of glucose levels might aggravate the retinal complications (9,10). We experienced some poorly controlled patients who, upon seeing a poor result, became depressed. Therefore, appropriate modification in the education for those patients is required to avoid a rapid progression of complications and prevent psychological depression. At least, during our study, we did not observe any rapid progression of complications.

The color-classified HbA_{1c} graph is, therefore, considered useful not only for the instruction of HbA_{1c} but also to motivate patients to improve their glycemic control. The color-classified HbA_{1c} graph serves as a fundamental device for helping diabetic patients reach an appropriate control goal. Furthermore, similar educational strategies can potentially be applied to patients with other chronic diseases, including hypertension, hyperlipidemia, and hyperuricemia.

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NIDDM, non-insulin-dependent diabetes mellitus; BMI, body mass index; HPLC, high-performance liquid chromatography.

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An Unsuspected Factor Contributing to Pizza Hyperglycemia

We read with interest the article by Ahern et al. (1) regarding the hyperglycemic effect of pizza in diabetic patients. This effect of pizza is a well-known phenomenon in our practice. Quite recently the father of one of our diabetic children, a 9-yr-old boy who uses an insulin infusion pump, conducted an isolated search for an explanation of this pizza-effect. This study revealed an unsuspected explanation for the post-pizza hyperglycemia: 9 of 11 pizza-makers interviewed admitted surreptitiously adding some cane sugar to the dough to give the pizza its peculiar

crackling sensation. This phenomenon should be taken into account as an extra factor adding to the results observed by Ahern et al. (1) (at least in Italy). Obviously, this cannot be the sole explanation because the pizza used in the study was prepared by the staff of the New Haven Hospital and contained only 17.9 g mono- and disaccharides.

We have recently investigated the matter further and have obtained evidence that cane sugar is also customarily added to bread, in this case especially, to give it the brown color of the crust. We are actually planning an experiment, with the help of some diabetic volunteers, to evaluate the impact of this manipulation to the glycemic index.

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The Pizza Saga

Ahern et al. (1) found that, in IDDM patients, a pizza meal produced a higher glycemic response than a control meal of the same composition, which contained high GI foods. However, when the GIs of the meals are calculated (Table 1), the pizza meal has a higher value than the control meal and, thus, may be expected to produce a higher glycemic response.

The GI analysis is based on USDA food tables (2) and published mean GI values for the major foods (3). White flour was ascribed the same GI value as white bread, which is also made from flour, and we have recently shown that pizza has the same glycemic response as bread (T.W., unpublished observations). The GI of the vegetables is based on their content of simple sugars, which roughly predicts the GI of fruits (4,5). The type of potato and whether it was fed with or without skin is not stated; I assumed no skin. The GI of white potato is 80, whereas a baked, Idaho russet potato is 116 (3). Using the GI for white or russett potatoes, respectively, results in GIs of 77.2 or 86.4 for the control meal—18 or 8.5% less than that of the pizza meal, which is 94.5 for the pizza meal (adjusting for unequal CHO). The former difference is similar to the 25% observed difference in incremental glycemic response area (calculated from Table 1).

Ahern et al. (1) concluded that pizza has properties that accentuate and sustain hyperglycemia. GI analysis suggests that the responsible factor is white flour.

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Table 1—*Meal GI calculation*

Pizza meal				Control meal			
	CHO (g)	Food GI	Meal GI		CHO (g)	Food GI	Meal GI
Food				Food			
Canned tomato	14.4	76	8.8	Bread	20.2	100	16.0
Tomato paste	4.4	76	2.7	Margarine	—	—	—
Olive oil	—	—	—	Mayonnaise	4.8	86	3.3
Onions	1.9	85	1.3	Turkey	—	—	—
Garlic	0.5	90	0.3	Cheese	3.3	46	1.2
Sucrose	6.7	86	4.6	Baked potato	32.3	80	20.5
Parmesan cheese	0.4	46	0.2	Lettuce	1.8	77	1.1
Mozzarella	4.3	46	1.6	Tomato	1.4	76	0.8
White flour	91.0	100	73.1	Oil	—	—	—
Yeast	0.9	100	0.7	Vinegar	—	—	—
Salt	—	—	—	Raisins	31.7	93	23.4
				Apple juice	30.6	45	10.9
Total	124.5	—	94.5*		126.1	—	77.2

*Adjusted for unequal CHO: 93.3 × 126.1/124.5 = 94.5.