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.

PATRIZIO TATTI, MD
PATRIZIA DI MAURO, MD
RICCARDO GUARISCO, MD

From the Centro Antidiabetico, Ospedale S. Giuseppe di Marino, (Ciampino-Marino), Rome, Italy.

Address correspondence to Patrizio Tatti, MD, Ospedale S. Giuseppe Di Marino, the Centro Antidiabetico, USL Rm. 32, Ciampino-Marino, Rome, Italy.

Reference

1. Ahern J, Gatcomb PM, Held NA, Petit WA, Tamborlane WV: Exaggerated hyperglycemia after a pizza meal in well-controlled diabetes. *Diabetes Care* 16: 578–80, 1993

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which is also made from flour, and we have recently shown that pizza has the same glycemic response as bread (T.W., unpublished observations).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.

THOMAS WOLEVER, MD, PHD

From the Department of Nutritional Sciences, University of Toronto, Toronto, Ontario, Canada.

Address correspondence to Thomas Wolever, MD, PhD, University of Toronto, Department of Nutritional Sciences, Fitzger-

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.

ald Building, 150 College Street, Toronto, Ontario, Canada M5S 1A8.

IDDM, insulin-dependent diabetes mellitus; GI, glycemic index; USDA, United States Department of Agriculture; CHO, carbohydrate.

References

1. Ahern JA, Gatcomb PM, Held NA, Petit WA, Tamborlane WV: Exaggerated hyperglycemia after a pizza meal in well-controlled diabetes. *Diabetes Care* 16: 578–80, 1993
2. United States Department of Agriculture: *Nutrient Database for Standard Reference, Full Version, Release 9.* (NTIS Order No. PB90–502527) NTIS Federal Computer Products Center, Springfield, VA
3. Wolever TMS: *The Glycemic Index: Aspects of Some Vitamins, Minerals and Enzymes in Health and Disease. World Rev Nutr Diet*, Bourne GH, Basel, Switzerland, Karger, Ed. 62:120–85, 1990
4. Ha M-A, Mann JI, Melton LD, Lewis-Barned NJ: Relationship between the glycaemic index and sugar content of fruits. *Diab Nutr Metab* 5:199–203, 1992
5. Wolever TMS, Vuksan V, Katzman-Relle L, Jenkins AL, Josse RG, Wong GS, Jenkins DJA: Glycaemic index of fruits and fruit products in patients with diabetes. *Int J Food Sci Nutr.* 43:205–12, 1993

Response To Wolever

We would like to thank Dr. Wolever for useful and insightful comments. He concludes that pizza has a higher GI, which results in higher blood glucose levels. However, we are reluctant to attribute the greater, late postprandial rise in plasma glucose, following the pizza meal compared with the control meal, solely to a higher GI for pizza. In most studies, the GI of a food is evaluated by the postprandial rise in plasma glucose within 2–3 h after ingestion of the food. In our study (1), blood glucose levels were similar for 3 h after both meals. It was only after 5 and 8 h that glucose levels were significantly higher after the pizza meal.

We appreciate Dr. Wolever's interest and expertise in this field and again thank him for his comments.

JoAnn Ahern, RN, MSN, CDE
Nancy Held, MS, RD, CDE
William Tamborlane, MD

From the Yale Program for the Management of Type I Diabetes, New Haven, Connecticut.

Address correspondence to JoAnn Ahern, MD, The Yale Program for the Management of Type I Diabetes, 20 York Street, Hunter 530, New Haven, CT, 06540.

GI, glycemic index.

References

1. Ahern JA, Gatcomb PM, Held NA, Petit WA, Tamborlane WV: Exaggerated hyperglycemia after a pizza meal in well-controlled diabetes. *Diabetes Care* 16: 578–80, 1993

Change of Lipoprotein(a) and Coagulative or Fibrinolytic Parameters in Diabetic Patients with Nephropathy

Lp(a) is a plasma lipoprotein of high atherogenicity that competes with plasminogen at the site of plasminogen receptors (1). We know diabetic patients show a hypercoagulable state, which might contribute to diabetic vascular complications. The role of Lp(a) in fibrinolysis in general and in diabetes in particular is a timely and important issue. In this study, we measured various lipoprotein and coagulative or fibrinolytic parameters in 3 groups of subjects: *1*) normal control subjects (n = 51), 2) NIDDM subjects without nephropathy (n = 39, no diabetic retinopathy and <50 mg/g · creatinine of urinary AEI in all subjects, and 3) NIDDM subjects with nephropathy (n = 29, diabetic retinopathy and >200 mg/g · creatinine of AEI in all subjects.

Creatinine was measured by Jaffe's rate assay. Urinary albumin was measured by means of latex turbidimetric immunoassay. TG and total cholesterol were measured by means of enzymatic determination. LDL cholesterol was measured by means of heparin/Ca precipitation method. ApoB100 and apoA-I were measured by a single radial immunodiffusion method. Lp(a) was measured by an ELISA method (Tint Elisa, Bio pool, Sweden). PT, APTT, and fibrinogen were measured by Baxter's kit. TAT and α2PIC were measured by enzyme immunoassay (for the former, Hoext Japan's kit, for the latter, Teijin's kit). D dimer was measured by an ELISA method (Dimertest EIA). The data were analyzed by a Student's t test.

BMI levels were not significantly different among the 3 groups. Fasting blood glucose and HbA_{1c} levels were not significantly different between the 2 diabetic groups. Levels of creatinine, TG, total cholesterol, LDL, apoB100, apoA-I, PT, APTT, and fibrinogen were not significantly different ($P > 0.05$) among the 3 groups; however, the diabetic subjects, in particular those with nephropathy, tended to have higher levels of TAT, α2PIC, and D-dimer as well as Lp(a). A significant positive correlation was detected between Lp(a) and α2PIC ($r = 0.4002$, $P < 0.05$) among the diabetic patients. α2PIC showed a significantly positive correlation with TAT ($r = 0.6188$, $P < 0.01$) in the diabetic patients, however, this significant correlation was not observed in the normal group. The observation was thought to