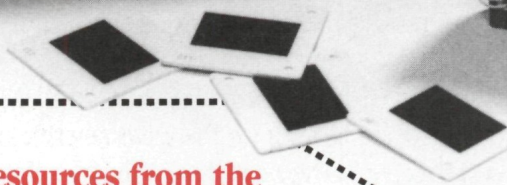
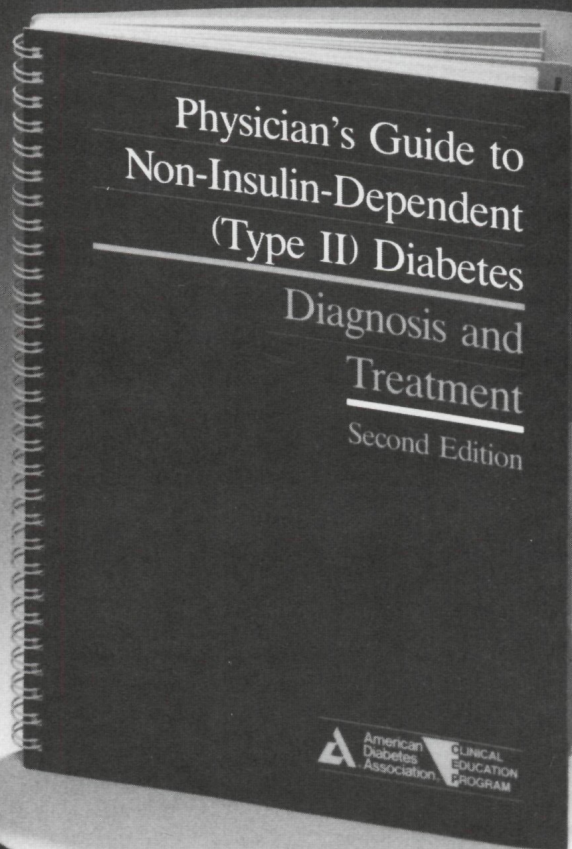
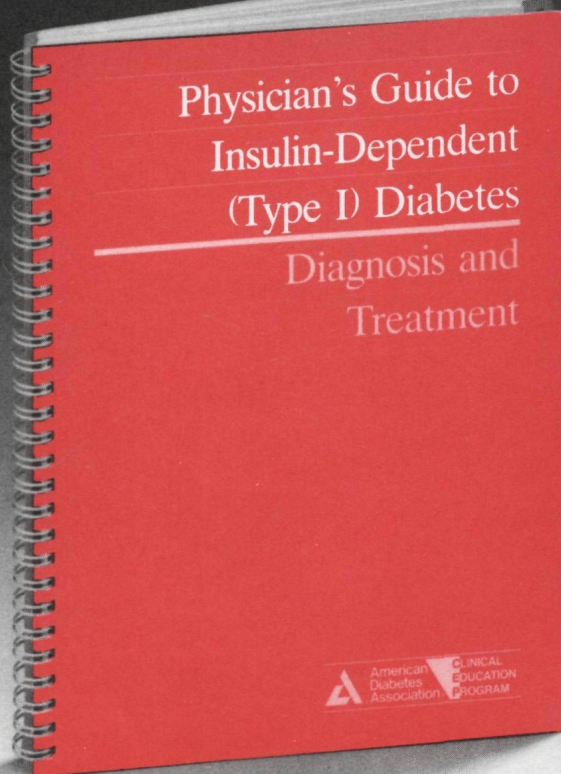


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Book Review, JAMA, May 26, 1989
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I would like to invite you to the 25th International Research Symposium of the American Diabetes Association, September 26-28, 1990, at the Marine Biological Laboratories in Woods Hole, Massachusetts.

During this 2½-day symposium, the possible mechanisms of hyperglycemia's adverse effects on insulin action and secretion, and metabolic function in vascular and neurological cells will be explored. With each topic, clinical evidence will be presented in combination with studies on the basic mechanism of hyperglycemia's effect at the cellular, biochemical, and molecular biological level.

The format of the meeting will be five half-day sessions. Original research will be presented as a series of plenary sessions, as well as abstract presentations.

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Looking forward to seeing you at Woods Hole.

George L. King, MD
Chairman, 25th International Research
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25th International Research Symposium of the American Diabetes Association

“How Does Hyperglycemia Cause Insulin Resistance, Complications and β -Cell Dysfunction in Diabetes Mellitus?”

Wednesday, September 26

7:30–8:30 am BREAKFAST

8:30–8:40 am

Welcome and Introduction—George L. King, MD, Symposium Chairman, Joslin Diabetes Center, Boston, Massachusetts

Session I

How Does Hyperglycemia Cause Insulin Resistance?

Moderators: Samuel W. Cushman, PhD and Jeffrey S. Flier, MD

8:40–9:15 am

Glucose Transport and Glucose Transporters: An Overview—Samuel W. Cushman, PhD, National Institutes of Health, Bethesda, MD

9:15–9:35 am

Studies Involving Human Adipocytes—Ulf Smith, MD, Sahlgrenska Hospital, Gothenburg, Sweden

9:35–9:55 am

Regulation of Insulin Action by Glucose and Insulin—W. Timothy Garvey, MD, Veterans Administration Medical Center, Indianapolis, IN

9:55–10:15 am

Hyperglycemia Causes Insulin Resistance—Luciano Rossetti, MD, University of Texas Health Science Center, San Antonio, TX

10:15–10:45 am BREAK

10:45–11:05 am

Effect of Insulin and Glucose on Phospholipids and Protein Kinase C—Robert J. Pollet, MD, PhD, Veterans Administration and Emory Medical Centers, Decatur, GA

11:05–11:30 am GENERAL DISCUSSION

11:30 am–1:30 pm LUNCH

Session II

How Does Hyperglycemia Decrease Insulin Secretion from the Islets?

Moderators: Barbara E. Corkey, PhD and Gordon C. Weir, MD

1:30–1:55 pm

Regulation of Insulin Secretion—Franz M. Matschinsky, MD, University of Pennsylvania School of Medicine, Philadelphia, PA

1:55–2:20 pm

Effect of Glucose on Phospholipid and Protein Kinase C—Claes B. Wollheim, MD, University of Geneva, Switzerland

2:20–2:45 pm

Desensitization of Insulin Secretion—Gerold M. Grodsky, PhD, University of California, San Francisco, CA

2:45–3:15 pm

Hyperglycemia and Structural Damage to β Cell—Roger H. Unger, MD, University of Texas Southwestern Medical School, Dallas, TX

3:15–3:45 pm BREAK

3:45–4:10 pm

Possible Mechanisms of Glucose's Adverse Effects on Islet Cells—Gordon C. Weir, MD, Joslin Diabetes Center, Boston, MA

4:10–4:30 pm GENERAL DISCUSSION

4:45–5:30 pm POSTER SESSION

5:30–7:00 pm DINNER

Thursday, September 27

7:30–8:30 am BREAKFAST

Session III

How Does Hyperglycemia Affect Vascular Cell Metabolism and Function?—Part I

Moderators: Peter F. Kador, PhD and George L. King, MD

8:30–8:50 am

Hyperglycemia and the Pathogenesis of Diabetic Vascular Complications—Timothy S. Kern, PhD, University of Wisconsin, Madison, WI

8:50–9:10 am

Regulation of Arterial Na^+K^+ ATPase and the Mechanism of Diabetic Complications—David A. Simmons, MD, University of Pennsylvania School of Medicine, Philadelphia, PA

9:10–9:30 am

Redox Potential and Vascular Dysfunction—Joseph R. Williamson, PhD, Washington University School of Medicine, St. Louis, MO

9:30–9:50 am

Changes in Prostaglandin Production—Daniel Deykin, MD, Boston University School of Medicine, Boston, MA

9:50–10:15 am

Changes in Vascular Protein Kinase C and Phospholipids—George L. King, MD, Joslin Diabetes Center, Boston, MA

10:15–10:45 am BREAK

10:45–11:00 am GENERAL DISCUSSION

11:30 am–1:30 pm LUNCH

Session IV

How Does Hyperglycemia Affect Vascular Cell Metabolism and Function?—Part II

Moderator: Michael Brownlee, MD

1:30–1:55 pm

Chemistry of Advanced Glycosylation Endproducts Formed In Vivo—Vincent M. Monnier, MD, Case Western Reserve University, Cleveland, OH

1:55–2:20 pm

Effects of Glycation on Molecular Interactions of Basement Membrane Molecules—Leo T. Furcht, MD, University of Minnesota, Minneapolis, MN

2:20–2:45 pm

High Glucose and Increased Expression of Basement Membrane Components—Mara Lorenzi, MD, Harvard Medical School, Boston, MA

2:45–3:15 pm

Cell-Mediated Interactions of Advanced Glycosylation Endproducts and the Vascular Wall—Helen Vlassara, MD, Rockefeller University, New York, NY

3:15–3:45 pm BREAK

3:45–4:10 pm

Pharmacologic Modulation of Advanced Glycosylation Product Formation—Michael Brownlee, MD, Albert Einstein College of Medicine, Bronx, NY

4:10–4:30 pm GENERAL DISCUSSION

4:45–5:30 pm POSTER SESSION

5:30–7:00 pm DINNER

Friday, September 28

7:30–8:30 am BREAKFAST

Session V

Neuropathy—How Does Hyperglycemia Cause Dysfunction in Nerve Tissue?

Moderators: Daniel Porte, Jr., MD and Neil Ruderman, MD, PhD

8:30–8:55 am

Role of Aldose Reductase and Na^+K^+ ATPase—Douglas A. Greene, MD, University of Michigan Medical Center, Ann Arbor, MI

8:55–9:20 am

The Knowledge Gained from the Cloning of Aldose Reductase—Kenneth H. Gabbay, MD, Baylor College of Medicine, Houston, TX

9:20–9:45 am

Alterations in CNS Transport and Metabolism in Hyperglycemic States—Anthony L. McCall, MD, PhD, Oregon Health Science University, Portland, OR

9:45–10:00 am BREAK

10:00–10:30 am GENERAL DISCUSSION

10:30–10:45 am CONCLUDING REMARKS

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<i>Diabetes Forecast</i>	•	•	•	•
<i>Diabetes '89</i>	•	•	•	•
<i>Professional Section Report</i>	•	•	•	•
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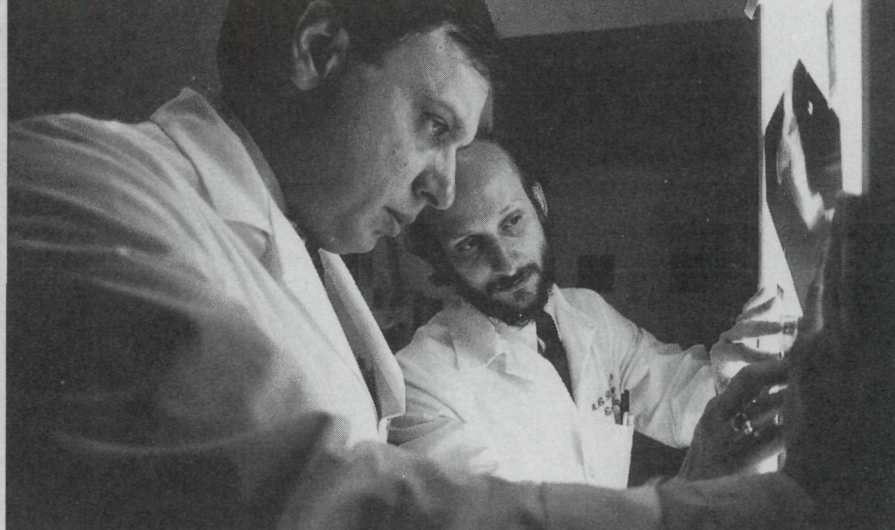
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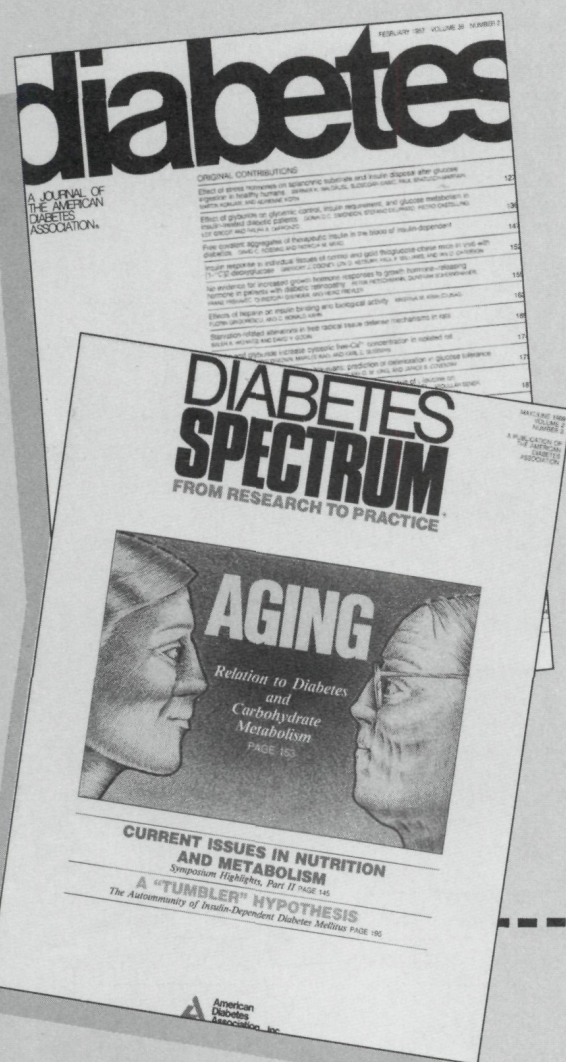
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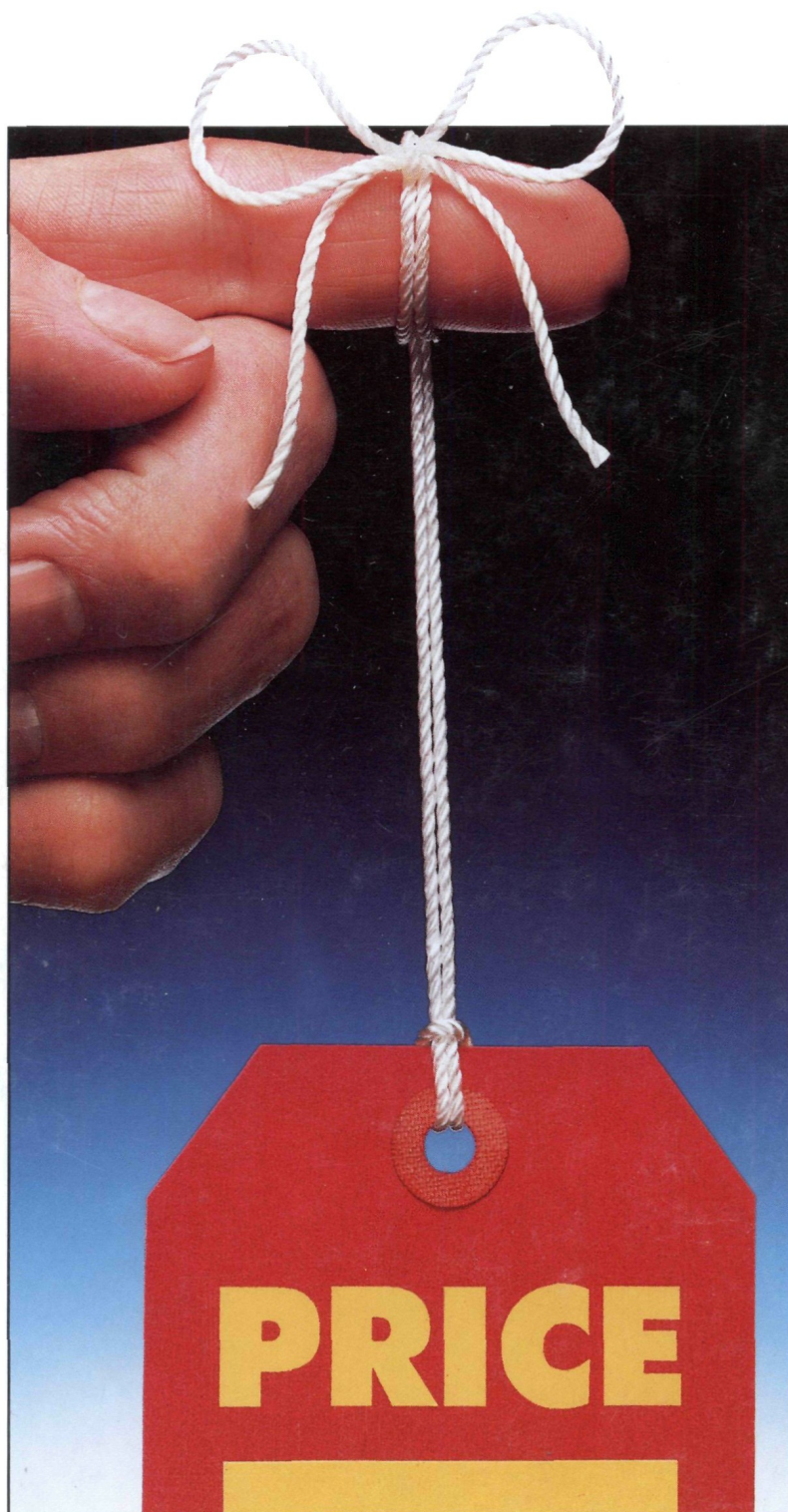
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References: 1. Data on file, Boehringer Mannheim Diagnostics.
2. Goldberg N, Trundle D, Prinsen J, et al: Evaluation of three blood glucose monitors for precision, accuracy, and patient ratings, abstract. *Diabetes* 1989; 38(suppl 2):169A.

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