We are very sad to report that Dr Ross Gerrity died of a heart attack at his home in Augusta, Ga on Tuesday, July 1, 2008. Ross played a major role in our understanding of the process of atherosclerosis over a period of over 40 years. He was an outstanding pathologist who was also interested in disease mechanisms. His major discoveries included the recognition that in normal animals the areas of aorta susceptible to atherosclerosis had a different cellular composition.1 He showed that, in animals fed a high-fat diet, lipoproteins first accumulated selectively in these susceptible areas. Today, the idea that inflammation plays an important role in the initiation of atherosclerosis is well accepted. However, in Ross Gerrity’s early career the role of monocytes was considered relatively unimportant. A major contribution of his research was recognition of the predominant role of monocytes in the process of atherogenesis. Ross was the first to document the early entry of monocytes but not neutrophils into the susceptible areas of the vessel wall in cholesterol-fed animals.2,3 These 2 papers were recognized by Nature Medicine as among the 100 most significant of the 20th century. He demonstrated that these monocytes took up the lipid that had accumulated, and that after some months smooth muscle cells proliferation was also increased in these areas. He documented that monocyte chemotactic factors accumulated in the lesion areas and, in collaborative studies with the UCLA group, that synthesis of one of the chemotactic factors, MCP-1, was induced by incubating endothelial cells and smooth muscle cells with oxidized lipoproteins. Ross also demonstrated that monocytes from hypercholesterolemic animals were more sensitive to chemotactic factors and showed that fat feeding caused monocytosis, thus effecting bone marrow as well as the vessel wall.4,5 The chemotactic factors mediating monocyte entry into lesions and the effects of lipids on monocyte maturation and phenotype are still being studied and remain important issues in atherosclerosis. One of Ross’ favorite discoveries was his finding in 2004 that αvβ3 integrin regulates macrophage foam cell formation via downregulation of CD36 and SR-A. In more recent years Ross was well known for his development of a pig model of diabetes and atherosclerosis.6 The atherosclerotic plaques that developed in these pigs were quite human-like, and ranged from fatty streaks to complicated plaques that ruptured; use of the diet allowed disease to develop over a period of months rather than years. Lynn became a close colleague and friend of Ross’ through his work on this pig model as part of a Program Project grant directed by Dr Jerry Nadler. NIH strongly recognized the major importance of this pig model for opening new research directions related to treatment of complications of Type 2 diabetes and for the development and testing of new pharmacotherapies. Indeed, one of Ross’ last research projects was the testing of short-acting versus long-acting insulins in this swine model to examine effects on blood glucose, tissue insulin resistance, and atherosclerosis.

Ironically, Ross died from complications of the diabetes disease he studied. Ross was a wonderful, intelligent gentleman with a great wit and great heart. He loved his family, his friends, and his science. We will miss his jolliness, his scientific intellect, and that signature twinkle in his eye. He made seminal discoveries in the field that the rest of us can now build on to help treat and prevent atherosclerosis and vascular complications of Type 2 diabetes.

References
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