But I definitely think that, since these were done in primates, a species which is closer to human species than the rodents and so forth, they definitely should be taken seriously and considered seriously.

Mr. GORDON. Thank you very much.

Dr. Sims, would you proceed with your statement? [1]

STATEMENT OF ETHAN A. H. SIMS, M.D., PROFESSOR OF MEDICINE, COLLEGE OF MEDICINE, UNIVERSITY OF VERMONT, BURLINGTON, VT.[2]

Dr. SIMS. Mr. Gordon and members of the committee a ritual of hornblowing seems to be in order at the beginning of these statements, so I will mention that I have had experience with a number of diabetic patients over a considerable period at Yale New Haven Hospital and in Vermont, though not as many as has Dr. Chester. I am a member of the workshop on obesity of the National Diabetes Commission and of the advisory and editorial group for the Fogarty International Center conferences on obesity. The background of a lot that I have to say is contained in the volume from the centers based on the last conference, which is to be released this summer by the Government Printing Office. I do not claim to be an expert in anything except in our research work persuading volunteers to gain weight.

I would like to acknowledge a major contribution to my written statement of my wife Dorothea, who is a Fellow in Health Care of the Radcliffe Institute, and is working on diabetes education, and also of my son Nat, who has been writing a history of the UGDP as his undergraduate thesis at Harvard. They have both been doing their best to educate me.

I have included a brief summary at the beginning of my written statement, but instead of that I will read a restatement of some of the points which I believe should be emphasized. To my knowledge they have not been emphasized at these hearings before.

I would like just to list the main points, which I want to be sure to get over. To my knowledge, they have not been emphasized in these hearings previously.

One: Obesity is now recognized as a factor predisposing to non-insulin dependent diabetes in those who are genetically susceptible. Untreated obesity represents a long-term risk in relation to cardiovascular and also other diseases.

Two: Insulin, in addition to its well-known action of lowering blood sugar, is a hormone which promotes the deposition of fat.

Three: The intense preoccupation with one aspect of the UGDP, the cardiovascular mortality, and the accompanying sometimes acrimonious debate has blurred our perception of the fact that at least 50 percent of the maturity onset diabetes in the study were overweight and underexercised and that both the sulfonylureas and insulin work to make them fatter. This is a threat to their well-being,

[1] See prepared statement, page 13676.

[2] Dr. Sims on sabbatical leave at the Endocrine Division Tufts-N. E. Medical Center, Boston, Mass.