Figure A1. Insulin-stimulated whole body glucose disposal in normal-glucose-tolerant (NGT) offspring of two T2DM parents and in healthy NGT subjects without family history of T2DM. From Gulli et al, reference #32.

Figure A2. Plasma insulin response during an OGTT and progression to T2DM in Pima Indians. From Lillioja, reference #3.
Figure A3. Plasma glucose, insulin, and C-peptide responses during an OGTT in normal-glucose-tolerant (NGT) offspring of two T2DM parents and in healthy NGT subjects without family history of T2DM. From Gulli et al, reference #32.

Figure A4. Insulin-stimulated glucose oxidative and non-oxidative glucose disposal in normal-glucose-tolerant (NGT) offspring of two T2DM parents and in healthy NGT subjects without family history of T2DM. From Gulli et al, reference #32.
Figure A5.  Insulin-mediated suppression of hepatic glucose production in normal-glucose-tolerant (NGT) offspring of two T2DM parents and in healthy NGT subjects without family history of T2DM.  From Gulli et al, reference #32.

Figure A6.  Plasma insulin and C-peptide responses during a +125 mg/dl hyperglycemic clamp in normal-glucose-tolerant (NGT) offspring of two T2DM parents and in healthy NGT subjects without family history of T2DM.  From Gulli et al, reference #32.
Figure A7. Insulin-stimulated whole body glucose uptake and forearm (muscle) glucose uptake/transport/phosphorylation in the insulin-resistant normal-glucose-tolerant (NGT) offspring of two T2DM parents, healthy NGT subjects without family history of T2DM, obese insulin-resistant NGT subjects, and lean/obese T2DM individuals during a euglycemic clamp. From Pendergrass et al, reference #45.

Figure A8. Natural history of insulin secretion and insulin sensitivity in insulin resistant (low-M) normal glucose tolerance (NGT) Pima Indians who progress (Progressors) and those who do not progress (Non-Progressors) to T2DM. From Weyer et al, reference #47.
Figure A9. Time course of effect of sustained (3 days) physiologic hyperinsulinemia (7 to 21 uU/ml) on insulin-stimulated total body glucose disposal (Rd) in healthy normal-glucose-tolerant subjects without family history of T2DM. From Del Prato et al, reference #50.

Figure A10. Effect of acute hyperinsulinemia on muscle tyrosine phosphorylation (IR-PY), IRS-1 tyrosine phosphorylation (IRS-1PY), and association of PI3-kinase activity with IRS-1 (PI3-K/IRS-1) in the normal-glucose-tolerant offspring of two T2DM parents and in healthy NGT subjects without FH of T2DM. From Pratipanawatr et al, reference #36.
Figure A11. IRS-1 serine phosphorylation in the basal state and basal/insulin-stimulated Akt phosphorylation in the normal-glucose-tolerant offspring of two T2DM parents and in healthy NGT subjects without FH of T2DM. From Morino et al, reference #57.

Figure A12. Insulin-mediated suppression of lipid oxidation and plasma FFA concentration in normal-glucose-tolerant (NGT) offspring of two T2DM parents and in healthy NGT subjects without family history of T2DM. From Gulli et al, reference #32.
Figure A13. Insulin-stimulated ATP synthesis, total body glucose disposal, and intramyocellular lipid content in the normal-glucose-tolerant offspring of two T2DM parents and in healthy NGT subjects without FH of T2DM. From Petersen, Short, et al, references #58 and #65.

Figure A14. Schematic representation of the role of insulin resistance and the organs responsible for the insulin resistance in the development of T2DM.
Figure A15. Plasma insulin response to oral glucose in Pima Indians and Caucasian subjects with normal glucose tolerance (top) and impaired glucose tolerance (bottom). From Lillioja et al, reference #68.