International Textbook of Diabetes Mellitus, 4th Ed., Chapter 7 - β-Cell biology of insulin secretion

References:

1. DeVos A,Heimberg H,Quartier E, et al.:Human and rat β cells differ in glucose transporter but not in glucokinase gene expression. Journal of Clinical Investigation 1995;96:2489–2495.
2. Matschinsky FM: Banting Lecture 1995. A lesson in metabolic regulation inspired by the glucokinase glucose sensor paradigm. Diabetes 1996;45:223–241.
3. Ishihara H, Asano T, Tsukuda K, et al.: Overexpression of hexokinase I but not GLUT1 glucose transporter alters concentration dependence of glucose-stimulated insulin secretion in pancreatic β-cell line MIN6. The Journal of Biological Chemistry 1994; 269:3081–3087.
4. Froguel P, Zouali H, Vionnet N, et al.: Familial hyperglycemia due to mutations in glucokinase. Definition of a subtype of diabetes mellitus. New England Journal of Medicine 1993;328:697–702.
5. Sekine N, Cirulli V, Regazzi R, et al.: Low lactate dehydrogenase and high mitochondrial glycerol phosphate dehydrogenase in pancreatic β-cells.Potential role in nutrient sensing. The Journal of Biological Chemistry 1994;269:4895–4902.
6. Soejima A, Inoue K, Takai D, et al.:Mitochondrial DNA is required for regulation of glucose-stimulated insulin secretion in a mouse pancreatic β cell line, MIN6. The Journal of Biological Chemistry 1996;271:26194–26199.
7. Tsuruzoe K, Araki E, Furukawa N, et al.: Creation and characterization of a mitochondrial DNA-depleted pancreatic β-cell line: impaired insulin secretion induced by glucose, leucine, and sulfonylureas. Diabetes 1998;47:621–631.
8. MacDonald MJ, Warner TF, Pellett JR: Increased mitochondrial glycerol phosphate dehydrogenase activity in insulinomas of two hypoglycemic infants. Journal of Clinical Endocrinology & Metabolism 1983;57:662–664.
9. Fernandez-Alvarez J, Conget I, Rasschaert J, et al.: Enzymatic, metabolic and secretory patterns in human islets of type 2 (non-insulin-dependent) diabetic patients. Diabetologia 1994;37: 177–181.
10. Eto K, Tsubamoto Y, Terauchi Y, et al.: Role of NADH shuttle system in glucose-induced activation of mitochondrial metabolism and insulin secretion. Science 1999;283:981–985.
11. Brown LJ, Koza RA, Everett C, et al.: Normal thyroid thermogenesis but reduced viability and adiposity in mice lacking the mitochondrial glycerol phosphate dehydrogenase. The Journal of Biological Chemistry 2002;277:32892–32898.
12. Corkey BE, GlennonMC, Chen KS, et al.: A role for malonyl-CoA n glucose-stimulated insulin secretion from clonal pancreatic β-cells.The Journal of Biological Chemistry 1989;264:21608–21612.
13. Deeney JT, Gromada J, Hoy M, et al.: Acute stimulation with long chain acyl-CoA enhances exocytosis in insulin-secreting cells (HIT T-15 and NMRI β-cells). The Journal of Biological Chemistry 2000;275:9363–9368.
14. Guay C,Madiraju SR, Aumais A, et al.: A role for ATP-citrate lyase, malic enzyme, and pyruvate/citrate cycling in glucose-induced insulin secretion. The Journal of Biological Chemistry 2007;282: 35657–35665.
15. Prentki M, Matschinsky FM: Ca2+, cAMP, and phospholipid derive messengers in coupling mechanisms of insulin secretion. Physiological Reviews 1987;67:1185–1248.

16. Ashcroft FM, Proks P, Smith PA, et al.: Stimulus-secretion coupling in pancreatic β cells. The Journal of Biological Chemistry 1994; 55(Suppl):54–65.
17. Rorsman P, Braun M, Zhang Q: Regulation of calcium in pancreatic α- and β-cells in health and disease. Cell Calcium 2012; 51:300–308.
18. Takasawa S, Akiyama T,NataK, et al.: CyclicADP-ribose and inosito 1,4,5-trisphosphate as alternate second messengers for intracellular Ca2+ mobilization in normal and diabetic β-cells. The Journal of Biological Chemistry 1998;273:2497–2500.
19. Pang ZP, Sudhof TC: Cell biology of Ca2+-triggered exocytosis. Current Opinion in Cell Biology 2010;22:496–505.
20. Gauthier BR, Wollheim CB: Synaptotagmins bind calcium to release insulin. American Journal of Physiology - Endocrinology and Metabolism 2008;295:E1279–1286.
21. Gustavsson N, Lao Y, Maximov A, et al.: Impaired insulin secretion and glucose intolerance in synaptotagmin-7 null mutant mice. PNAS 2008;105:3992–3997.
22. Henquin JC: Triggering and amplifying pathways of regulation of insulin secretion by glucose. Diabetes 2000;49:1751–1760.
23. MacDonald MJ, Tang J, Polonsky KS: Low mitochondrial glycerol phosphate dehydrogenase and pyruvate carboxylase in pancreatic islets of Zucker diabetic fatty rats. Diabetes 1996;45:1626–1630.
24. MacDonald MJ, Longacre MJ, Langberg EC, et al.:Decreased levels of metabolic enzymes in pancreatic islets of patients with type 2 diabetes. Diabetologia 2009;52:1087–1091.
25. Farfari S, Schulz V, Corkey B, et al.: Glucose-regulated anaplerosis and cataplerosis in pancreatic β-cells: possible implication of a pyruvate/citrate shuttle in insulin secretion. Diabetes 2000;49: 718–726.
26. Curry DL, Bennett LL, Grodsky GM:Dynamics of insulin secretion by the perfused rat pancreas. Endocrinology 1968;83:572–584.
27. Henquin JC: Regulation of insulin secretion: a matter of phase control and amplitude modulation. Diabetologia 2009;52:739–751.
28. Rorsman P, Renstrom E: Insulin granule dynamics in pancreatic β cells. Diabetologia 2003;46:1029–1045.
29. Olofsson CS, Gopel SO, Barg S, et al.: Fast insulin secretion reflects exocytosis of docked granules in mouse pancreatic B-cells. Pflügers Archiv 2002;444:43–51.
30. Straub SG, Sharp GW: Hypothesis: one rate-limiting step controls the magnitude of both phases of glucose-stimulated insulin secretion. American Journal of Physiology—Cell Physiology 2004;287:C565–571.
31. Shibasaki T, Takahashi H, Miki T, et al.: Essential role of Epac2/Rap1 signaling in regulation of insulin granule dynamics by cAMP. PNAS 2007;104:19333–19338.
32. Wang Z, Thurmond DC: Mechanisms of biphasic insulin-granule exocytosis -- roles of the cytoskeleton, small GTPases and SNARE proteins. Journal of Cell Science 2009;122:893–903.
33. Gerber SH, Sudhof TC: Molecular determinants of regulated exocytosis. Diabetes 2002;51(Suppl 1):S3–11.
34. Kasai H, Takahashi N, Tokumaru H: Distinct initial SNARE configurations underlying the diversity of exocytosis. Physiological Reviews 2012;92:1915–1964.

35. Vikman J, Ma X, Hockerman GH, et al.: Antibody inhibition of synaptosomal protein of 25 kDa (SNAP-25) and syntaxin 1 reduces rapid exocytosis in insulin-secreting cells. Journal of Molecular Endocrinology 2006;36:503–515.
36. Misura KM, Scheller RH, Weis WI: Three-dimensional structure of the neuronal-Sec1-syntaxin 1a complex. Nature 2000;404: 355–362.
37. Dulubova I, Sugita S, Hill S, et al.: A conformational switch in syntaxin during exocytosis: role of munc18. EMBO Journal 1999; 18:4372–4382.
38. Oh E, Kalwat MA, Kim MJ, et al.: Munc18-1 regulates first-phase insulin release by promoting granule docking to multiple syntaxin isoforms. The Journal of Biological Chemistry 2012;287: 25821–25833.
39. Richmond JE, Weimer RM, Jorgensen EM: An open form of syntaxin bypasses the requirement for UNC-13 in vesicle priming. Nature 2001;412:338–341.
40. Sudhof TC, Rizo J: Synaptic vesicle exocytosis. Cold Spring Harbor Perspectives in Biology 2011;3.
41. Yasuda T, Shibasaki T, Minami K, et al.: Rim2α determines docking and priming states in insulin granule exocytosis. Cell Metabolism 2010;12:117–129.
42. Kang L, He Z, Xu P, et al.: Munc13-1 is required for the sustained release of insulin from pancreatic β cells. Cell Metabolism 2006;3:463–468.
43. Grosshans BL, Ortiz D, Novick P: Rabs and their effectors: achieving specificity in membrane traffic. PNAS 2006;103:11821–11827.
44. Izumi T: Physiological roles of Rab27 effectors in regulated exocytosis. Endocrine Journal 2007;54:649–657.
45. Regazzi R, Ravazzola M, Iezzi M, et al.: Expression, localization and functional role of small GTPases of the Rab3 family in insulin-secreting cells. Journal of Cell Science 1996;109(Pt 9): 2265–2273.
46. Yi Z, Yokota H, Torii S, et al.: The Rab27a/granuphilin complex regulates the exocytosis of insulin-containing dense-core granules. Molecular and Cellular Biology 2002;22:1858–1867.
47. Ozaki N, Shibasaki T, Kashima Y, et al.: cAMP-GEFII is a direct target of cAMP in regulated exocytosis. Nature Cell Biology 2000;2: 805–811.
48. Wang J, Takeuchi T, YokotaH, et al.:Novel rabphilin-3-like protein associates with insulin-containing granules in pancreatic β cells. The Journal of Biological Chemistry 1999;274:28542–28548.
49. Gomi H,Mizutani S, Kasai K, et al.:Granuphilin molecularly docks insulin granules to the fusion machinery. Journal of Cell Biology 2005;171:99–109.
50. Ahrén B: Islet G protein-coupled receptors as potential targets for treatment of type 2 diabetes. Nature Reviews Drug Discovery 2009;8:369–385.
51. Aspinwall CA, Lakey JR, Kennedy RT: Insulin-stimulated insulin secretion in single pancreatic β cells. The Journal of Biological Chemistry 1999;274:6360–6365.
52. Kulkarni RN: Receptors for insulin and insulin-like growth factor-1 and insulin receptor substrate-1 mediate pathways that regulate islet function. Biochemical Society Transactions 2002; 30:317–322.
53. Luciani DS, Johnson JD: Acute effects of insulin on β-cells from transplantable human islets. Molecular and Cellular Endocrinology 2005;241:88–98.
54. Kaneko K, Ueki K, Takahashi N, et al.: Class IA phosphatidylinositol 3-kinase in pancreatic β cells controls insulin secretion by multiple mechanisms. Cell Metabolism 2010;12:619–632.
55. Drucker DJ: The biology of incretin hormones. Cell Metabolism 2006;3:153–165.
56. Holst JJ: The physiology of glucagon-like peptide 1. Physiological Reviews 2007;87:1409–1439.

57. Nauck MA: Unraveling the science of incretin biology. American Journal of Medical Genetics Part A 2009;122:S3–S10.
58. Winzell MS, Ahren B: Role of VIP and PACAP in islet function. Peptides 2007;28:1805–1813.
59. Soga T, Ohishi T, Matsui T, et al.: Lysophosphatidylcholine enhances glucose-dependent insulin secretion via an orphan G-protein-coupled receptor. Biochemical and Biophysical Research Communications 2005;326:744–751.
60. Overton HA, Babbs AJ, Doel SM, et al.: Deorphanization of a G protein-coupled receptor for oleoylethanolamide and its use in the discovery of small-molecule hypophagic agents. Cell Metabolism 2006;3:167–175.
61. Seino S, Shibasaki T: PKA-dependent and PKA-independent pathways for cAMP-regulated exocytosis. Physiological Reviews 2005;85:1303–1342.
62. Renstrom E, Eliasson L, Rorsman P: Protein kinase A-dependen and -independent stimulation of exocytosis by cAMP in mouse pancreatic β-cells. Journal of Physiology 1997;502:105–118.
63. Kashima Y, Miki T, Shibasaki T, et al.: Critical role of cAMPGEFII-Rim2 complex in incretin-potentiated insulin secretion. The Journal of Biological Chemistry 2001;276:46046–46053.
64. Gloerich M, Bos JL: Epac: defining a new mechanism for cAMP action. Annual Review Pharmacology and Toxicology 2010;50:355–375.
65. Shibasaki T, SunagaY, FujimotoK, et al.: InteractionofATPsensor, cAMP sensor, Ca2+ sensor, and voltage-dependent Ca2+ channel in insulin granule exocytosis. The Journal of Biological Chemistry 2004;279:7956–7961.
66. Eliasson L, Ma X, Renstrom E, et al.: SUR1 regulates PKA-independent cAMP-induced granule priming in mouse pancreatic B-cells. Journal of General Physiology 2003;121:181–197.
67. Fujimoto K, Shibasaki T, Yokoi N, et al.: Piccolo, a Ca2+ sensor in pancreatic β-cells. Involvement of cAMP-GEFII.Rim2. Piccolo complex in cAMP-dependent exocytosis. The Journal of Biological Chemistry 2002;277:50497–50502.
68. Kang G, Joseph JW, Chepurny OG, et al.: Epac-selective cAMP analog 8-pCPT-2'-O-Me-cAMP as a stimulus for Ca2+-induced Ca2+ release and exocytosis in pancreatic β-cells. The Journal of Biological Chemistry 2003;278:8279–8285.
69. Holz GG: Epac: A new cAMP-binding protein in support of glucagon-like peptide-1 receptor-mediated signal transduction in the pancreatic β-cell. Diabetes 2004;53:5–13.
70. Zhang CL, Katoh M, Shibasaki T, et al.: The cAMP sensor Epac2 is a direct target of antidiabetic sulfonylurea drugs. Science 2009; 325:607–610.
71. Seino S, Zhang CL, Shibasaki T: Sulfonylurea action re-revisited. Journal of Diabetes Investigation 2010;1:37–39.
72. Seino S, Shibasaki T, Minami K: Dynamics of insulin secretion and the clinical implications for obesity and diabetes. Journal of Clinical Investigation 2011;121:2118–2125.
73. Leiser M, Fleischer N: cAMP-dependent phosphorylation of the cardiac-type α1 subunit of the voltage-dependent Ca2+ channel in a murine pancreatic β-cell line. Diabetes 1996;45:1412–1418.
74. Safayhi H, Haase H, Kramer U, et al.: L-type calcium channels in insulin-secreting cells: biochemical characterization and phosphorylation in RINm5F cells. Molecular Endocrinology 1997; 11:619–629.
75. Thorens B, Deriaz N, Bosco D, et al.: Protein kinase A-dependent phosphorylation of GLUT2 in pancreatic β cells.The Journal of Biological Chemistry 1996;271:8075–8081.

76. Ilardi JM, Mochida S, Sheng ZH: Snapin: a SNARE-associated protein implicated in synaptic transmission. Nature Neuroscience 1999;2:119–124.
77. Song WJ, Seshadri M, Ashraf U, et al.: Snapin mediates incretin action and augments glucose-dependent insulin secretion. Cell Metabolism 2011;13:308–319.
78. Colledge M, Scott JD: AKAPs: from structure to function. Trends in Cell Biology 1999;9:216–221.
79. Creutzfeldt W: The incretin concept today. Diabetologia 1979; 16:75–85.
80. Kieffer TJ, Habener JF: The glucagon-like peptides. Endocrine Reviews 1999;20:876–913.
81. Vilsboll T, Krarup T, Madsbad S, et al.: Both GLP-1 and GIP are insulinotropic at basal and postprandial glucose levels and contribute nearly equally to the incretin effect of a meal in healthy subjects. Regulatory Peptides 2003;114:115–121.
82. Farilla L, Bulotta A, Hirshberg B, et al.: Glucagon-like peptide 1 inhibits cell apoptosis and improves glucose responsiveness of freshly isolated human islets. Endocrinology 2003;144:5149–5158.
83. Lynn FC, Pamir N, Ng EH, et al.: Defective glucose-dependent insulinotropic polypeptide receptor expression in diabetic fatty Zucker rats. Diabetes 2001;50:1004–1011.
84. Piteau S, Olver A, Kim SJ, et al.: Reversal of islet GIP receptor down-regulation and resistance to GIP by reducing hyperglycemia in the Zucker rat. Biochemical and Biophysical Research Communications 2007;362:1007–1012.
85. Younan SM, Rashed LA: Impairment of the insulinotropic effect of gastric inhibitory polypeptide (GIP) in obese and diabetic rats is related to the down-regulation of its pancreatic receptors. General Physiology and Biophysics 2007;26:181–193.
86. Kieffer TJ, Heller RS, Unson CG, et al.: Distribution of glucagon receptors on hormone-specific endocrine cells of rat pancreatic islets. Endocrinology 1996;137:5119–5125.
87. Jiang G, Zhang BB: Glucagon and regulation of glucose metabolism. American Journal of Physiology - Endocrinology and Metabolism 2003;284:E671–678.
88. Pipeleers DG, in't Veld PA, Van deWinkel M, et al.: A new in vitro model for the study of pancreatic A and B cells. Endocrinology 1985;117:806–816.
89. Sakamoto Y, Inoue H, Kawakami S, et al.: Expression and distribution of Gpr119 in the pancreatic islets of mice and rats: predominant localization in pancreatic polypeptide-secreting PP-cells. Biochemical and Biophysical Research Communications 2006;351:474–480.
90. Chu ZL, Jones RM, He H, et al.: A role for β-cell-expressed G protein-coupled receptor 119 in glycemic control by enhancing glucose-dependent insulin release. Endocrinology 2007;148: 2601–2609.
91. Schmitz-Peiffer C, Biden TJ: Protein kinase C function in muscle, liver, and β-cells and its therapeutic implications for type 2 diabetes. Diabetes 2008;57:1774–1783.
92. Ahrén B: Autonomic regulation of islet hormone secretion: implications for health and disease. Diabetologia 2000;43:393–410.
93. Rodriguez-Diaz R, Abdulreda MH, Formoso AL, et al.: Innervation patterns of autonomic axons in the human endocrine pancreas. Cell Metabolism 2011;14:45–54.
94. Itoh Y, Kawamata Y, Harada M, et al.: Free fatty acids regulate insulin secretion from pancreatic β cells through GPR40. Nature 2003;422:173–176.

95. Salehi A, Flodgren E, Nilsson NE, et al.: Free fatty acid receptor 1 (FFA(1)R/GPR40) and its involvement in fatty-acid-stimulated insulin secretion. Cell Tissue Research 2005;322:207–215.
96. Luo J, Swaminath G, Brown SP, et al.: A potent class of GPR40 full agonists engages the enteroinsular axis to promote glucose control in rodents. PLoS One 2012;7:e46300.
97. Kaku K, Araki T, Yoshinaka R: Randomized, double-blind, dose-ranging study of TAK-875, a novel GPR40 agonist, in Japanese patients with inadequately controlled type 2 diabetes. Diabetes Care 2013;36:245–250.
98. Sharp GW: Mechanisms of inhibition of insulin release. American Journal of Physiology 1996;271:C1781–1799.
99. Broglio F, Arvat E, Benso A, et al.: Ghrelin, a natural GH secretagogue produced by the stomach, induces hyperglycemia and reduces insulin secretion in humans. Journal of Clinical Endocrinology & Metabolism 2001;86:5083–5086.
100. Yamada Y, Reisine T, Law SF, et al.: Somatostatin receptors, an expanding gene family: cloning and functional characterization of human SSTR3, a protein coupled to adenylyl cyclase. Molecular Endocrinology 1992;6:2136–2142.
101. Yamada Y, Post SR, Wang K, et al.: Cloning and functional characterization of a family of human and mouse somatostatin receptors expressed in brain, gastrointestinal tract, and kidney. PNAS 1992;89:251–255.
102. Yamada Y, Kagimoto S, Kubota A, et al.: Cloning, functional expression and pharmacological characterization of a fourth (hSSTR4) and a fifth (hSSTR5) human somatostatin receptor subtype. Biochemical and Biophysical Research Communications 1993; 195:844–852.
103. Gromada J, Hoy M, Buschard K, et al.: Somatostatin inhibits exocytosis in rat pancreatic α-cells by Gi2-dependent activation of calcineurin and depriming of secretory granules. Journal of Physiology 2001;535:519–532.
104. Kailey B, van de BuntM, Cheley S, et al.: SSTR2 is the functionally dominant somatostatin receptor in human pancreatic β- and α-cells. American Journal of Physiology - Endocrinology and Metabolism 2012;303:E1107–1116.
105. Ullrich S, Wollheim CB: Expression of both α1- and α2-adrenoceptors in an insulin-secreting cell line. Parallel studies of cytosolic free Ca2+ and insulin release. Molecular Pharmacology 1985; 28:100–106.
106. Reimer MK, Pacini G, Ahrén B: Dose-dependent inhibition by ghrelin of insulin secretion in the mouse. Endocrinology 2003 144:916–921.
107. Dezaki K, Hosoda H, Kakei M, et al.: Endogenous ghrelin in pancreatic islets restricts insulin release by attenuating Ca2+ signaling in β-cells: implication in the glycemic control in rodents. Diabetes 2004;53:3142–3151.
108. Date Y, Kojima M, Hosoda H, et al.: Ghrelin, a novel growth hormone-releasing acylated peptide, is synthesized in a distinct endocrine cell type in the gastrointestinal tracts of rats and humans. Endocrinology 2000;141:4255–4261.
109. Rindi G, Savio A, Torsello A, et al.: Ghrelin expression in gut endocrine growths. Histochemistry and Cell Biology 2002;117: 521–525.
110. Eto K, Yamashita T, Tsubamoto Y, et al.: Phosphatidylinositol 3-kinase suppresses glucose-stimulated insulin secretion by affecting post-cytosolic [Ca2+] elevation signals. Diabetes 2002; 51:87–97.
111. Zawalich WS, Zawalich KC: A link between insulin resistance and hyperinsulinemia: inhibitors of phosphatidylinositol 3-kinase augment glucose-induced insulin secretion from islets of lean, but not obese, rats. Endocrinology 2000;141:3287–3295.

112. Muller D, Huang GC, Amiel S, et al.: Gene expression heterogeneity in human islet endocrine cells in vitro: the insulin signalling cascade. Diabetologia 2007;50:1239–1242.
113. Kulkarni RN, Bruning JC, Winnay JN, et al.: Tissue-specific knockout of the insulin receptor in pancreatic β cells creates an insulin secretory defect similar to that in type 2 diabetes. Cell 1999; 96:329–339.
114. Rothenberg PL, Willison LD, Simon J, et al.: Glucose-induced insulin receptor tyrosine phosphorylation in insulin-secreting β-cells. Diabetes 1995;44:802–809.
115. Leney SE,Tavare JM:Themolecular basis of insulin-stimulated glucose uptake: signalling, trafficking and potential drug targets. Journal of Endocrinology 2009;203:1–18.
116. Vanhaesebroeck B, Guillermet-Guibert J, Graupera M, et al.: The emerging mechanisms of isoform-specific PI3K signalling. Nature Reviews Molecular Cell Biology 2010;11:329–341.
117. Johnson JD, Misler S: Nicotinic acid-adenine dinucleotide phosphate-sensitive calcium stores initiate insulin signaling in human β cells. PNAS 2002;99:14566–14571.
118. Aspinwall CA, Qian WJ, RoperMG, et al.: Roles of insulin receptor substrate-1, phosphatidylinositol 3-kinase, and release of intracellular Ca2+ stores in insulin-stimulated insulin secretion in β -cells. The Journal of Biological Chemistry 2000;275:22331–22338.
119. Leibiger B,Moede T,Uhles S, et al.: Insulin-feedback via PI3K-C2α activated PKBα/Akt1 is required for glucose-stimulated insulin secretion. FASEB Journal 2010;24:1824–1837.
120. Dominguez V, Raimondi C, Somanath S, et al.: Class II phosphoinositide 3-kinase regulates exocytosis of insulin granules in pancreatic β cells. The Journal of Biological Chemistry 2011; 286:4216–4225.
121. Butler AE, Janson J, Bonner-Weir S, et al.: Beta-cell deficit and increased beta-cell apoptosis in humans with type 2 diabetes. Diabetes 2003;52:102–110.
122. McCulloch DK, Raghu PK, Johnston C, et al.: Defects in β-cell function and insulin sensitivity in normoglycemic streptozocin treated baboons: a model of preclinical insulin-dependent diabetes. Journal of Clinical Endocrinology & Metabolism 1988;67:785–792.
123. Kendall DM, Sutherland DE,Najarian JS, et al.: Effects of hemi pancreatectomy on insulin secretion and glucose tolerance in healthy humans. New England Journal of Medicine 1990;322:898–903.
124. Cerasi E, Luft R: The plasma insulin response to glucose infusion in healthy subjects and in diabetes mellitus. Acta Endocrinologica 1967;55:278–304.
125. Gerich JE: Is reduced first-phase insulin release the earliest detectable abnormality in individuals destined to develop type 2 diabetes? Diabetes 2002;51(Suppl 1):S117–121.
126. Polonsky KS, Given BD, Hirsch LJ, et al.: Abnormal patterns of insulin secretion in non-insulin-dependent diabetes mellitus. New England Journal of Medicine 1988;318:1231–1239.
127. Byrne MM, Sturis J, Fajans SS, et al.: Altered insulin secretory responses to glucose in subjects with a mutation in the MODY1 gene on chromosome 20. Diabetes 1995;44:699–704.