



Review Article

A review on management of diabetes mellitus among adult populationShivam Dubey^{1*} ¹Rani Durgavati Vishwavidyalaya, Jabalpur, Madhya Pradesh, India**Abstract**

Diabetes mellitus is a chronic, complex, and non-transmissible endocrine disease that is expanding quickly and has presented therapeutic difficulties all over the world. It is frequently associated with risks associated with patients' complex metabolic development. It is characterized by high blood levels of lipids and glucose as well as oxidative stress, which leads to long-term problems with many bodily organs, primarily the kidneys, eyes, nerves, and blood vessels. According to the World Health Organization, there is a significant risk of sickness and death from this epidemic. The illness also highlights a growing epidemic that is causing a significant socioeconomic strain on nations throughout the world. Since the standard approaches to treating diabetes mellitus have not fully addressed the underlying causes of the condition and are fraught with serious side effects, new therapy alternatives are emerging quickly.

Keywords: Human health, Diabetes mellitus, Metabolic disorder, Hyperglycemia.

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1. Introduction

The International Diabetes Federation predicted that 537 million adults worldwide will suffer from diabetes in 2021, and by 2045, the prevalence is expected to increase to about 800 million, or 10.9% of the adult population.¹ Hyperglycemia is not the sole symptom of type 2 diabetes mellitus; the disease also causes a number of consequences, including kidney failure, blindness, heart attacks, strokes, and amputations of lower limbs.² Type 2 diabetes mellitus is a condition with several causes linked to both polygenic and diverse environmental variables, according to growing evidence from epidemiological research.³ Because of genetic polymorphism and other risk factors, type 2 diabetes mellitus is therefore too complex to treat.

The yearly incidence of type 1 diabetes mellitus is increasing, despite the fact that type 2 diabetes mellitus associated with obesity accounts for the majority of cases.⁴ Approximately 10% of individuals with diabetes are estimated to have type 1 diabetes mellitus. Both types, however, carry the danger of decreased blood glucose and a longer-term risk of circulatory system complications.⁵ There

is substantial evidence that achieving normoglycemia would reduce the risk of complications associated with diabetes mellitus.⁶ However, in individuals with type 1 diabetes mellitus, the achievement of near normoglycemia is restricted by hypoglycemia episodes. Type 1 diabetes mellitus can develop in diabetics who are unaware of their hypoglycemia state, making it more difficult for them to achieve the necessary glycemic control. Many people with type 1 diabetes mellitus who have frequent low blood glucose levels are seen in diabetes mellitus health facilities worldwide, and the concept of hypoglycemia unconsciousness presents significant therapeutic difficulties. Fortunately, there are a lot of intriguing and beneficial advancements in the field of diabetes mellitus research, including gene therapy.⁷

At the moment, injecting insulin-like substances and taking hypoglycemic medications orally are the primary treatment approaches for type 2 diabetes mellitus. Despite having several adverse effects, these medications are essential in the treatment of type 2 diabetes mellitus.^{8,9} Since its creation, insulin has been the mainstay of treatment for uncontrolled insulin-deficient diabetic mellitus.¹⁰ It is true that the infusion of exogenous insulin is essential for life

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because of the acute shortage of beta cells. Despite the progress in understanding the causes, consequences, and persistence of diabetes mellitus, as well as the development of insulin and its analogs, there are still major challenges in maintaining strict glycemic control without adverse side effects like low blood glucose and weight gain.^{11,12} This emphasizes the significance of other methods or supplements to insulin.¹³

1.1. Etiology

Alpha cells that secrete glucagon and beta cells that produce insulin are the two primary categories of endocrine cells found in the pancreatic islets of Langerhans. Depending on the glucose environment, beta and alpha cells continuously alter the amount of hormone secreted. When insulin and glucagon are not balanced, the glucose levels become unnecessarily unbalanced. Hyperglycemia results from either insufficient or impaired insulin activity (insulin resistance) in people with diabetes mellitus. The hallmark of type 1 diabetes mellitus is the degeneration of pancreatic beta cells, which usually results from an autoimmune process. Type 2 diabetes mellitus has a more subtle beginning, with an imbalance between insulin levels and insulin sensitivity leading to a functional deficit of insulin. The outcome is the complete loss of beta cells, and as a result, insulin is either nonexistent or very low. Although there are many contributing factors, obesity and age are the most prevalent causes of insulin resistance.

Because glucose cannot be synthesized or stored in the brain, maintaining a near-normal glucose content is crucial for cardiovascular and central nervous system function.^{14,15} Endogenous glucose synthesis and peripheral tissue glucose utilization are dynamically and minute-by-minute regulated to maintain systemic glucose homeostasis.¹⁶ The liver and, to a lesser extent, the kidneys are responsible for gluconeogenesis or glycogenolysis, which produces glucose.^{17,18} The liver's process of turning non-carbohydrate precursors like lactate, alanine, and glycerol into glucose is

known as gluconeogenesis. Glycogen is produced by polymerizing excess glucose and is mostly stored in the muscle and liver.

Increased gluconeogenesis, faster glycogenolysis, and decreased peripheral tissue glucose uptake all contribute to hyperglycemia. Peripheral tissues' ability to absorb glucose is hampered by decreased insulin and an overabundance of counter-regulatory hormones (glucagon, cortisol, catecholamines, and growth hormone), which also promote lipolysis and protein breakdown (proteolysis). Osmotic diuresis brought on by hyperglycemia results in volume loss, a drop in glomerular filtration rate, and increasing hyperglycemia. At the cellular level, elevated blood glucose levels cause endothelial dysfunction by preventing the synthesis of nitric oxide and mitochondrial damage by producing reactive oxygen species. Immune system dysfunction results from hyperglycemia's elevation of pro-inflammatory cytokines including interleukin [IL]-6 and tumor necrosis factor- α (TNF- α). Eventually, these alterations may result in a higher risk of infection, poor wound healing, multiple organ failure, an extended hospital stay, and even death. Type 1 diabetes mellitus is typically diagnosed based on a characteristic history and elevated serum glucose levels (fasting glucose >126 mg/dL, random glucose >200 mg/dL, or hemoglobin A1C >6.5%), with or without antibodies to insulin and glutamic acid decarboxylase. Glycation hemoglobin tests and fasting glucose levels are helpful in the early detection of type 2 diabetes mellitus. If borderline, a glucose tolerance test can be used to assess blood glucose response to an oral glucose tolerance test as well as fasting glucose levels. A fasting blood glucose level of 100 to 125 mg/dL or a glucose level of 140 to 200 mg/dL two hours after an oral glucose tolerance test are indicative of prediabetes, which frequently occurs before type 2 diabetes mellitus.

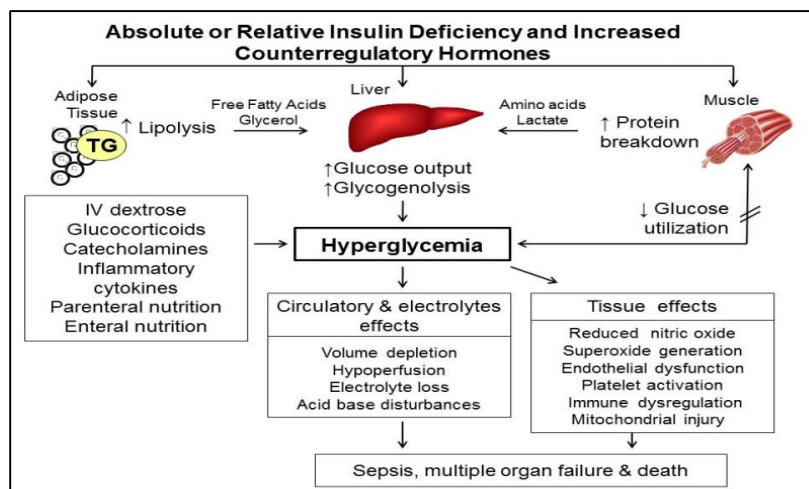


Figure 1: Pathogenesis of hyperglycemia.²³

Diabetes is linked to several risk factors. The development of diabetes is considerably accelerated by these risk factors. Age, weight, a family history of diabetes, smoking, and race/ethnicity are a few of these.^{19,20} Type 2 diabetes mellitus is a disorder that affects adults, whereas type 1 diabetes mellitus mostly affects young people. Age-related insulin secretion deficiencies and increasing insulin resistance brought on by changes in body composition enhance the risk of type 2 diabetes mellitus.²¹ Diabetes is a disorder that is closely linked to diabetes and an increase in body weight that results in obesity. This is because rising body weight causes insulin resistance to rise.²² (**Figure 1**)

Smokers have a 30–40% higher risk of developing type 2 diabetes mellitus than nonsmokers, according to the FDA. Additionally, smoking can lead to insulin resistance, which increases the amount of insulin needed by patients to regulate their blood sugar levels.²⁴ Diabetes is inherited. It is recommended that those with the family history follow lifestyle choices that lower their chance of acquiring diabetes.

2. Discussion

Diabetes mellitus is a multifactorial, progressive, and intricate metabolic disease that requires more sophisticated therapies over time. Researchers from all around the world have been hard at work finding and creating new medications to treat diabetes. More than 90% of adult instances of diabetes are caused by type 2 diabetes mellitus. The main factor causing persistent hyperglycemia in diabetic individuals is resistance to the effects of insulin. Activation of many pathways and variables related to insulin resistance and β -cell malfunction results in type 2 diabetes mellitus. Additionally, a complicated interaction between environmental and genetic variables is part of the genesis of type 2 diabetes mellitus. To improve diabetes control, lifestyle changes can effectively regulate this interaction. The purpose of this study is to explore the primary causes of type 2 diabetes mellitus that are not addressed by anti-diabetic medication.

2.1. Management of diabetes mellitus

The treatment of diabetes involves a number of contemporary strategies. Nonetheless, reaching any goals established in the therapy of the condition depends on early detection.

2.1.1. Changes in lifestyle

An essential component of managing diabetes is changing one's lifestyle. Both individuals with pre-diabetes and those with diabetes are advised to take it. Among the suggested lifestyle changes are healthier meals, more physical activity, and a less sedentary lifestyle. The patient's condition may influence the appropriate workout. Exercise aids in lowering plasma glucose levels. Diabetic people should restrict meals rich in fat and sugar, prefer lean meats and nonfat dairy products, and consume a lot of fruits, vegetables, and whole grains for a balanced diet. Reducing alcohol consumption and quitting smoking are two more lifestyle adjustments.

2.1.2. Use of nanotechnology

Nanomedicine is the word used to describe the use of nanotechnology in medicine. The application of nanotechnology expertise to the use of medications or diagnostic chemicals, which often enhances their capacity to target certain cells or tissues, is known as nanomedicine. Through the use of innovative nanotechnology-based glucose testing and insulin administration methods, nanotechnology in diabetes research has improved the results of diabetic treatment in a number of ways.^{25,26} Innovative diabetes diagnostics, immune cell activity and beta-cell mass detection, glucose level monitoring, non-invasive insulin administration, and other applications are just a few of the ways that nanotechnology is crucial in the treatment of diabetes. Clinical therapies for the arrest of beta cell loss can be used immediately if the stage of the loss is promptly detected using nanotechnology. For example, magnetic nanoparticles are exceptional contrast media for magnetic resonance imaging due to their unique physical characteristics. This may make it possible to identify the stages of beta-cell loss early on.

Microcomputer closed-loop or nano pumps are being developed to enable the timely administration of insulin while maintaining continuous glucose monitoring in order to overcome the current delivery issues experienced by the conventional techniques. To put it another way, this system is designed to connect the supply of insulin to the level of plasma glucose.

2.1.3. Nutrition therapy for medical conditions

A licensed dietitian nutritionist administers medical nutrition therapy (MNT), a nutrition-based treatment. In order to help manage diabetes mellitus, it includes dietary diagnostics as well as expert counseling and therapy treatments. Since calorie restriction is essential for managing overweight or obesity, medical nutrition treatment is a crucial component of diabetes education and management. The treatment has had a major influence on patients, particularly women and babies, and has been shown to be essential in the treatment of different forms of diabetes mellitus. However, among other things, medical nutrition treatment has not yet determined the ideal diet for people with diabetes mellitus in terms of energy content, distribution, quality, and quantity of macronutrients.²⁷

2.1.4. Gene therapy

The process of restoring the symptoms of a disease caused by a faulty gene by introducing the external normal gene is known as gene therapy. Its benefit is that every condition may be cured with a single treatment, and gene therapy is currently creating new treatment choices across several medical specialties.²⁸ Currently, gene manipulation includes gene editing and regulation in addition to gene insertion.^{29,30} The insertion of a gene or gene alteration into a cell as a curative regimen in the treatment of illness is another way to define

gene therapy.³¹ The goal of this strategy is to correct faulty genes that have been identified as the cause of any disease and to effectively stop the disease's onset or progression.

Somatic gene therapy and germline gene therapy are two further subcategories of gene therapy. Germline gene therapy targets the reproductive cells, whereas somatic gene therapy primarily targets the somatic cells, also known as the sick cells. In following generations, germline treatment prevents the illness from progressing.³² Because gene therapies have the potential to cure a wide range of conditions that are challenging to treat with traditional medicines, such as diabetes mellitus, autoimmune disorders, heart illnesses, and malignancies, they are being used as trends in emerging therapeutics.³³ Many genes have been considered as a potential treatment for type 2 diabetes mellitus due to the disease's strong hereditary predisposition, even though gene therapy for the condition mostly targets type 1 diabetic mellitus.³⁴ Through genetic linkage research, over 75 distinct genetic loci have been shown to be responsible with type 2 diabetes mellitus, and many new treatment targets have also been identified. Unlike the frequency and development of illnesses with limited impacts, genetic loci may have a significant influence on treatment responsiveness.³⁵

2.1.5. Stem cell treatment

The current cellular-based therapy approach for managing diabetes mellitus relies on islet-cell transplantation or the pancreas to restore beta cells for the release of insulin. The scarcity of donor organs limits this strategy. These issues prompt research into the potential use of stem cells to create beta cells. Stem cells' unique capacity for reconstruction may make them a valuable tool for the treatment of diabetes mellitus. In addition to providing diabetes mellitus patients with a sustained supply of beta cells for insulin production, the development of a replenishable islet source employing stem cells may help prevent the current supply/demand issues in islet transplantation. Therefore, research on stem cells has emerged as a viable strategy for the treatment of diabetes mellitus.³⁶ The goal of stem cell treatment for diabetes mellitus is to use pluripotent or multipotent stem cells to replace damaged or dysfunctional pancreatic cells. The capacity of several types of stem cells, such as induced pluripotent stem cells (iPSCs), embryonic stem cells (ESCs), and adult stem cells, to generate surrogate beta cells or restore the physiologic function of the beta cell has been utilized in this approach in a variety of ways.³⁷

The generation of stem cells from several tissue sources, including adipose tissue, skin, bone marrow, umbilical cord blood, periosteum, and dental pulp, has been made easier by technological advancements. The pancreas is typically the first organ of choice when looking for potential stem cells. A tiny amount of pancreatic tissue, when made accessible, may restore the ideal mass of pancreatic beta-cells, according to research using animal models.³⁸ This is a result of the pancreatic duct's differentiated beta cells going through

dedifferentiation and replication, which creates pluripotent cells that produce additional beta cells. According to other research, these ductal cell populations may be generated in vitro and targeted to form clusters that synthesize insulin.^{39,40} Furthermore, mesenchymal stem cells (MSCs) and hemopoietic adult stem cells (HSCs) have the capacity to transdifferentiate into a wide variety of cell lineages, including those of the liver, brain, and lung in addition to gastrointestinal tract cells.^{41,42}

3. Conclusion

In addition to glycemic control, multifactorial interventions utilizing various treatment regimens, such as gene therapy, nanotechnology, stem cells, medical nutrition therapy, and lifestyle modification, have significantly reduced the effects of diabetes mellitus, albeit not without some difficulties. Although nanotechnology is a promising technology with the potential to improve diabetes care, there are still some obstacles to overcome. Diabetes mellitus has emerged as a public health concern that needs immediate attention, and the rising number of cases is expected to last for many more decades. Diabetes mellitus does not currently have a permanent cure. Numerous therapy plans have demonstrated encouraging outcomes in the management of diabetes mellitus. However, diabetes mellitus is still a significant problem that might endanger public health despite the promise of these massive treatment efforts. For a clinical management plan to be strong, effective, and safe, the issues with each of these techniques must be resolved. Optimal metabolic management of blood pressure, glucose, and body weight is required, necessitating appropriate education and support for dietary improvements, physical activity, and weight loss. An emphasis on public policies that support health care access and resources, the encouragement of a patient-centered care approach, and environmental infrastructures that promote health are necessary for the effective and successful management of this condition.

4. Source of Funding

None.

5. Conflict of Interest

None.

References

1. I.D.F. Diabetes Atlas 2021. Available from <https://diabetesatlas.org/> [accessed on 21st March 2025].
2. W.H.O. Global Report on Diabetes. W.H.O.; Geneva, Switzerland: 2016. Available online: <http://www.who.int/diabetes/global-report/en/> [accessed on 21st March 2025].
3. Wu Y, Ding Y, Tanaka Y, Zhang W. Risk Factors Contributing to Type 2 Diabetes and Recent Advances in the Treatment and Prevention. *Int J Med Sci.* 2014;11(11):1185–200. doi: 10.7150/ijms.10001.
4. Group T.D.P. Incidence and trends of childhood Type 1 diabetes worldwide 1990–1999. *Diabet Med.* 2006;23(8):857–66. doi: 10.1111/j.1464-5491.2006.01925.x.

5. Rask-Madsen C, King GL. Vascular complications of diabetes: Mechanisms of injury and protective factors. *Cell Metab.* 2013;17(1):20–33. doi: 10.1016/j.cmet.2012.11.012.
6. Diabetes Control and Complications Trial Research Group. Nathan DM, Genuth S, Lachin J, Cleary P, Crofford O, Davis M et al. The effect of Intensive Treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *N Engl J Med.* 1993;329(14):977–86. doi: 10.1056/NEJM199309303291401.
7. Callejas D, Mann CJ, Ayuso E, Lage R, Grifoll I, Roca C et al. Treatment of diabetes and long-term survival after insulin and glucokinase gene therapy. *Diabetes.* 2013;62(5):1718–29. doi: 10.2337/db12-1113.
8. Palmer SC, Mavridis D, Nicolucci A, Johnson DW, Tonelli M, Craig JC et al. Comparison of Clinical Outcomes and Adverse Events Associated With Glucose-Lowering Drugs in Patients With Type 2 Diabetes: A Meta-analysis. *JAMA.* 2016;316(3):313–24. doi: 10.1001/jama.2016.9400.
9. Defronzo R, Fleming GA, Chen K, Bicsak TA. Metformin-associated lactic acidosis: Current perspectives on causes and risk. *Metab Clin Exp.* 2016;2(65):20–29. doi: 10.1016/j.metabol.2015.10.014.
10. Banting FG, Best CH. Pancreatic extracts. *J Lab Clin Med.* 1990;115(2):254–72.
11. The Diabetes Control and Complications Trial Research Group. Hypoglycemia in the diabetes control and complications trial. *Diabetes.* 1997;46(2):271–86. doi: 10.2337/diab.46.2.271.
12. Weight gain associated with intensive therapy in the diabetes control and complications trial. The DCCT Research Group. *Diabetes Care.* 1988;11(7):567–73. doi: 10.2337/diacare.11.7.567.
13. Meek TH, Morton GJ. The role of leptin in diabetes: Metabolic effects. *Diabetologia.* 2016;59(5):928–32.
14. Cryer PE. Hypoglycemia, functional brain failure, and brain death. *J Clin Invest.* 2014;117(4):868–70.
15. Amiel SA, Aschner P, Childs B, Cryer PE, de Galan BE, Frier BM et al. Hypoglycemia, cardiovascular disease, and mortality in diabetes: epidemiology, pathogenesis, and management. *Lancet Diabetes Endocrinol.* 2019;7(5):385–96.
16. Corssmit EP, Romijn JA, Sauerwein HP. Regulation of glucose production with special attention to nonclassical regulatory mechanisms: A review. *Metabolism.* 2001;50(7):742–55. doi: 10.1053/meta.2001.24195.
17. Boden G. Gluconeogenesis and glycogenolysis in health and diabetes. *J Invest Med.* 2004;52(6):375–8.
18. Gerich JE, Meyer C, Woerle HJ, Stumvoll M. Renal gluconeogenesis: its importance in human glucose homeostasis. *Diabetes Care.* 2001;24(2):382–91.
19. Noh JW, Jung JH, Park JE, Lee JH, Sim KH, Park J et al. The relationship between age of onset and risk factors including family history and life style in Korean population with type 2 diabetes mellitus. *J Phys Ther Sci.* 2018;30(2):201–6. doi: 10.1589/jpts.30.201.
20. Asiiimwe D., Mauti G.O., Kiconco R. Prevalence and Risk Factors Associated with Type 2 Diabetes in Elderly Patients Aged 45–80 Years at Kanungu District. *J Diabetes Res.* 2020; 2020:5152146. doi: 10.1155/2020/5152146.
21. Mordarska K., Godziejewska-Zawada M. Diabetes in the elderly. *Prz. Menopauzalny.* 2017;16(2):38–43. doi: 10.5114/pm.2017.68589.
22. Ye J. Mechanisms of insulin resistance in obesity. *Front Med China.* 2013;7(1):14–24. doi: 10.1007/s11684-013-0262-6.
23. Hulkower RD, Pollack RM, Zonszein J. Understanding hypoglycemia in hospitalized patients. *Diabetes Manag (Lond).* 2014;4(2):165–76.
24. F.D.A. Cigarette Smoking: A Risk Factor for Type 2 Diabetes. 2020 Available online: [https://www.fda.gov/search?&Cigarette+Smoking%3A+A+Risk+](https://www.fda.gov/search?&Cigarette+Smoking%3A+A+Risk+Factor+for+Type+2+Diabetes&sort_bef_combine=rel_DESC)
25. Veisoh O, Tang BC, Whitehead KA, Anderson DG. Langer R. Managing diabetes with nanomedicine: Challenges and opportunities. *Nat Rev Drug Discov.* 2015;14(1):45–57. doi: 10.1038/nrd4477.
26. Disanto RM, Subramanian V, Gu Z. Recent advances in nanotechnology for diabetes treatment. *Wiley Interdiscip Rev Nanomed Nanobiotechnol.* 2015;7(4):548–64.
27. Moreno-Castilla C, Mauricio D, Hernandez M. Role of Medical Nutrition Therapy in the Management of Gestational Diabetes Mellitus. *Curr Diab Rep.* 2016 Apr;16(4):22. doi: 10.1007/s11892-016-0717-7.
28. Dunbar CE, High KA, Joung JK, Kohn DB, Ozawa K, Sadelain M. Gene therapy comes of age. *Science.* 2018;359(6372):eaan4672. doi: 10.1126/science.aan4672.
29. Xu R, Li H, Lai-yin T, Hsiang-fu K, Lu H, Lam K. Diabetes Gene Therapy: Potential and Challenges. *Curr Gene Ther.* 2003;3(1):65–82. doi: 10.2174/156652303347444.
30. Wong MS, Hawthorne WJ, Manolios N. Gene therapy in diabetes. *Self/Nonself.* 2010;1:165–75. doi: 10.4161/self.1.3.12643.
31. Mali S. Delivery systems for gene therapy. *Indian J Hum Genet.* 2013;19(1):3–8. doi: 10.4103/0971-6866.112870.
32. Kaufmann K.B., Büning H., Galy A., Schambach A., Grez M. Gene therapy on the move. *EMBO Mol Med.* 2013;5(11):1642–61. doi: 10.1002/emmm.201202287.
33. Tsokos GC, Nepom GT. Gene therapy in the treatment of autoimmune diseases. *J Clin. Investig.* 2000;106(2):181–3. doi: 10.1172/JCI10575.
34. Florez JC. Pharmacogenetics in type 2 diabetes: Precision medicine or discovery tool? *Diabetologia.* 2017;60(5):800–7. doi: 10.1007/s00125-017-4227-1.
35. McCall MD, Toso C, Baetge EE, Shapiro AMJ. Are stem cells a cure for diabetes? *Clin Sci.* 2009;118(2):87–97. doi: 10.1042/CS20090072.
36. Abdulazeez SS. Diabetes treatment: A rapid review of the current and future scope of stem cell research. *Saudi Pharm J.* 2015;23(4):333–40. doi: 10.1016/j.jsps.2013.12.012.
37. Bonner-Weir S, Baxter LA, Schuppin GT, Smith FE. A second pathway for regeneration of adult exocrine and endocrine pancreas: A possible recapitulation of embryonic development. *Diabetes.* 1993;42(12):1715–20. doi: 10.2337/diab.42.12.1715.
38. Bonner-Weir S, Taneja M, Weir GC, Tatarkiewicz K, Song KH, Sharma A et al. In vitro cultivation of human islets from expanded ductal tissue. *Proc Natl Acad Sci USA.* 2000;97(14):7999–8004. doi: 10.1073/pnas.97.14.7999.
39. Gao R, Ustinov J, Pulkkinen MA, Lundin K, Korsgren O, Otonkoski T. Characterization of endocrine progenitor cells and critical factors for their differentiation in human adult pancreatic cell culture. *Diabetes.* 2003;52(8):2007–15. doi: 10.2337/diabetes.52.8.2007.
40. Brazelton TR, Rossi FM, Keshet GI, Blau HM. From Marrow to Brain: Expression of Neuronal Phenotypes in Adult Mice Timothy. *Science.* 2000;290(5497):1775–9. doi: 10.1126/science.290.5497.1775.
41. Krause DS, Theise ND, Collector MI, Henegariu O, Hwang S, Gardner R et al. Multi-organ, multi-lineage engraftment by a single bone marrow-derived stem cell. *Cell.* 2001;105(3):369–77. doi: 10.1016/S0092-8674(01)00328-2.
42. Jiang Y, Jahagirdar BN, Reinhardt RL, Schwartz RE, Keene CD, Ortiz-Gonzalez XR et al. Pluripotency of mesenchymal stem cells derived from adult marrow. *Nature.* 2002;418(6893):41–9. doi: 10.1038/nature00870.

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