



INTERNATIONAL JOURNAL OF

PHARMACEUTICAL SCIENCES

[ISSN: 0975-4725; CODEN(USA): IJPS00]

Journal Homepage: <https://www.ijpsjournal.com>



Review Paper

Advancement in the Management of the Diabetes: A Comprehensive Review

Purushottam Kumar, Mamta Kadawal, Neha Yadav, Rupesh Kumar, Ankit Kumar, Kirti Yadav*

BM College of Pharmacy, Farrukhnagar, Gurugram.

ARTICLE INFO

Published: 06 Feb 2026

Keywords:

GLP-1 receptor agonists and SGLT2 inhibitors; Artificial Intelligence; regenerative medicine; ADA Standards of Care.

DOI:

10.5281/zenodo.18507335

ABSTRACT

Diabetes care has evolved beyond glucose monitoring to a patient-centered, cardiovascular and renal complications-focused approach. GLP-1 receptor agonists and SGLT2 inhibitors are now the standard of therapy for individuals with pre-existing cardiovascular disease, heart failure, or chronic kidney disease due to medicinal innovation. Dual-action treatments like tirzepatide have transformed hyperglycemia and obesity management. In parallel with drug advances, technological integration has reached a new peak. Continuous Glucose Monitoring (CGM) is now universally recommended for a wider demographic, including non-insulin-dependent Type 2 patients, and more precise Automated Insulin Delivery (AID) systems are now supported for diverse patient profiles. Technological integration attained "democratization" in 2025. CGM is currently recommended for most diabetics, including Type 2 non-insulin users. Through AI-driven algorithms and smartphone connectivity, improved Automated Insulin Delivery (AID) systems—or "artificial pancreas" technologies—can now treat Type 1 diabetes (T1D) more precisely and hands-off. In high-risk people, disease-modifying treatments like teplizumab may delay T1D development by years. Clinical advancements in regenerative medicine are imminent. Encapsulation and stem cell-derived islet transplants attempt to restore endogenous insulin production, possibly making some patients insulin-independent. Despite these advances, technology costs and global health inequities remain obstacles. To provide fair access to next-generation care, future management will use digital health platforms, telemedicine, and cost-effective domestic technologies. In the digital age, AI can accurately anticipate hypoglycemia and diabetic ketoacidosis. Fully integrated telemedicine solutions provide real-time data exchange and virtual coaching for underprivileged groups. Research on stem cell therapy and β -cell replacement in regenerative medicine gives promise for long-term remission in Type 1 Diabetes by restoring insulin production. Beyond clinical intervention, the 2025 ADA Standards of

***Corresponding Author:** Kirti Yadav

Address: Assistant Professor, BM College of Pharmacy, Farrukhnagar, Gurugram.

Email✉: kirtiyadav4498@gmail.com

Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.



Care elevate holistic treatment, bringing sleep health—ideally 6–9 hours each night—on level with food and exercise. These advances, however, are hindered by expensive technology costs. Thus, legislative change, greater insurance coverage, and the discovery of cost-effective indigenous medicines are needed to democratize diabetes care and make life-saving advances available to all socioeconomic groups.

INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic condition that causes hyperglycemia due to insulin secretion or action abnormalities. The International Diabetes Federation (IDF) predicts that 589 million adults—1 in 9—have the illness in 2025. Type 2 Diabetes (T2DM), which accounts for over 90% of cases, is connected to obesity, sedentary lifestyles, and an aging global population. Nearly 4 in 10 persons are ignorant of their illness, putting them at risk for life-threatening microvascular and macrovascular consequences such renal failure, blindness, and cardiovascular disease (3, 4). In 2025, diabetes treatment has shifted from "glucocentric" to "organ-protective". Traditional methods concentrated on decreasing blood glucose and HbA1c, while new clinical standards protect against cardiorenal consequences. Regardless of baseline glucose, ADA 2025 recommendations recommend early use of SGLT2 inhibitors and GLP-1 receptor agonists to protect the heart and kidneys (5, 6). Advanced technologies like Continuous Glucose Monitoring (CGM) and Automated Insulin Delivery (AID) systems support this holistic framework, as does a stronger focus on "Positive Health Behaviors" like standardized sleep health (6–9 hours per night) and resistance training (7, 8). Selection and use of a glucose-lowering treatment rely on severity of hyperglycemia, hepatic and renal functions, risks of hypoglycemia, body mass index, capacity to self-monitor blood glucose, and prescription cost. Type 1 diabetes treatments include GLP analogues

like Exenatide and Liraglutide [8, 9], insulin injections for β cell defects, DPP-4 inhibition with Sitagliptin [10, 11], and INGAP peptide therapy for islet cell regeneration [12].

2. Classification of Diabetes

Diabetes is classified into two types: Type 1 diabetes (Insulin dependent diabetes) and Type 2 diabetes (Non-Insulin dependent diabetes) [13].

2.1 Insulin Dependent Diabetes Mellitus (IDDM)

Type 1 diabetes, which arises when the immune system targets and kills pancreatic cells, affects 5–10% of the population and has several names. Also known as juvenile onset diabetes. Most instances involve younger individuals, although anybody may get it. Maintaining blood glucose levels requires regular insulin injections. While infants and children have fast degradation of β cells, adults experience progressive degeneration. Minor fasting hyperglycemia may lead to severe hyperglycemia or ketoacidosis in response to stress or illness, although adolescents and young adults are more likely to develop it [22]. These individuals are more likely to develop celiac, autoimmune hepatitis, myasthenia gravis, Hashimoto's thyroiditis, Addison's, pernicious anemia, and Grave's illnesses [22]. Hereditary diabetes disproportionately affects African and Asian populations [23].

2.2 Idiopathic Diabetes

Few people with type 1 diabetes have no known cause; these people tend to be of Asian or African descent. The risk of ketoacidosis is high, and they have insulinopenia that never goes away. Ketoacidosis occurs in episodes, and the severity of insulin shortage varies from episode to episode. Insulin replacement therapy is essential for

patients with idiopathic diabetes, a disorder that has a hereditary component [22].

2.3 Noninsulin Dependent Diabetes Mellitus (NIDDM)

This form of diabetes, which comprises 90–95% of all cases, is also known as adult onset diabetes. There has been a type 2 diabetes epidemic due to major metabolic disorders such as obesity, insulin resistance, and dyslipidaemia [24]. In this form of diabetes, oral hypoglycemic medications and dietary changes are the mainstays of treatment. The development of illness is facilitated by insulin resistance and decreased insulin production. With a twofold excess mortality and a two- to fourfold greater risk of coronary heart disease and stroke, type 2 diabetes mellitus—the most common form of the disease—is the fourth biggest cause of death in industrialized nations.

2.4 Gestational Diabetes

Pregnancy is associated with different levels of glucose intolerance, which may result in hyperglycemia of different intensities [26]. Impaired glucose intolerance is a major form of gestational diabetes mellitus (GDM) that is first identified during pregnancy [27]. It raises the risk of type 2 diabetes in mothers and affects 14% of pregnant women, or 135,000 women in the US each year [28]. The reported risk may vary in size due to variations in type 2 diabetes and GDM tests, ethnicity, and selection criteria [29]. Gestational diabetes may cause the fetus to have macrosomia, the infant to have low blood sugar, and respiratory distress syndrome. Newborns are more likely to have birth problems such as shoulder dystocia, traumatic brain damage, and cesarean sections. According to new recommendations, maintaining appropriate glycemic control may help reduce these effects for both mother and child. The majority of women with gestational

diabetes benefit from diet and exercise, but some may need insulin or oral diabetic medication.

2.5 Catamenial Hyperglycaemia

Diabetic ketoacidosis (DKA) may be caused by a number of conditions, including as infections, inadequate insulin or poor insulin adherence, acute pancreatitis, stroke, medicines, anomalies in the body's metabolism, or therapeutic negligence [30]. Women who have uncontrolled hyperglycemia with diabetic ketoacidosis before to their menstrual cycle are said to have catamenial diabetic ketoacidosis, also known as catamenial hyperglycemia. Uncontrolled hyperglycemia resulted in insulin requirements that were up to four times greater. The situation deteriorates despite ongoing insulin infusion, leading to hyperglycemia, vomiting, severe acidosis, and ketonuria. It seemed strange that every test, including those for thyroid function, renal function, ECG, chest radiographs, blood and urine cultures, and inflammatory markers, came back normal. The reason of catamenial hyperglycemia is currently unknown [31]. Hormonal changes throughout the menstrual cycle and differences in diet and activity intake are potential contributing factors [32]. In addition to increasing the amount of insulin injections, diabetic ketoacidosis and other diabetic crises should be treated with a nutritious diet and frequent exercise [32]. An infusion dose would be the appropriate medication approach to treat diabetic ketoacidosis and avoid diabetic crises [32].

3. Advancement in the Management of the Diabetes

3.1 Use of the Nanotechnology in Diabetes

Innovative techniques for insulin delivery and glucose testing have resulted from the use of nanotechnology in diabetes treatment. Researchers have shown that closed-loop insulin delivery

systems and glucose monitors may aid in the treatment of both type 1 and type 2 diabetes. A microcapsule containing holes that has shown potential for medication delivery is known as a nanomedical device. These holes are tiny enough to allow larger immune system chemicals like immunoglobulins and virus particles to pass through, but they are large enough for smaller molecules like oxygen, glucose, and insulin. Diabetes patients may get subcutaneous implants of microcapsules containing replacement islets of Langerhans cells, mostly derived from pigs. The body's delicate glucose management feedback loop may be momentarily restored without the need for strong immunosuppressants, which might increase the patient's risk of infection. [35] Table 2 lists the major problems associated with diabetes as well as how nanomedicine might help cure it. By focusing on certain tissues, organs, and tumors, the nanoparticle-targeted drug delivery technique improves the bioavailability of pharmaceuticals and ensures that the maximum quantity of medicine reaches the intended location. The difficulty of increasing the size of nanoparticles is one of the most challenging aspects in technology. Since there are now no established techniques for creating three-dimensional nanostructures, the process is more difficult than creating two-dimensional layer-shaped nanosurfaces. The possibility that being near nanoparticles might be harmful to your health is another concern. Concerns concerning the potential negative consequences of produced nanomaterials, such as carbon buckyballs and nanotubes, when inhaled, consumed, or absorbed via the skin are growing [35]. Insulin is an essential need for those with severe type 1 and type 2 diabetes. Infections, uncomfortable administration, and poor patient compliance have all been linked to traditional insulin delivery methods. By controlling insulin distribution using pulmonary, nasal, transdermal, and closed-loop devices, recent developments in

micro- and nanotechnologies have simplified the insulin administration process [36].

3.2 Stem Cell Technology

Due to the underlying causes of both type 1 and type 2 diabetes, which are defined by defects in pancreatic β cells that result in inadequate insulin production, research into prospective diabetes therapeutics has increasingly centered on stem cell technologies. Techniques are designed to increase insulin sensitivity or correct these cells' malfunction [37]. Although β cell replacement techniques provide novel resources, the lack of donors limits existing methods such as islet cell and pancreas transplants. Insulin resistance and β cell malfunction are associated with type 2 diabetes, while autoimmune loss of β cells causes type 1 diabetes [38, 39]. Because mesenchymal stem cells (MSCs) have immunosuppressive qualities and may effectively modify immune responses both *in vitro* and *in vivo*, MSC therapy has become a viable treatment for type 1 diabetes. Additionally, hematopoietic stem cells, which have the ability to develop into many blood cell types and display immunomodulatory effects, may potentially help newly diagnosed type 1 diabetics by enhancing β cell activity. Furthermore, adult fibroblasts from patients with type 1 diabetes may develop into insulin-producing cells using induced pluripotent stem (iPS) cells, which offers benefits for cellular replacement therapy and disease models [40, 41]. According to some research, MSCs produced from bone marrow may also develop into cells that produce insulin. Although human embryonic stem cells (ESCs) have attracted a lot of interest due to their pluripotent benefits, they also have drawbacks, including the production of certain cell types, immunological rejection, and unchecked growth after transplantation. Stem cell technology has great potential for treating diabetes, despite several ethical and scientific obstacles [42, 43].



3.3 Statin Therapy

Low-density lipoprotein (LDL) cholesterol is effectively reduced by statin medication, which considerably lowers the risk of coronary heart disease. The Scottish Intercollegiate Guidelines Network (SIGN) and the National Institute for Health and Clinical Excellence (NICE) both recommend lipid-lowering therapy as primary prevention for patients over 40 with type 2 diabetes (Grade A recommendation), while those over 40 with type 1 diabetes (Grade B recommendation) [44, 45]. Despite these recommendations, new research from a European diabetes conference shows that a large American cohort of more than 100,000 people with type 2 diabetes underutilize statin medication. Statins have drawbacks despite their effectiveness in reducing cardiovascular events in those with moderate cholesterol levels and no pre-existing cardiovascular disease [46, 47]. Notably, uncommon hepatic dysfunction, muscular problems (from myositis to rhabdomyolysis), and renal failure are among the possible adverse effects. Younger people and those without illness had poor statin drug compliance, according to a study with 6,422 participants. Therefore, elderly patients should be the main target of the treatment, especially those with significant risk factors for cardiovascular problems [48, 49]. Furthermore, while typically well-tolerated, statin therapy may nevertheless cause problems such as myopathies and raised liver enzymes in those with type 2 diabetes [50]. It has also been observed that statin use may modestly boost blood sugar levels, possibly leading to diabetes mellitus.

3.4 Use of the Natural Products in Diabetes

The use of herbal medicines to treat diabetes, both insulin-dependent and non-insulin-dependent, has long been noted in the literature. Plants having antidiabetic qualities have historically been seen as

sources of novel hypoglycemic chemicals or as possible supplements to current treatments. Ayurvedic books such as the Sushruta Samhita, which was written in the fourth and fifth centuries BC, mention diabetes and its acknowledgment goes back to the Brahmic period. Two forms of diabetes are discussed in these texts: one that results from dietary errors and the other that is inherited [51, 52]. Due to their affordability and very low side effects, herbal medicines have become quite popular. Even while plant-based therapies have been effectively used in traditional medicine around the world, many of these herbs' underlying processes are still unclear and inconsistent. Recent research has shown that plant-based bioactive chemicals have antidiabetic effects that are on par with or even stronger than those of well-known oral hypoglycemic medications like daonil, tolbutamide, and chlorpropamide. Nevertheless, many plant-based active chemicals remain poorly described [53, 54]. The ethnobotanical community is very interested in plants with antidiabetic qualities because of their important medicinal components and varying hypoglycemic activity, according to research, including those done by Grover et al. [55, 56]. It is possible to separate and use the bioactive components of different plant species as pharmacological agents or drug lead compounds, which may provide natural remedies for the difficulties associated with diabetes [57, 58]. A phytomolecule's antidiabetic effectiveness is greatly influenced by its chemical structure. According to Jung et al., a number of plant species are rich in terpenoids, flavonoids, phenolics, and coumarins that have been shown to be effective in decreasing blood glucose levels. In addition to *Murraya koenigii* (L.) Spreng. (Rutaceae), *Allium sativum* Linn. (Liliaceae), *Gymnema sylvestre* (Retz.) Schult (Asclepiadaceae), *Allium cepa* (Liliaceae), *Withania somnifera* Dunal (Solanaceae), and *Ferula foetida* Linn. In



experimental diabetes models, (*Umbelliferae*) have shown encouraging antidiabetic qualities. The significance of *G. sylvestre* in the management and treatment of diabetes has been well investigated [59, 60].

CONCLUSION

After a comprehensive search across many databases, this review article outlines the significant findings and developments in our understanding and management of DR during the last few years. They covered the epidemiology, pathophysiology, systemic risk factors, screening methods, and treatment strategies of DR in great detail. The epidemiological data presented shows a concerning rise in diabetes incidence globally, with a particular emphasis on the Caribbean, Africa, the Middle East, and North America. Among other demographic disparities, the frequency of DR varies significantly by gender and diabetes. DR puts one's vision at risk in addition to being a possible predictor of overall health problems. The pathophysiology section explains the intricate mechanisms of DR, with hyperglycemia playing a major role in the breakdown of retinal microvascular tissue. Vascular damage brought on by hyperglycemia may alter blood flow, harm the blood-retina barrier, thicken the basement membrane, kill endothelial cells, and more. The metabolic processes connected to this damage are investigated in this work. The research also discusses novel biomarkers that might be helpful for DR diagnosis and prognosis. These consist of advanced glycation products, C-reactive protein, and neuroglobin. The main factors that promote DR neovascularization are VEGF and pro-angiogenic cytokines. The effects of glycemic control, medication usage, diabetes duration, hypertension, obesity, and hyperlipidemia on DR are thoroughly examined. The review emphasizes how crucial it is to manage these risk factors in

order to halt DR in its tracks. The authors provided insight into current DR screening standards and practices by compared opportunistic and systematic screening techniques. They also examine the many screening techniques, including telemedicine-based possibilities, emphasizing the cost-effectiveness and potential to improve access to treatment. The section on treatment techniques explores the several options for treating DR, including as surgery, laser photocoagulation, and medication. Important treatments for diabetic macular oedema include intravitreal glucocorticoids and antiVEGF medications, however laser photocoagulation remains the most successful treatment option. Pars plana vitrectomy can be a possibility if existing therapies are unable to halt the vitreous hemorrhage or if the retina separates. In conclusion, this review study provides an excellent summary of the state of knowledge on DR. It emphasizes the need of risk factor management, early screening, and novel treatment approaches. The article emphasizes the need for ongoing research and innovation to counteract the growing global effect of DR and its significant impact on public health.

CONFLICT OF INTEREST

None

REFERENCES

1. S. R. Joshi, "Metabolic syndrome—emerging clusters of the Indian phenotype," *Journal of Association of Physicians of India*, vol. 51, pp. 445–446, 2003
2. R. Deepa, S. Sandeep, V. Mohan et al., "Abdominal obesity, visceral fat and type 2 diabetes—Asian Indian phenotype," in *Type 2 Diabetes in South Asians: Epidemiology, Risk Factors and Prevention*, V. Mohan and G. H. R. Rao, Eds., pp. 138–152, Jaypee Brothers Medical Publishers, New Delhi, India, 2006



3. M. Chandalia, N. Abate, A. Garg, J. Stray-Gundersen, and S.M. Grundy, "Relationship between generalized and upper bodyobesity to insulin resistance in Asian Indian men," *The Journal of Clinical Endocrinology & Metabolism*, vol. 84, no. 7, pp. 2329–2335, 2009
4. Ramachandran, C. Snehalatha, V. Viswanathan, M.Viswanathan, and S. M. Haffner, "Risk of non insulin dependentdiabetes mellitus conferred by obesity and central adiposity indifferent ethnic groups: a comparative analysis between Asian Indians, Mexican Americans and Whites," *Diabetes Research and Clinical Practice*, vol. 36, no. 2, pp. 121–125, 2007
5. J. B. Buse, J. Rosenstock, G. Sesti et al., "Liraglutide once a day versus exenatide twice a day for type 2 diabetes: a 26-week randomised, parallel-group, multinational, open-label trial (LEAD-6)," *The Lancet*, vol. 374, no. 9683, pp. 39–47, 2009
6. K. F. Croom and P. L. McCormack, "Liraglutide: a review of its use in type 2 diabetes mellitus," *Drugs*, vol. 69, no. 14, pp. 1985–2004, 2009
7. S.-J. Kim, C. Nian, D. J. Doudet, and C. H. S. McIntosh, "Inhibition of dipeptidyl peptidase IV with sitagliptin (MK0431) prolongs islet graft survival in streptozotocin-induced diabetic mice," *Diabetes*, vol. 57, no. 5, pp. 1331–1339, 2008
8. S.-J. Kim, C. Nian, D. J. Doudet, and C. H. S. McIntosh, "Dipeptidyl peptidase IV inhibition with MK0431 improves islet graft survival in diabetic NOD mice partially via T-cell modulation," *Diabetes*, vol. 58, no. 3, pp. 641–651, 2009
9. R. Rafaeloff, G. L. Pittenger, S. W. Barlow et al., "Cloning and sequencing of the pancreatic islet neogenesis associated protein (INGAP) gene and its expression in islet neogenesis in hamsters," *The Journal of Clinical Investigation*, vol. 99, no. 9, pp. 2100–2109, 1997
10. J. B. Buse, "Overview of current therapeutic options in type 2 diabetes. Rationale for combining oral agents with insulin therapy," *Diabetes Care*, vol. 22, pp. 65–70, 1999
11. M. C. Riddle, "Combined therapy with a sulfonylurea plus evening insulin: safe, reliable, and becoming routine," *Hormone and Metabolic Research*, vol. 28, no. 9, pp. 430–433, 1996
12. A. Golay, N. Guillet-Dauphine, A. Fendel, C. Juge, and J. P. Assal, "The insulin-sparing effect of metformin in insulin-treated diabetic patients," *Diabetes/Metabolism Reviews*, vol. 11, no. 1, pp. S63–S67, 1995
13. J. B. Buse, B. Gumbiner, N. P. Mathias, D. M. Nelson, B. W. Faja, and R. W. Whitcomb, "For the Troglitazone study group: troglitazone use in insulin-treated type 2 diabetic patients," *Diabetes Care*, vol. 21, no. 9, pp. 1455–1461, 1998
14. OMIM—Online Mendelian Inheritance in Man, <http://www.ncbi.nlm.nih.gov/omim>
15. V. Mohan, S. Shah, and B. Saboo, "Current glycemic status and diabetes related complications among type 2 diabetes patients in India: data from the A1chieve study," *The Journal of the Indian Association of Physicians of India*, vol. 61, supplement, pp. 12–15, 2013
16. S. E. Inzucchi, "Oral antihyperglycemic therapy for type 2 diabetes," *The Journal of the American Medical Association*, vol. 287, no. 3, pp. 360–372, 2002
17. R. Chakrabarti and R. Rajagopalan, "Diabetes and insulin resistance associated disorders: disease and the therapy," *Current Science*, vol. 83, no. 12, pp. 1533–1538, 2002
18. J. B. Buse, R. R. Henry, J. Han, D. D. Kim, M. S. Fineman, and A. D. Baron, "Effects of

exenatide (exendin-4) on glycemic control over 30 weeks in sulfonylurea-treated patients with type 2 diabetes,” *Diabetes Care*, vol. 27, no. 11, pp. 2628–2635, 2004

19. American Diabetes Association, “Diagnosis and classification of diabetes mellitus,” *Diabetes Care*, vol. 33, supplement 1, pp. S62–S69, 2010

20. V. S. Reddy, R. K. Sahay, and S. K. Bhadada, “Newer oral antidiabetic agents,” *Journal of Indian Academy of Clinical Medicine*, vol. 1, pp. 245–251, 2000

21. D. E. Moller, “New drug targets for type 2 diabetes and the metabolic syndrome,” *Nature*, vol. 414, no. 6865, pp. 821–827, 2001

22. J. McKinlay and L. Marceau, “US public health and the 21st century: diabetes mellitus,” *The Lancet*, vol. 356, no. 9231, pp. 757–761, 2000

23. Definition, Diagnosis and Classification of Diabetes Mellitus and Its Complications, World Health Organization, Department of Non Communicable Disease Surveillance, Geneva, Switzerland, 1999

24. American Diabetes Association, “Gestational diabetes mellitus,” *Diabetes Care*, vol. 23, pp. 77–79, 2000

25. L. Jovanovic and D. J. Pettitt, “Gestational diabetes mellitus,” *The Journal of the American Medical Association*, vol. 286, no. 20, pp. 2516–2518, 2001

26. D. Coustan, “Gestational diabetes,” in *Diabetes in America*, M. I. Harris, C. C. Cowie, M. P. Stern, E. J. Boyko, G. E. Reiber, and P. H. Bennett, Eds., pp. 703–717, U.S. Government Printing Office, Washington, DC, USA, 2nd edition, 1995

27. H. M. Kronenberg, S. Melmed, K. S. Polonsky, and P. R. Larsen, *Williams Textbook of Endocrinology*, Saunders, Philadelphia, PA, USA, 11th edition, 2008

28. F. Ovalle, T. B. Vaughan III, J. E. Sohn, and B. Gower, “Catamenial diabetic ketoacidosis and catamenial hyperglycemia: case report and review of the literature,” *The American Journal of the Medical Sciences*, vol. 335, no. 4, pp. 298–303, 2008

29. K. K. Trout and G. Scheiner, “Blood glucose control and the menstrual cycle,” *Endocrinology Reviews*, pp. 27–29, 2008

30. National Institute for Clinical Excellence, *Technology Appraisal Guidance 60. Guidance on the Use of Patient Education Models for Diabetes*, NICE, 2003

31. R. M. DiSanto, V. Subramanian, and Z. Gu, “Recent advances in nanotechnology for diabetes treatment,” *Wiley Interdisciplinary Reviews: Nanomedicine and Nanobiotechnology*, 2015

32. J. Dhutia, “Research paper based on pathology lectures at Medlink 2007 and Vet-Medlink,” 2007

33. R. Mo, T. Jiang, J. Di, W. Tai, and Z. Gu, “Emerging micro- and nanotechnology based synthetic approaches for insulin delivery,” *Chemical Society Reviews*, vol. 43, no. 10, pp. 3595–3629, 2014

34. R. V. Shah and A. B. Goldfine, “Statins and risk of new-onset diabetes mellitus,” *Circulation*, vol. 126, no. 18, pp. e282–e284, 2012

35. P. Gæde, P. Vedel, N. Larsen, G. V. H. Jensen, H.-H. Parving, and O. Pedersen, “Multifactorial intervention and cardiovascular disease in patients with type 2 diabetes,” *The New England Journal of Medicine*, vol. 348, no. 5, pp. 383–393, 2003

36. Y.-H. Chen, B. Feng, and Z.-W. Chen, “Statins for primary prevention of cardiovascular and cerebrovascular events in diabetic patients without established cardiovascular diseases: a meta-analysis,” *Experimental and Clinical Endocrinology*



and Diabetes, vol. 120, no. 2, pp. 116–120, 2012

37. J. Buse, “Statin treatment in diabetes mellitus,” Clinical Diabetes, vol. 21, no. 4, pp. 168–172, 2003

38. R. S. Drummond, M. J. Lyall, and J. A. McKnight, “Statins should be routinely prescribed in all adults with diabetes,” Practical Diabetes International, vol. 27, no. 9, pp. 404–406, 2010

39. L. Radican and T. Seck, “Underutilisation of statins in patients with type 2 diabetes treated with an antihyperglycaemic regimen,” in Proceedings of the 46th Annual Meeting of the European Association for the Study of Diabetes (EASD ’10), Abstract 1302, Stockholm, Sweden, 2010

40. J. W. Jukema, C. P. Cannon, A. J. M. De Craen, R. G. J. Westendorp, and S. Trompet, “The controversies of statin therapy: weighing the evidence,” Journal of the American College of Cardiology, vol. 60, no. 10, pp. 875–881, 2012

41. J. J. Meier, A. Bhushan, and P. C. Butler, “The potential for stem cell therapy in diabetes,” Pediatric Research, vol. 59, no. 4, pp. 65R–73R, 2006

42. E. Butler, J. Janson, S. Bonner-Weir, R. Ritzel, R. A. Rizza, and P. C. Butler, “ β -cell deficit and increased β -cell apoptosis in humans with type 2 diabetes,” Diabetes, vol. 52, no. 1, pp. 102–110, 2003

43. R. Abdi, P. Fiorina, C. N. Adra, M. Atkinson, and M. H. Sayegh, “Immunomodulation by mesenchymal stem cells: a potential therapeutic strategy for type 1 diabetes,” Diabetes, vol. 57, no. 7, pp. 1759–1767, 2008

44. G. M. Spaggiari, H. Abdelrazik, F. Becchetti, and L. Moretta, “MSCs inhibit monocyte-derived DC maturation and function by selectively interfering with the generation of immature DCs: central role of MSC-derived prostaglandin E2,” Blood, vol. 113, no. 26, pp. 6576–6583, 2009

45. J. Wu, Z. Sun, H. S. Sun et al., “Intravenously administered bone marrow cells migrate to damaged brain tissue and improve neural function in ischemic rats,” Cell Transplantation, vol. 16, no. 10, pp. 993–1005, 2008

46. Tyndall, U. A. Walker, A. Cope et al., “Immunomodulatory properties of mesenchymal stem cells: a review based on an interdisciplinary meeting held at the Kennedy Institute of Rheumatology Division, London, UK, 31 October 2005,” Arthritis Research & Therapy, vol. 9, article 301, 2007

47. J. C. Voltarelli, C. E. B. Couri, A. B. P. L. Stracieri et al., “Autologous nonmyeloablative hematopoietic stem cell transplantation in newly diagnosed type 1 diabetes mellitus,” The Journal of the American Medical Association, vol. 297, no. 14, pp. 1568–1576, 2007

48. R. Maehr, S. Chen, M. Snitow et al., “Generation of pluripotent stem cells from patients with type 1 diabetes,” Proceedings of the National Academy of Sciences of the United States of America, vol. 106, no. 37, pp. 15768–15773, 2009

49. D.-Q. Tang, L.-Z. Cao, B. R. Burkhardt et al., “In vivo and invitro characterization of insulin-producing cells obtained from murine bone marrow,” Diabetes, vol. 53, no. 7, pp. 1721–1732, 2004

50. Ianus, G. G. Holz, N. D. Theise, and M. A. Hussain, “In vivo derivation of glucose-competent pancreatic endocrine cells from bone marrow without evidence of cell fusion,” The Journal of Clinical Investigation, vol. 111, no. 6, pp. 843–850, 2003

51. S. Ghannam, C. Bouffi, F. Djouad, C. Jorgensen, and D.

Noël, "Immunosuppression by mesenchymal stem cells: mechanisms and clinical applications," *Journal of Stem Cell Research and Therapy*, vol. 1, no. 1, article 2, 2010

52. K. R. Chien, A. Moretti, and K.-L. Laugwitz, "Development.ES cells to the rescue," *Science*, vol. 306, no. 5694, pp. 239–240, 2004

53. Y. Hori, "Insulin-producing cells derived from stem/progenitor cells: therapeutic implications for diabetes mellitus," *Medical Molecular Morphology*, vol. 42, no. 4, pp. 195–200, 2009

54. L. Chan, M. Fujimiya, and H. Kojimaa, "In vivo gene therapy for diabetes mellitus," *Trends in Molecular Medicine*, vol. 9, no. 10, pp. 430–435, 2003.[58] M. Zalzman, S. Gupta, R. K. Giri et al., "Reversal of hyperglycemia in mice by using human expandable insulin-producing cells differentiated from fetal liver progenitor cells," *Proceedings of the National Academy of Sciences of the United States of America*, vol. 100, no. 12, pp. 7253–7258, 2003

55. H.-S. Jun and J.-W. Yoon, "Approaches for the cure of type 1 diabetes by cellular and gene therapy," *Current Gene Therapy*, vol. 5, no. 2, pp. 249–262, 2005

56. N. Morral, "Novel targets and therapeutic strategies for type 2 diabetes," *Trends in Endocrinology and Metabolism*, vol. 14, no. 4, pp. 169–175, 2003

57. N. Morral, R. McEvoy, H. Dong et al., "Adenovirus-mediated expression of glucokinase in the liver as an adjuvant treatment for type 1 diabetes," *Human Gene Therapy*, vol. 13, no. 13, pp. 1561–1570, 2002

58. R. M. O'Doherty, P. B. Jensen, P. Anderson et al., "Activation of direct and indirect pathways of glycogen synthesis by hepatic overexpression of protein targeting to glycogen," *The Journal of Clinical Investigation*, vol. 105, no. 4, pp. 479–488, 2000

59. C. B. Newgard, M. J. Brady, R. M. O'Doherty, and A. R. Saltiel, "Organizing glucose disposal: emerging roles of the glycogen targeting subunits of protein phosphatase-1," *Diabetes*, vol. 49, no. 12, pp. 1967–1977, 2000

60. H. Dong and S. L. C. Woo, "Hepatic insulin production for type 1 diabetes," *Trends in Endocrinology and Metabolism*, vol. 12, no. 10, pp. 441–446, 2001.[66] J. W. Yoon and H. S. Jun, "Recent advances in insulin gene therapy for type 1 diabetes," *Trends in Molecular Medicine*, vol. 8, no. 2, pp. 62–68, 2002

HOW TO CITE: Purushottam Kumar, Mamta Kadawal, Neha Yadav, Rupesh Kumar, Ankit Kumar, Kirti Yadav Advancement in the Management of the Diabetes: A Comprehensive Review Int. J. of Pharm. Sci., 2026, Vol 4, Issue 2, 873-882. <https://doi.org/10.5281/zenodo.18507335>