JOURNAL OF PHARMA INSIGHTS AND RESEARCH ISSN NO. 3048-5428

REVIEW ARTICLE

Role of Oral PCSK9 Inhibitors in Lipid Management

Edward Raju Gope[1], Durga Bhavani Puppala*[2], Navya Andhugula[2], Naga Navya Donga[2], Durga Prasad Madu[2], Raghava Doonaboyina[3], Nageswara Rao Kavala[4]

[1] *Assistant Professor, Department of Pharmaceutical Analysis, KGRL College of Pharmacy, Bhimavaram, Andhra Pradesh, India*
[2] *UG Scholar, Department of Pharmaceutical Analysis, KGRL College of Pharmacy, Bhimavaram, Andhra Pradesh, India*
[3] *Principal and Professor, Department of Pharmaceutical Chemistry, KGRL College of Pharmacy, Bhimavaram, Andhra Pradesh, India*
[4] *Director and Professor, Department of Pharmaceutical Analysis, KGRL College of Pharmacy, Bhimavaram, Andhra Pradesh, India*

Publication history: Received on 16th October 2025; Revised on 31st October 2025; Accepted on 5th November 2025

Article DOI: 10.69613/0zjp0634

Abstract: Cardiovascular diseases (CVDs) remain the leading cause of global mortality, with elevated low-density lipoprotein cholesterol (LDL-C) identified as a primary modifiable risk factor. While statins serve as the foundational therapy for lipid management, a significant proportion of patients fail to achieve recommended LDL-C targets due to intolerance or insufficient efficacy. The introduction of proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors marked a significant advancement; however, the initial monoclonal antibody formulations require parenteral administration, limiting broad patient acceptance and accessibility. This review discusses about the emerging class of oral PCSK9 inhibitors, which aim to combine the potent efficacy of biologic agents with the convenience of oral dosing. The pharmacological mechanisms of novel small-molecule inhibitors and macrocyclic peptides, especially recent clinical data for candidates such as MK-0616 and AZD0780 are detailed in this review. Phase 2 clinical trials indicate that these oral agents can achieve LDL-C reductions comparable to injectable formulations, with safety profiles similar to placebo. Moreover, the challenges of oral bioavailability, the potential for synergistic therapy with statins, and the implications for global cardiovascular health were also discussed in this paper. The successful development of oral PCSK9 inhibitors represents a transformative step in preventative cardiology, potentially enhancing adherence and reducing the burden of atherosclerotic cardiovascular disease.

Keywords: Oral PCSK9 Inhibitors; Low-Density Lipoprotein Cholesterol; Atherosclerosis; MK-0616; AZD0780

1. Introduction

Atherosclerotic cardiovascular disease (ASCVD) remains the preeminent cause of morbidity and mortality worldwide, presenting a formidable challenge to public health systems. Central to the pathogenesis of ASCVD is the dysregulation of lipid metabolism, specifically the retention of low-density lipoprotein cholesterol (LDL-C) within the arterial intima. This accumulation precipitates an inflammatory response, leading to plaque formation, progression, and ultimately, rupture, which manifests clinically as myocardial infarction or ischemic stroke [1]. Consequently, the reduction of circulating LDL-C has become the primary surrogate endpoint and therapeutic target in preventative cardiology, with extensive evidence showing a linear relationship between absolute LDL-C reduction and decreased cardiovascular risk [2].

For the past three decades, 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors, or statins, have served as the pharmacologic cornerstone for lipid management. Their ability to inhibit cholesterol biosynthesis and upregulate hepatic LDL receptors has saved millions of lives [3]. However, despite their ubiquity and proven efficacy, a significant "residual risk" persists. Epidemiological data suggest that a substantial proportion of high-risk patients do not achieve guideline-recommended LDL-C goals. This treatment gap is driven by two primary factors: the insufficient potency of statin monotherapy in patients with severe hypercholesterolemia (such as Familial Hypercholesterolemia) and the prevalence of statin-associated muscle symptoms (SAMS), which leads to discontinuation or dose-limitation in approximately 10-15% of patients [4].

The identification of proprotein convertase subtilisin/kexin type 9 (PCSK9) as a key regulator of hepatic LDL receptor degradation opened a new frontier in lipidology. PCSK9 binds to LDL receptors on the hepatocyte surface, promoting their lysosomal degradation and preventing their recycling to the cell surface. The subsequent development of monoclonal antibodies (mAbs) targeting PCSK9, namely evolocumab and alirocumab, demonstrated that inhibiting this protein could lower LDL-C by an additional 50–60% on top of statin therapy [5]. Large-scale cardiovascular outcomes trials confirmed that these agents significantly reduce major adverse cardiovascular events (MACE).

* Corresponding author: Durga Bhavani Puppala

Despite this clinical triumph, the adoption of PCSK9 inhibitors has been sluggish. The requirement for subcutaneous administration creates a psychological and logistical barrier for many patients, while the high cost of biologic manufacturing imposes significant economic constraints on healthcare systems [6]. Moreover, the need for cold-chain storage complicates distribution in resource-limited settings. This dichotomy between the proven biological efficacy of PCSK9 inhibition and the practical limitations of antibody therapy has catalyzed the search for small-molecule or peptide-based inhibitors that can be administered orally. The development of an oral PCSK9 inhibitor represents the "holy grail" of modern lipid management: a therapy offering the potent efficacy of a biologic with the convenience, stability, and potential cost-effectiveness of a traditional pill. This review critically examines the pharmacological innovations, clinical efficacy, and safety profiles of the emerging oral PCSK9 inhibitors, specifically focusing on the macrocyclic peptide MK-0616 and the small molecule AZD0780.

2. Pathophysiology and Therapeutic Targets

2.1. The Physiological Role of PCSK9

PCSK9 is a serine protease predominantly secreted by hepatocytes. Its primary physiological function is the regulation of cell-surface LDL receptor (LDLR) density. In the absence of PCSK9, LDLRs bind to circulating LDL-C, internalize the lipoprotein-receptor complex, and release the LDL cargo for lysosomal degradation while the receptor recycles back to the cell surface [7]. This recycling mechanism allows a single receptor to clear multiple LDL particles.

When PCSK9 is secreted, it binds to the epidermal growth factor-like repeat A (EGF-A) domain of the LDLR. This binding prevents the conformational change required for receptor recycling within the endosome. Instead, the PCSK9-LDLR complex is sorted for lysosomal degradation [8]. Consequently, elevated PCSK9 levels result in reduced LDLR density, impaired hepatic clearance of LDL-C, and hypercholesterolemia.

2.2. Mechanism of Pharmacological Inhibition

Therapeutic inhibition of PCSK9 disrupts its interaction with the LDLR, thereby preserving receptor density and enhancing the clearance of plasma cholesterol.

1. Monoclonal Antibodies: Agents like evolocumab sequester circulating PCSK9, preventing it from binding to the receptor.
2. Small Interfering RNA (siRNA): Inclisiran inhibits the hepatic synthesis of the PCSK9 protein [9].
3. Oral Inhibitors: The emerging class of oral agents utilizes small molecules or macrocyclic peptides to bind to the PCSK9 catalytic or allosteric sites, preventing the protein-protein interaction with LDLR. This approach presents a significant bioengineering challenge due to the large, flat surface area of the PCSK9-LDLR binding interface, which is traditionally considered difficult to target with small molecules [10]

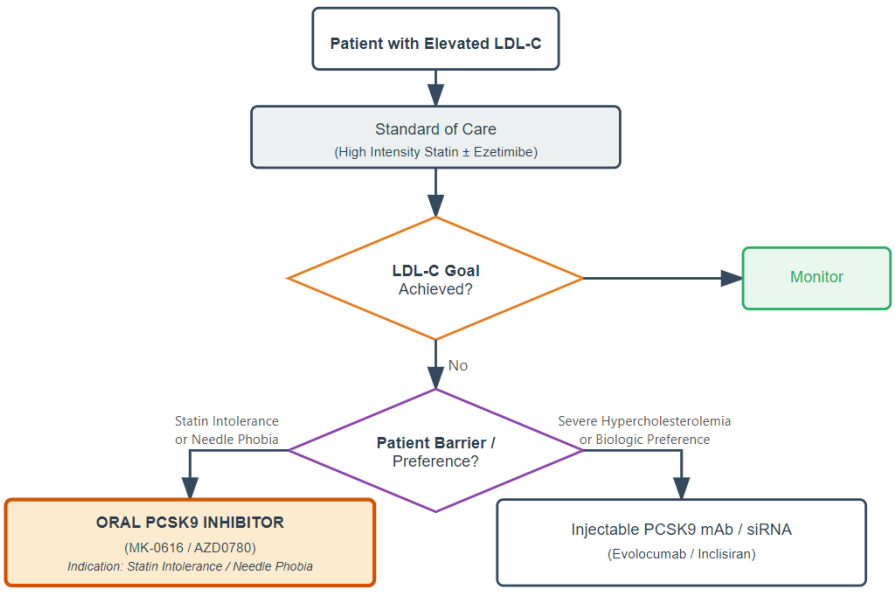

Figure 1. Therapeutic Applications of Oral PCSK9 Inhibitors

3. Oral PCSK9 Inhibitors

3.1. Design Challenges and Innovations

Developing an oral inhibitor for PCSK9 involves overcoming two primary obstacles: molecular size and bioavailability. The binding site on PCSK9 is relatively large, requiring a molecule with sufficient surface area to effectively block LDLR binding. However, larger molecules (such as peptides) typically suffer from poor oral bioavailability and rapid degradation by gastric enzymes [11]. Recent advancements in macrocyclic peptide chemistry and permeation enhancers have enabled the development of orally active agents that maintain high affinity for PCSK9.

Table 1. Technical Challenges and Solutions in Oral PCSK9 Development

Challenge	Physiological Barrier	Solution
Gastric Stability	Low pH and proteolytic enzymes (pepsin) in the stomach degrade proteins/peptides.	Macrocyclization: Constraining the peptide structure to resist enzymatic cleavage. Enteric Coating: Protecting the drug until it reaches the intestine.
Intestinal Permeability	Large molecular size prevents passive diffusion across the intestinal epithelium.	Permeation Enhancers: Agents (e.g., sodium caprate) that temporarily loosen tight junctions to facilitate absorption.
Target Binding Surface	The PCSK9-LDLR interface is flat and expansive (approx. 530 Å^2), difficult for small molecules to cover.	Macrocyclic Peptides: Larger than small molecules, capable of mimicking the "footprint" of an antibody. Allosteric Inhibition: Binding to a non-active site to induce conformational changes (Small molecules).

3.2. Investigational Drugs

Two primary candidates have emerged in advanced clinical trials, demonstrating the viability of this administration route.

3.2.1. MK-0616 (Macrocyclic Peptide)

MK-0616 is an orally bioavailable macrocyclic peptide developed to inhibit the PCSK9-LDLR interaction. Unlike traditional small molecules, its macrocyclic structure allows it to bind with high specificity to the large flat interface of PCSK9, mimicking the action of monoclonal antibodies [12].

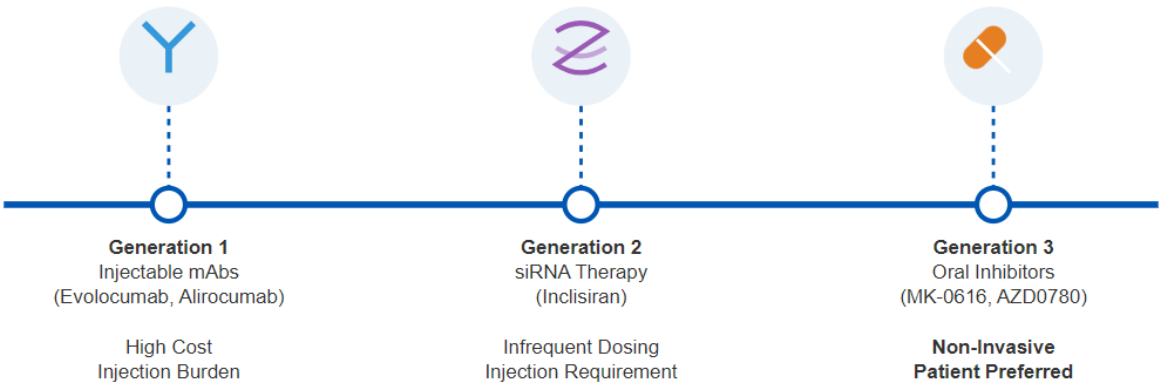

Figure 2. The Evolution of PCSK9 Targeting Molecules

Clinical Efficacy: In a Phase 2b randomized clinical trial involving 381 participants with hypercholesterolemia, MK-0616 demonstrated a dose-dependent reduction in LDL-C. At week 8, daily dosing resulted in LDL-C reductions of up to 60.9% compared to placebo [13]. The efficacy was consistent across various demographic subgroups and background lipid-lowering therapies.

3.2.2. AZD0780 (Small Molecule Inhibitor)

AZD0780 represents a small-molecule approach to PCSK9 inhibition. It is designed to be absorbed orally and inhibit the interaction between PCSK9 and the LDL receptor.

Clinical Efficacy: Data from the PURSUIT Phase 2b trial, presented recently, highlighted the efficacy of AZD0780 in patients with dyslipidemia on stable statin therapy. The study randomized patients to varying doses of AZD0780 (1 mg, 3 mg, 10 mg, and 30 mg) or placebo. At 12 weeks, the 30 mg once-daily dose achieved a placebo-adjusted LDL-C reduction of 52% [14]. When added to standard-of-care, this resulted in a total LDL-C reduction of nearly 80% from baseline. The trial indicated that the drug could be administered without food restrictions, enhancing patient convenience [15]

Table 2. Clinical Trials of Emerging Oral PCSK9 Inhibitors

Investigational Drug	Developer	Mechanism	Trial Name / Phase	Study Population (N)	Primary Endpoint (LDL-C Reduction)	Reference
MK-0616	Merck & Co.	Macrocyclic Peptide	Phase 2b (NCT05261193)	Hypercholesterolemia (n=381)	-41.2% to -60.9% (at Week 8)	[13]
AZD0780	AstraZeneca	Small Molecule	PURSUIT / Phase 2b	Dyslipidemia on Statins (n=~400)	-52% (at 30 mg) vs. Placebo	[14]
NNC0385-0434	Novo Nordisk	Oral Peptide	Phase 1	Healthy Volunteers	-46% (Max reduction)	*Ongoing*

Table 3. Pharmacokinetics of PCSK9 Inhibitors

Feature	Monoclonal Antibodies (e.g., Evolocumab)	Small Interfering RNA (e.g., Inclisiran)	Oral PCSK9 Inhibitors (e.g., MK-0616)
Molecular Structure	Large Protein (~145 kDa)	Double-stranded RNA	Macrocyclic Peptide or Small Molecule (< 2 kDa)
Route of Administration	Subcutaneous Injection	Subcutaneous Injection	Oral (Tablet/Capsule)
Dosing Frequency	Every 2 or 4 Weeks	Every 6 Months (Maintenance)	Once Daily
Mechanism	Extracellular Sequestration	Intracellular mRNA Degradation	Extracellular Binding / Allosteric Inhibition
Half-Life	11–17 Days	Unknown (Effect persists months)	Hours (Requires daily dosing)
Storage Requirements	Refrigeration (2–8°C)	Refrigeration (2–8°C)	Room Temperature

4. Comparison of Lipid-Lowering Modalities

The therapeutic landscape for hypercholesterolemia has evolved into a multi-modal arena, offering clinicians various tools to tailor treatment. However, understanding the distinct pharmacological and logistical profiles of these classes is crucial for optimal patient selection.

4.1. Pharmacokinetics and Administration

Statins and the novel oral PCSK9 inhibitors (MK-0616, AZD0780) share the convenience of daily oral dosing, which aligns well with existing patient routines for chronic disease management. In contrast, monoclonal antibodies (mAbs) like evolocumab require subcutaneous injections every two to four weeks, and the siRNA therapy inclisiran is administered biannually. While the infrequent dosing of injectables can be advantageous for patients struggling with daily adherence, the "pill burden" is generally preferred by the majority of the population over the discomfort and anxiety associated with needles [11, 16].

4.2. Mechanism and Efficacy

Mechanistically, statins inhibit intracellular cholesterol synthesis, which triggers a compensatory upregulation of LDLRs. However, this also triggers a compensatory increase in PCSK9 levels, which can attenuate the drug's efficacy over time. PCSK9 inhibitors work synergistically with statins by preventing this receptor degradation. While mAbs sequester PCSK9 in the plasma (extracellularly) and inclisiran prevents its synthesis (intracellularly), oral small molecules like AZD0780 bind to the PCSK9 protein to block its interaction with the receptor. Clinical data indicates that oral agents can match the high efficacy bar set by mAbs, offering LDL-C reductions in the range of 50–60%, significantly outperforming ezetimibe (~20% reduction) and bempedoic acid [14].

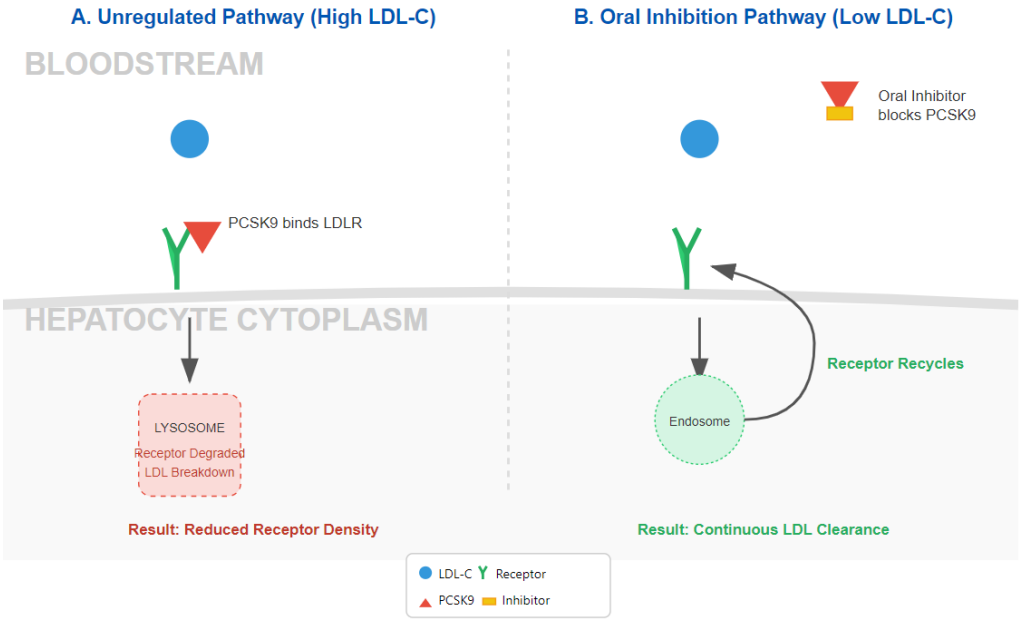

Figure 3. Mechanism of Action of Oral PCSK9 Inhibitors

[(A) In the unregulated state, PCSK9 binds to the LDL receptor (LDLR) on the hepatocyte surface, preventing recycling and promoting lysosomal degradation of the receptor. This leads to reduced LDL clearance. (B) Oral PCSK9 inhibitors (e.g., MK-0616, AZD0780) bind to circulating PCSK9, preventing its interaction with the LDLR. The receptor remains free to bind LDL-C, internalize it, and recycle back to the cell surface, thereby maintaining high receptor density and efficient cholesterol clearance.)]

4.3. Logistics and Access

A critical differentiator is stability. Biologics (mAbs and siRNA) are temperature-sensitive proteins that require strict cold-chain maintenance from manufacture to administration. This complicates distribution, particularly in developing nations or rural areas. Oral small molecules and macrocyclic peptides are chemically stable at room temperature, facilitating easier storage, transport, and distribution. This scalability could potentially lower the cost of therapy, addressing the primary barrier affordability that currently restricts PCSK9 inhibitor use to a fraction of eligible patients [17].

Table 4. Comparison of various drugs used in Lipid Management

Feature	Statins	Injectable PCSK9 mAbs	Oral PCSK9 Inhibitors
Drug Class	HMG-CoA Reductase Inhibitors	Monoclonal Antibodies	Small Molecules / Peptides
Route	Oral	Subcutaneous Injection	Oral
Frequency	Daily	Every 2–4 Weeks	Daily
LDL-C Reduction	30–50%	50–60%	50–60% (Projected)
Primary Limitation	Muscle symptoms, Resistance	Cost, Injection burden	Bioavailability (under study)
Status	Standard of Care	Approved (2nd/3rd line)	Clinical Development

5. Clinical Safety and Tolerability

Ensuring the safety of oral PCSK9 inhibitors is paramount, particularly given the historical challenges of developing oral drugs that interact with large protein surfaces. Early-phase clinical trials have provided encouraging safety signals that distinguish these agents from other lipid-lowering classes.

5.1. Effect on Muscles and Liver

A major concern with high-intensity statin therapy is statin-associated muscle symptoms (SAMS), ranging from myalgia to rhabdomyolysis. In the Phase 2b trial of MK-0616, no increase in muscle-related adverse events was observed compared to placebo, nor was there any signal of dose-dependent liver enzyme elevations (ALT/AST) [13]. This suggests that oral PCSK9 inhibitors function without entering the skeletal muscle or disrupting intracellular mevalonate pathways, making them a viable alternative for statin-intolerant patients.

5.2. Gastrointestinal Tolerability

Given the use of permeation enhancers (such as sodium caprate) to facilitate the absorption of macrocyclic peptides like MK-0616, gastrointestinal tolerability was a specific focus of investigation. Clinical data reported mild gastrointestinal disturbances, such as dyspepsia and diarrhea, but these were generally transient and did not lead to significant discontinuation rates [12]. Similarly, the AZD0780 PURSUIT trial reported a safety profile comparable to placebo, with discontinuation rates due to adverse events remaining very low (<2%) across all dosage groups [15].

5.3. Immunogenicity and Off-Target Effects

Unlike monoclonal antibodies, which can induce the formation of anti-drug antibodies (ADAs) that may attenuate efficacy over time, small molecules and peptides typically have lower immunogenic potential. However, the long-term implications of inhibiting PCSK9 via small molecules potentially affecting glucose metabolism or neurocognitive function remain theoretical risks that must be monitored in larger Phase 3 cardiovascular outcome trials. To date, no specific safety flags regarding cognitive changes or new-onset diabetes have been reported in the available Phase 2 data sets [14]

Table 5. Safety and Tolerability Profile in Phase 2 Studies

Adverse Event (AE) Category	MK-0616 (Phase 2b)	AZD0780 (PURSUIT)	Placebo (Pooled Analysis)
Any Adverse Event	~40%	~38.2%	~35%
Discontinuation due to AE	< 2%	1.5%	< 2%
Muscle-Related Events (Myalgia)	Rare; No significant difference from placebo	No significant signal reported	Low incidence
Liver Enzyme Elevation (>3x ULN)	None observed	No significant signal	None
Gastrointestinal Issues	Mild (Diarrhea, Dyspepsia)	Mild	Minimal
Injection Site Reactions	N/A	N/A	N/A

6. Future Perspectives and Global Impact

6.1. Addressing the Adherence Gap

The "pill burden" is a known factor in medication non-adherence; however, patients generally prefer oral medications over injections. The availability of an oral PCSK9 inhibitor could significantly improve compliance in asymptomatic patients who are reluctant to self-inject. Furthermore, the potential for fixed-dose combinations (e.g., a single pill containing a statin and an oral PCSK9 inhibitor) could streamline treatment regimens and maximize LDL-C reduction [16].

6.2. Economic and Accessibility Considerations

Biologic therapies (mAbs) are inherently complex and expensive to manufacture. Small molecule and peptide synthesis is generally more scalable and cost-effective. If oral PCSK9 inhibitors can be priced competitively, they may overcome the reimbursement

hurdles that have restricted the use of evolocumab and alirocumab [17]. This is particularly relevant for low- and middle-income countries where the burden of CVD is rising, but access to biologics is negligible.

Table 6. Potential Patient Candidates for Oral PCSK9 Therapy

Patient Population	Clinical Characteristic	Rationale for Oral PCSK9i
Statin Intolerant	History of statin-associated muscle symptoms (SAMS) or liver toxicity.	Provides a non-statin alternative with high efficacy without muscle-related side effects.
High-Risk ASCVD	Patients with recurrent events despite maximal tolerated statin therapy.	Offers additive LDL-C lowering (~50-60%) to achieve strict targets (<55 mg/dL).
Needle Phobic	Patients refusing injectable mAbs due to anxiety or discomfort.	Improves adherence through a familiar oral daily regimen.
Resource-Limited Settings	Regions lacking cold-chain infrastructure for biologics.	Oral tablets are shelf-stable and easier to distribute and store.

6.3. Recent Trends

Current research is focussing on:

1. Long-term Cardiovascular Outcomes: verifying that the LDL-C lowering observed with oral agents translates into a reduction in Major Adverse Cardiovascular Events (MACE).
2. Renal and Hepatic Safety: evaluating the metabolism of these novel agents in patients with comorbidities.
3. Polypharmacology: investigating the utility of these drugs in combination with other emerging lipid-lowering agents, such as angiopoietin-like 3 (ANGPTL3) inhibitors.

7. Conclusion

The development of oral PCSK9 inhibitors signifies a potential turning point in the management of dyslipidemia. By targeting a validated pathway with a patient-friendly delivery system, agents like MK-0616 and AZD0780 address the critical unmet needs of efficacy, convenience, and potentially, affordability. Early clinical data suggest these drugs can match the potency of injectable biologics while maintaining a safety profile suitable for chronic oral administration. As these therapies progress through Phase 3 trials, they hold the promise of reshaping treatment guidelines and reducing the global incidence of atherosclerotic cardiovascular disease

References

[1] Ference BA, Ginsberg HN, Graham I, et al. Low-density lipoproteins cause atherosclerotic cardiovascular disease. 1. Evidence from genetic, epidemiologic, and clinical studies. Eur Heart J. 2017;38(32):2459-2472.

[2] Libby P. The changing landscape of atherosclerosis. Nature. 2021;592(7855):524-533.

[3] Baigent C, Keech A, Kearney PM, et al. Efficacy and safety of cholesterol-lowering treatment: prospective meta-analysis of data from 90,056 participants in 14 randomised trials of statins. Lancet. 2005;366(9493):1267-1278.

[4] Stroes ES, Thompson PD, Corsini A, et al. Statin-associated muscle symptoms: impact on statin therapy European Atherosclerosis Society Consensus Panel Statement on Assessment, Aetiology and Management. Eur Heart J. 2015;36(17):1012-1022.

[5] Sabatine MS, Giugliano RP, Keech AC, et al. Evolocumab and Clinical Outcomes in Patients with Cardiovascular Disease. N Engl J Med. 2017;376(18):1713-1722.

[6] Warden BA, Fazio S, Shapiro MD. The Journey to an Oral PCSK9 Inhibitor. Curr Atheroscler Rep. 2023;25(5):251-259.

[7] Seidah NG, Benjannet S, Wickham L, et al. The secretory proprotein convertase neural apoptosis-regulated convertase 1 (NARC-1): liver regeneration and neuronal differentiation. Proc Natl Acad Sci U S A. 2003;100(3):928-933.

[8] McNutt MC, Lagace TA, Horton JD. Catalytic activity is not required for secreted PCSK9 to reduce low density lipoprotein receptors in HepG2 cells. J Biol Chem. 2007;282(29):20799-20803.

[9] Ray KK, Wright RS, Kallend D, et al. Two Phase 3 Trials of Inclisiran in Patients with Elevated LDL Cholesterol. N Engl J Med. 2020;382(16):1507-1519.

[10] Lagace TA. PCSK9 and LDL receptors: specific binding, endocytosis and degradation. Curr Opin Lipidol. 2014;25(5):387-393.

[11] Drucker DJ. Advances in oral peptide therapeutics. Nat Rev Drug Discov. 2020;19(4):277-289.

[12] Johns DG, Campeau LC, Walji AM, et al. Orally Bioavailable Macrocyclic Peptide That Inhibits Binding of PCSK9 to the Low Density Lipoprotein Receptor. Circulation. 2023;148(2):144-158.

[13] Ballantyne CM, Banka P, Mendez G, et al. Phase 2b Randomized Trial of the Oral PCSK9 Inhibitor MK-0616. J Am Coll Cardiol. 2023;81(16):1553-1564.

[14] Koren MJ, Vega RB, Agrawal N, et al. An Oral PCSK9 Inhibitor for Treatment of Hypercholesterolemia: The PURSUIT Randomized Trial. J Am Coll Cardiol. 2025;85(21):1996-2007.

[15] Vega R, Agrawal N, Xu Y, et al. AZD0780, the first oral small molecule PCSK9 inhibitor for the treatment of hypercholesterolemia: results from a randomized, single-blind, placebo-controlled phase 1 trial. Atherosclerosis. 2024;395:118514.

[16] Talha K, Sharma G. Oral PCSK9 inhibitors: A new era in lipid management. Am J Prev Cardiol. 2024;17:100612.

[17] Annemans L, Packard CJ, Briggs A, et al. "Highest risk-highest benefit" strategy: a pragmatic, cost-effective approach to targeting PCSK9 inhibitor therapy. Eur Heart J. 2018;39(27):2546-2550.

Author's Short Biography

Mr. Edward Raju Gope

Mr. Edward Raju Gope is an Assistant Professor of Pharmaceutical Analysis at K. G. R. L College of Pharmacy in Bhimavaram, Andhra Pradesh. He holds a Master's degree in Pharmaceutical Analysis. Edward is passionate about educating students in developing effective and industrially applicable pharmaceutical formulations. He constantly strives to make the subject engaging and research-oriented for learners. Edward also encourages collaboration with industries through student projects and facility visits.

Miss Durga Bhavani Puppala

Currently pursuing B.Pharmacy at KGRL College of Pharmacy, Bhimavaram. She has shown keen interest in pharmaceutical analysis and has participated in various college-level research projects. Her academic focus includes understanding basic analytical techniques and quality control in pharmaceuticals.

Miss Navya Andhugula

An undergraduate B.Pharmacy student at KGRL College of Pharmacy with strong academic performance. She has participated in several workshops on pharmaceutical analysis and has developed interest in chromatographic techniques. Her academic projects focus on basic analytical method development.

Miss Naga Navya Donga

Currently pursuing B.Pharmacy in Pharmaceutical Analysis at KGRL College of Pharmacy, Bhimavaram. Her research focuses on analytical method development and validation using chromatographic techniques. She has participated in several national conferences and workshops on pharmaceutical analysis and quality control.

Mr. Durga Prasad Madu

A B.Pharmacy student at KGRL College of Pharmacy with particular interest in pharmaceutical analysis and quality control. He has actively participated in college laboratory sessions and shows enthusiasm in learning modern analytical techniques. His academic work includes projects in basic pharmaceutical analysis.

Dr. Raghava D

Dr. Raghava D, is the Principal of K.G.R.L. College of Pharmacy, Bhimavaram, India is an eminent Pharmacy professional having 15 years of experience in Pharmacy teaching and pharmaceutical Industry.

Dr. Nageswara Rao K

Dr. Kavala Nageswara Rao, M.Pham., Ph.D from Andhra University having 22 years of experience in Pharma Industry in India. He worked as a Community Pharmacist in abroad for 9 years, kingdom of Saudi Arabia and 17 years of teaching in Bhimavaram. He served in various capacities of many reputed multinational companies like Rallis India Ltd., Raptakos, Brette & Co. Ltd.,Mumbai.