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Formulation and Evaluation of Obeticholic Acid Sustained-Release Formulations: Enhancing Solubility and Bioavailability—A Review

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Abstract
Currently used to treat Primary Biliary Cholangitis (PBC), obeticholic acid (OCA) is a semisynthetic bile acid analogue and a strong, selective agonist of the Farnesoid X Receptor (FXR). It is also being studied for Non-Alcoholic Steatohepatitis (NASH). OCA is a BCS Class II medication that is pharmacologically effective, but its clinical performance is limited by poor aqueous solubility, inconsistent gastrointestinal absorption, and extensive hepatic first-pass metabolism. These factors all require higher dosages and contribute to dose-related side effects like pruritus. The pharmacology, biopharmaceutical constraints, and formulation strategies pertinent to OCA are compiled in this study, with a focus on solubility-enhancement methods (solid dispersion). amorphous solid forms, cyclodextrin complexation, lipid-based carriers, nanotechnology, and sustained-release matrix technology using hydrophilic polymers like Hydroxypropyl Methylcellulose (HPMC K100M). In the long-term management of chronic cholestatic liver disease, sustained-release formulations in conjunction with solid-dispersion-based solubilisation are emphasised as a logical approach to achieve controlled drug release, improved dissolution, enhanced bioavailability, decreased dosing frequency, and better patient compliance.

Keywords: Obeticholic acid, solid dispersion, prolonged release, farnesoid X receptor, HPMC K100M, increased bioavailability, and primary biliary cholangitis.

I. Introduction

Primary biliary cholangitis (PBC) and non-alcoholic steatohepatitis (NASH) are two illnesses where dysregulated bile acid metabolism is crucial to the onset and progression of chronic liver diseases, which constitute a significant worldwide health burden. In addition to their role in lipid digestion, bile acids also function as signalling molecules that control hepatic and systemic metabolic pathways.

Excessive intrahepatic buildup of bile acids causes oxidative stress, inflammation&fibrosis.[1,2]
At the Farnesoid X Receptor, obeticholic acid—which is produced from chenodeoxycholic acid— is about 100 times more powerful than the natural ligand. Because of its increased potency, OCA can significantly affect bile acid synthesis, bile flow, glucose and lipid homeostasis, inflammatory signalling, and fibrogenesis. It was approved as a second-line PBC treatment after pivotal trials in patients with UDCA-refractory PBC showed notable decreases in bilirubin and alkaline phosphatase.[1,3,4]

OCA's clinical utility is restricted by unfavourable biopharmaceutical properties, despite this therapeutic promise. Its poor aqueous solubility limits its ability to dissolve in gastrointestinal fluids; this is exacerbated by hepatic metabolism and extensive enterohepatic circulation, which together result in variable and frequently low oral bioavailability. The focus of this review is on advanced solubility-enhancing and controlled-release formulation strategies for OCA, which are justified by these limitations.[5]

II. Pharmacology and Mechanism of Action

The liver, colon, kidney, and adrenal glands are the main organs that express the ligand-activated nuclear receptor FXR. The rate-limiting enzyme of bile acid synthesis, cholesterol 7α-hydroxylase (CYP7A1), is suppressed by the Small Heterodimer Partner (SHP) that FXR induces upon activation by OCA. Additionally, intestinal FXR activation stimulates Fibroblast Growth Factor 19 (FGF19) signalling, which further represses CYP7A1 via hepatic FGFR4. When combined, these routes restrict the buildup of harmful bile acid and decrease the generation of bile acid in the liver.[6–9]

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By upregulating the Bile Salt Export Pump (BSEP) and Multidrug Resistance Protein 3 (MDR3) on the hepatocyte canalicular membrane, OCA simultaneously promotes bile acid efflux, improving bile flow and decreasing cholestatic injury. This effect is clinically manifested in PBC patients' declining serum alkaline phosphatase levels. [6–9]

Beyond maintaining bile acid homeostasis, FXR activation limits collagen deposition and extracellular matrix accumulation by suppressing NF-κB-mediated transcription of proinflammatory cytokines (TNF-α, IL-1β, and IL-6), decreasing hepatic macrophage activation, and preventing hepatic stellate cell activation. The disease-modifying potential of OCA in chronic cholestatic and steatohepatitic liver disease is supported by these anti-inflammatory and antifibrotic properties as well as bile acid control. [6–9]

III. Biopharmaceutical Limitations of Obeticholic Acid

OCA is a BCS Class II molecule that is extremely lipophilic and has limited water solubility, which restricts its ability to dissolve in gastrointestinal fluids. A significant portion of an oral OCA dosage may pass through the gastrointestinal tract unabsorbed, lowering systemic exposure and delaying commencement of effect because dissolution is often the rate-limiting stage in absorption for poorly soluble medicines.[5]

Inter-individual differences in stomach pH, bile secretion, gastrointestinal motility, and fed/fasted condition all affect the degree of drug solubility and, in turn, plasma concentration, which further complicates OCA absorption. Some individuals may have subtherapeutic exposure as a result of this variability, while others may experience greater systemic exposure, which carries a risk of toxicity.

OCA is extensively metabolised by the liver after absorption, which further lowers the amount of active medication that enters the bloodstream. Patients are more likely to experience dose-related side effects like pruritus, higher treatment costs, and decreased adherence in a condition that usually necessitates lifelong therapy because higher doses are frequently needed to make up for poor solubility, variable absorption, and first-pass metabolism. [undefined] Stability issues, since OCA may be degraded by heat, moisture, and light, as well as the manufacturing complexity of solubility-enhancing methods such nanocarriers, lipid systems, and solid dispersions, further limit formulation development. The quest for sophisticated, solubilityenhancing sustained-release delivery methods for OCA is justified by these issues taken together: low solubility, uneven absorption, first-pass metabolism, high dosage needs, formulation complexity, and stability problems.

IV. Formulation Strategies for Solubility and Bioavailability Enhancement

4.1 Solid Dispersion Systems

By increasing wettability and decreasing crystallinity to improve dissolution, solid dispersion entails dispersing a poorly soluble medication in an amorphous or finely crystalline state within an inert, hydrophilic carrier. Common methods include solvent evaporation, hot-melt extrusion, spray drying, and lyophilization, usually using hydrophilic polymers as carriers like PVP, PEG, HPMC, or Soluplus®. [10, 11]

In the case of OCA in particular, solid dispersion using Poloxamer 188/407 via a fusion technique has been demonstrated to increase aqueous solubility to 0.347 mg/mL and achieve nearly complete (99.63%) drug release within 60 minutes, confirming solid dispersion as an efficient solubilityenhancement method for this molecule. The medicinal significance of polymer-stabilized amorphous OCA systems has been highlighted by a patented formulation that similarly stabilised amorphous OCA utilising HPMC, HPMC-AS, Eudragit grades, PVP, copovidone, and Soluplus®. [46,48]

4.2 Lipid-BasedFormulations

Lipid-based delivery systems, such as Self-Emulsifying and Self-Microemulsifying Drug Delivery Systems (SEDDS/SMEDDS), Solid Lipid Nanoparticles, Nanostructured Lipid Carriers, liposomes, bilosomes, and nanoemulsions, provide a mechanistically appropriate method of solubilisation due to the strong lipophilicity of OCA. In the gastrointestinal tract, these systems naturally create fine oil-in-water dispersions that offer a sizable interfacial area for dissolution and, in some situations, encourage lymphatic transport that avoids hepatic first-pass metabolism. [12– 14]

4.3 Nanotechnology-Based Approaches

According to the Noyes–Whitney relationship, nanocrystals, polymeric nanoparticles, nanoemulsions, nanosuspensions, and polymeric micelles use an elevated surface-area-to-volume ratio to speed up dissolution. For a lipophilic steroidal acid like OCA, polymeric micelles—which are created when amphiphilic polymers self-assemble—are

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especially appealing because their hydrophobic core may encapsulate the medication while the hydrophilic shell preserves water dispersibility.[15–17]

4.4 Cyclodextrin Complexation

Cyclodextrins, especially hydroxypropyl-β-cyclodextrin, create inclusion complexes where lipophilic drug molecules are accommodated in the hydrophobic cavity. This improves aqueous solubility, dissolution rate, and chemical stability while providing protection from oxidation, moisture, and photodegradation. Higher formulation costs and comparatively limited drug-loading capacity are drawbacks. [18–20]

4.5 Amorphous Drug Forms

Converting a crystalline medication to its amorphous state removes the lattice energy barrier to conversion dissolution, resulting in increased apparent solubility and quicker dissolving. However, amorphous OCA systems need stabilising polymers like PVP, HPMC, Soluplus®, or copovidone to sustain performance during shelf life since they are thermodynamically unstable and prone to recrystallisation. [21–23]

4.6 Ancillary Solubility- and Permeability-Enhancing Techniques

Particle-size reduction (micronization and nanonization) to increase surface area for dissolution, salt formation where ionisable groups allow, solvent evaporation and spray drying to produce amorphous solid dispersions, microemulsion systems for direct solubilisation, and permeation enhancers like bile salts, fatty acids, and surfactants to facilitate transcellular or paracellular transport after the drug has dissolved are additional methods applicable to OCA. [24–34]

V. Polymers, Excipients and Sustained-Release Matrix Design

In sustained-release systems, excipients offer processing efficiency and structural integrity, while rate-controlling polymers govern drug release over a prolonged duration. While hydrophobic polymers (ethyl cellulose, Eudragit RS/RL) mainly limit drug release through diffusion within a largely non-swelling matrix, hydrophilic polymers (HPMC, hydroxyethyl cellulose, hydroxypropyl cellulose, sodium carboxymethyl cellulose, and polyethylene oxide) hydrate upon contact with gastrointestinal fluid to form a gel layer that controls drug diffusion. Natural polymers with biodegradability and economic benefits include xanthan gum, guar gum, chitosan, and alginate. [35–39]

While surfactants like polysorbate 80 and poloxamers can further enhance the wettability and dissolution of poorly soluble medications like OCA within the matrix, functional excipients— diluents (lactose, microcrystalline cellulose), binders (PVP, hydroxypropyl cellulose), lubricants (magnesium stearate, stearic acid), and glidants (colloidal silicon dioxide) support tablet manufacturing. [35–39]

Because of their easy production process and consistent release behaviour, which is controlled by water penetration, polymer hydration and gel-layer development, drug diffusion through the gel, and gradual matrix degradation, matrix tablets continue to be the most popular sustained-release type. Mucoadhesive polymers (chitosan, Carbopol, sodium alginate) and coating systems (sustained-release, enteric, or taste-masking) provide complementary methods for extending gastrointestinal residence length and regulating release site or rate. [35–39] Sustained-release delivery has several benefits for chronic conditions like PBC and NASH that need to be treated indefinitely. These benefits include the preservation of plasma drug concentrations within the therapeutic window, fewer doses and better adherence, more thorough and consistent dissolution-limited absorption, and blunting of peak plasma concentrations linked to dose-dependent side effects like pruritus. The development of a sustained-release OCA pill based on HPMC is justified by these advantages taken together. [40–43]

VI. Analytical and Formulation Precedents in the Literature

Validated analytical procedures are precondition to formulation work. RP-HPLC and UHPLC/UV methods for OCA using C18 columns with acetonitrile- or methanol-based mobile phases have demonstrated linearity ($R^2 \approx 0.999$) over low-microgram concentration ranges, recovery of 98–102% and %RSD below 2% on precision and robustness testing, confirming their suitability for assay, dissolution and stability-indicating applications. [45, 47]

Matrix-based sustained-release design employing HPMC has been verified across numerous different poorly soluble or high-dose actives: HPMC K100M-based propranolol hydrochloride tablets achieved diffusion-controlled (Higuchi) release extending to 12 hours, while HPMC K4M/K100M combinations with rosuvastatin achieved 99.4%

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release over 20 hours following first-order, non-Fickian kinetics — both supporting HPMC K100M as a robust rate-controlling polymer applicable to OCA sustained-release design. [44, 49]

Beyond OCA alone, solid dispersion has been repeatedly demonstrated to improve the dissolution of poorly soluble actives: PVP K30-based solid dispersions significantly increased the solubility and dissolution rate of carvedilol, while PVP K30 combined with Gelucire 44/14 or Vitamin E TPGS enhanced the intrinsic dissolution rate of carbamazepine approximately four times compared to the pure drug. DSC and XRD confirmed that both drugs exist in an amorphous, molecularly dispersed state. A comparable solid-dispersion-first method (using HPMC-AS, PEG 6000, low-substituted hydroxypropyl cellulose, and crospovidone) is used in an industrial patent for OCA tablets in order to precisely address poor powder flow and blending issues related to OCA. [50–52]

VII. Conclusion and Future Perspective

The literature reviewed here indicates that no single solubilisation technique is universally superior; rather, solid dispersion, lipid-based carriers, nanotechnology, cyclodextrin complexation and amorphous stabilisation each offer complementary advantages that can be matched to the specific physicochemical and manufacturing constraints of OCA. Coupling a solid-dispersionbased solubility enhancement step with a hydrophilic polymer (HPMC K100M) matrix sustainedrelease system represents a particularly rational strategy, since it addresses both the dissolutionlimited absorption of OCA and the need for prolonged, consistent plasma exposure in chronic
PBC/NASH therapy while reducing peak-related adverse effects and dosing frequency.

In order to support clinical translation, future research should concentrate on establishing in vitro–in vivo correlations for such combined solid-dispersion sustained-release OCA systems, verifying the long-term physical stability of the amorphous phase within the matrix, and producing comparative bioavailability data against the commercial immediate-release product.

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