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**Review Article**

# Green Tea Niosomal Gel: An Approach For Protection Against Blue Light-Induced Skin Damage

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## ABSTRACT

Blue light emitted from sunlight and digital devices has been recognized as an important environmental factor that contributes to oxidative stress, inflammation, hyperpigmentation, and premature skin aging. Green tea (Camellia sinensis) extract is rich in polyphenolic compounds, particularly epigallocatechin-3-gallate (EGCG), which possesses potent antioxidant, anti-inflammatory, and photoprotective properties. However, the topical application of green tea extract is limited by its poor stability and inadequate skin penetration. The present study aims to develop and evaluate a green tea extract-loaded niosomal gel for enhanced topical delivery and protection against blue light-induced skin damage. Niosomes are non-ionic surfactant-based vesicular systems capable of improving the stability, encapsulation efficiency, controlled release, and skin permeation of bioactive compounds. In this study, green tea extract-loaded niosomes will be prepared using the ether injection method and characterized for vesicle size, polydispersity index (PDI), zeta potential, entrapment efficiency, and physical stability. The optimized niosomal suspension will be incorporated into a Carbopol 934 gel base and evaluated for physicochemical properties, antioxidant activity, and anti-blue light potential. The developed formulation is expected to provide sustained release of EGCG, improve skin retention, reduce oxidative stress, and offer enhanced protection against blue light-induced skin damage, making it a promising herbal nanocosmeceutical for dermatological and cosmetic applications.

## INTRODUCTION

### 1.1 Topical drug delivery: -

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A topical drug delivery system is a traditional drug delivery system having a history of more than a thousand years. In ancient days ointments and salves are made from plant, animal and mineral extracts. These preparations are delivered through a topical route for treatment of skin diseases. This method was employed by Chinese, Egyptians, and Babylonians.[1] The skin acts as a barrier for the entry and exit of many chemicals, prevents moisture loss, and controls body temperature to maintain homeostasis within the body. Topical preparations are useful in preventing gastric degradation of drugs and avoiding first-pass metabolism. So, there is an increase in bioavailability of the drug, and these topical preparations give its action directly at the site of action.[2]

**VARIOUS TOPICAL FORMULATIONS AVAILABLE IN THE MARKET ARE LISTED BELOW: -**

- Semi -Solid preparations: -

Eg: - Gels, Paste, Creams, Ointments.

- Liquid Preparations: -

Eg: - Lotions, Solutions, Liniments.

- Solid Preparations: -

Eg: - Powders.

- Other Preparations: -

Eg: - Transdermal patches, Foams, Sprays, etc.[3]

**BENEFITS OF TOPICAL DRUG DELIVERY SYSTEM: -**

☐ Avoidance of First-Pass Metabolism.

☐ Targeted, Localized Therapy.

☐ Improved Patient Compliance.

☐ Reduced Systemic Side Effects.

☐ Patient-Friendly Formulations.

☐ Lower Dose and Enhanced Efficacy.

☐ Non-Invasive and Easily Reversible.

☐ Controlled or Sustained Release Possibilities.[4]

**1.2 Anatomy and physiology of skin: -**

The largest organ of human body is skin it covers about 15% of total adult body weight it performs many important functions like protection against external physical, chemical and biological agents, it prevents excessive water loss from the body. The skin is composed of three main layers: -1) Epidermis, 2) Dermis and 3) Subcutaneous fascia .[5] Epidermis is the outermost layer of the skin having different layers such as stratum spinosum, stratum granulosum, stratum lucidum, stratum basale. Cells of epidermis includes keratinocytes, melanocytes, langerhans and marker cells. Dermis is a second layer of skin which is connected to epidermis by the basement membrane. The dermis is made up of two layers of connective tissue, the reticular and papillary, which blend together without obvious separation. The higher dermal layer, known as the papillary layer, is thinner and made up of loose connective tissue that comes into touch with the epidermis. The deeper, thicker, and less cellular layer is called the reticular layer. Collagen fiber bundles make up the dense connective tissue that makes up this layer. Hypodermis also known as Subcutaneous fascia, it is located beneath the dermis so it is called as hypodermis. It is the deepest layer of the skin, it consists of sensory neurons, blood vessels, hair follicle and adipose lobules.[6]

The skin structure and physiology play a crucial role in the delivery and permeation of drug via this route. The presence of hair follicles and pores plays a critical role in drug permeation.[7] Stratum Corneum is layer of skin composing of 40% lipids, 40% proteins and only 20% water. So, this composition helps in permeation of liphophillic drug. This liphophillic character of drug suitable for topical delivery of drugs. However hydrophilic drugs are difficult to pass through stratum corneum due to its less water content. Here the hair follicles and pores play role in the absorption of drugs.[8]

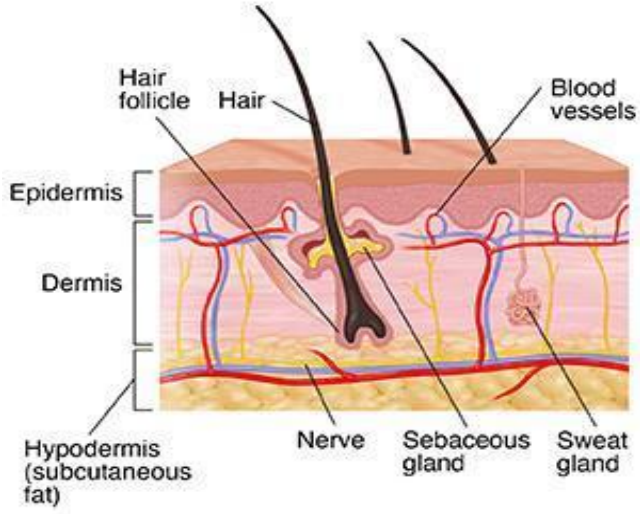



**Fig 1.1: - Structure of Skin**

### 1.3 Skin Aging

Skin aging is a progressive biological process characterized by structural and functional deterioration.

It is classified into:

**Intrinsic Aging**

Occurs naturally due to:

- Genetics
- Hormonal alterations
- Cellular senescence
- Reduced collagen synthesis

Characteristics include:

- Fine wrinkles
- Thin epidermis
- Dry skin
- Reduced elasticity

**Extrinsic Aging :**

Major contributors Results from environmental factors.

include:

- Ultraviolet radiation
- Blue light
- Air pollution
- Cigarette smoke
- Poor nutrition
- Psychological stress

Extrinsic aging is largely mediated through oxidative stress.

### 1.4 Photoaging

Photoaging refers to premature aging caused by chronic exposure to environmental radiation.

Although ultraviolet radiation has traditionally been considered the principal cause of photoaging, increasing evidence suggests that prolonged exposure to visible blue light also contributes significantly to:

- Hyperpigmentation
- ROS generation
- DNA oxidation
- Collagen degradation
- Matrix metalloproteinase activation
- Wrinkle formation
- Skin barrier dysfunction

Unlike UVB, blue light penetrates deeper into the dermis, affecting fibroblasts and extracellular matrix proteins responsible for maintaining skin elasticity.

### 1.5 Blue Light: Sources and Characteristics

Visible light constitutes approximately **44%** of the solar electromagnetic spectrum and ranges from **400 to 700 nm**. Blue light, also referred to as **High-Energy Visible (HEV) light**, occupies the wavelength range of **400–500 nm**. Owing to its relatively short wavelength and high photon energy, blue light possesses greater biological activity than other visible wavelengths and has become an important focus of dermatological and cosmetic research.

Historically, sunlight has been the major source of blue light. However, modern lifestyles have significantly increased exposure to artificial blue light through digital devices and LED-based illumination. Recent estimates suggest that people spend nearly **90% of their time indoors**, where LED lighting and electronic screens have become the predominant sources of visible light exposure.

#### 1.5.1 Sources of Blue Light

Natural sources include:

- Sunlight
- Clear daylight
- Reflected solar radiation

Artificial sources include:

- Smartphones
- Tablets
- Laptop computers
- Desktop monitors
- LED televisions
- LED bulbs
- Computer monitors
- Gaming devices
- Medical LEDs
- Cosmetic LED

#### 1.5.2 Physical Properties of Blue Light

| Property | Value |
|---|---|
| **Wavelength** | 400–500 nm |
| **Photon energy** | Highest among visible wavelengths |
| **Skin penetration** | Extends into the dermis |
| **Major source** | Sunlight and LED devices |
| **Biological effect** | ROS generation and oxidative stress |

Because photon energy is inversely proportional to wavelength (Planck's equation), blue light possesses considerably greater energy than green, yellow, orange, or red light. This high energy

enables interactions with endogenous chromophores and photosensitizers present within skin cells, initiating photochemical reactions that generate reactive oxygen species

### 1.5.3 Blue Light-Induced Skin Damage

Increasing evidence demonstrates that blue light induces numerous adverse biological effects on the skin. While ultraviolet radiation has traditionally been regarded as the principal environmental cause of photoaging, visible blue light has recently emerged as an independent contributor to oxidative skin damage.

Blue light penetrates more deeply than UVB radiation and reaches the dermis, where fibroblasts, collagen fibers, and elastin fibers reside. Consequently, prolonged exposure promotes extracellular matrix degradation and accelerates skin aging.

**Major biological effects include:**

**1. ROS Generation**

Blue light excites endogenous photosensitizers such as flavins, porphyrins, NADH, and lipofuscin. Excited photosensitizers transfer energy to molecular oxygen, generating ROS including:

- Superoxide anion ($O_2^{\bullet -}$)
- Hydrogen peroxide ($H_2O_2$)
- Hydroxyl radicals ($\bullet OH$)
- Singlet oxygen ($^1O_2$)

ROS production represents the primary mechanism responsible for blue light-induced phototoxicity.

**2. Oxidative Damage**

Excess ROS attack cellular macromolecules:

- DNA
- Lipids
- Proteins
- Mitochondria
- Cell membranes

This oxidative damage disrupts normal cellular metabolism and promotes apoptosis.

**3. DNA Damage**

Experimental studies demonstrate that blue light induces:

- DNA strand breaks
- Oxidized nucleotides
- 8-OHdG formation
- Chromosomal instability

Persistent DNA damage accelerates cellular senescence and contributes to photoaging.

**4. Inflammatory Response**

Blue light stimulates inflammatory signaling pathways including:

- NF-κB
- MAPK
- AP-1
- TRPV1 activation

These pathways promote production of inflammatory mediators such as:

- TNF-α
- IL-1β
- IL-6
- COX-2

Inflammation subsequently enhances collagen degradation and impairs skin repair

**5. Hyperpigmentation**

Blue light activates melanocytes through:

- Opsin-3 receptor
- Calcium signaling
- MITF activation
- Increased tyrosinase expression

These events stimulate melanin synthesis, producing persistent pigmentation, particularly in individuals with darker skin phototypes.

**6. Skin Barrier Dysfunction**

Blue light reduces epidermal barrier integrity by:

- Increasing lipid peroxidation
- Disrupting keratinocyte differentiation
- Altering ceramide metabolism

Barrier impairment increases trans-epidermal water loss and skin sensitivity.

**1.5.4 Mechanism of Blue Light-Induced Oxidative Stress**

The mechanism of blue light-induced skin damage involves a cascade of molecular events.

**Step 1**

Blue light penetrates the epidermis and dermis.

**Step 2**

Endogenous chromophores absorb photons.

**Step 3**

Generation of ROS.

**Step 4**

Oxidative stress exceeds antioxidant defenses.

**Step 5**

Activation of:

NF-κB

MAPK

AP-1

TRPV1

**Step 6**

Production of inflammatory cytokines and matrix metalloproteinases.

**Step 7**

- Collagen degradation
- Elastin fragmentation
- DNA oxidation
- Hyperpigmentation
- Premature aging

This oxidative pathway provides the rationale for using antioxidant-rich botanical formulations to neutralize ROS before irreversible tissue damage occurs.

**1.6 Gel: -**

As per USP Gels are defined as a semi solid system containing either suspensions made up of small organic or inorganic molecules interpenetrated by a liquid. These are the preparation which are intended for application on the skin. Gels are generally considered to be more rigid because gels contain more covalent cross links, a higher density of physical bonds. [10] A gel consists of natural or synthetic polymers forming a three-dimensional matrix throughout a hydrophilic liquid or a dispersion medium. A gel is made up of two layers cross-linked, three-dimensional substance that contains a significant volume of liquid to create a stiff network which immobilizes the liquid continuous phase.

**Ideal properties of topical gel:**

- Gel should be clear and homogenous.
- Should be inert in nature.
- It should be stable, non-irritant.
- It should be non-sticky.
- It should have anti- microbial activity.

**Advantages of Gel: -**

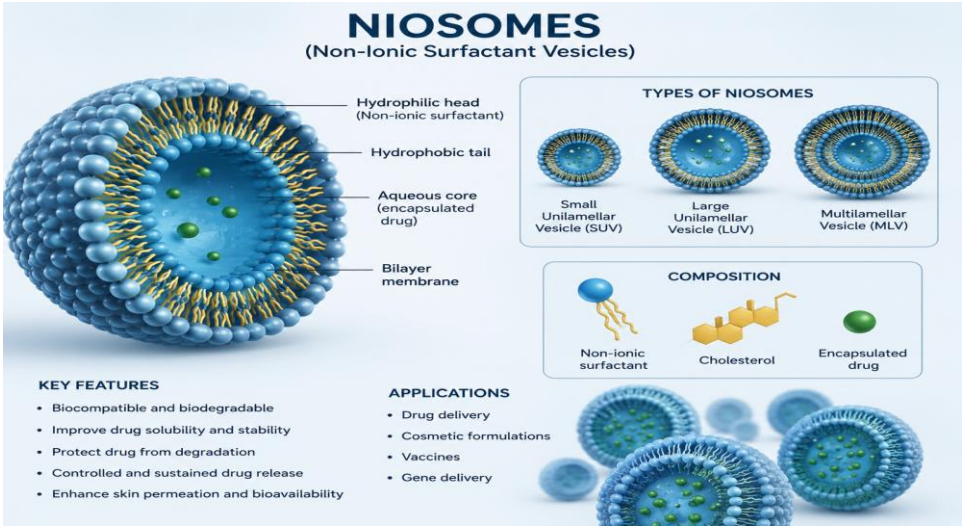
- It can be used as controlled release formulation.
- They can be used to administer both polar and non-polar drugs.
- Easy to formulate as compared to other semi solid dosage form.
- They are biodegradable and biocompatible.
- They are washable and nontoxic in nature.
- Retention time is higher than other topical dosage forms.
- They provide excellent spread ability and cooling effect.

**Disadvantages: -**

- Some drug may degrade in gel formulation due to presence of polymer.
- Gelling agent may precipitate and result in salting out.
- Flocculation in some gel may produce an unstable gel.
- Solvent evaporation from the formulation may result in drying of the gel.
- The additives may induce irritation.
- The water content may increase the chances of microbial or fungal attack in gel.[10]

**1.6.1 Niosomal gel: -**

Niosomal gels are semisolid systems where niosomes are embedded within a gel matrix, combining enhanced solubility, stability, and controlled release of therapeutic agents with easy topical application and prolonged residence time.[11] These systems improve penetration, bioavailability, and patient compliance while enabling sustained and targeted delivery.

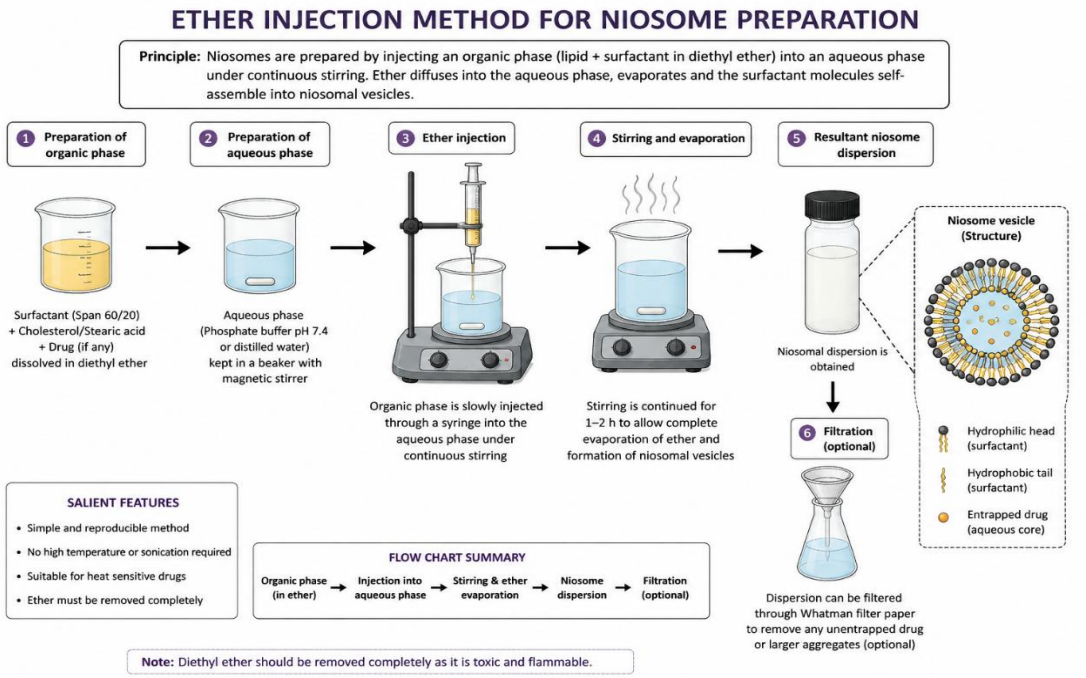



**1.7 Method of preparation of Niosomes: -**

**1. Ether injection method**

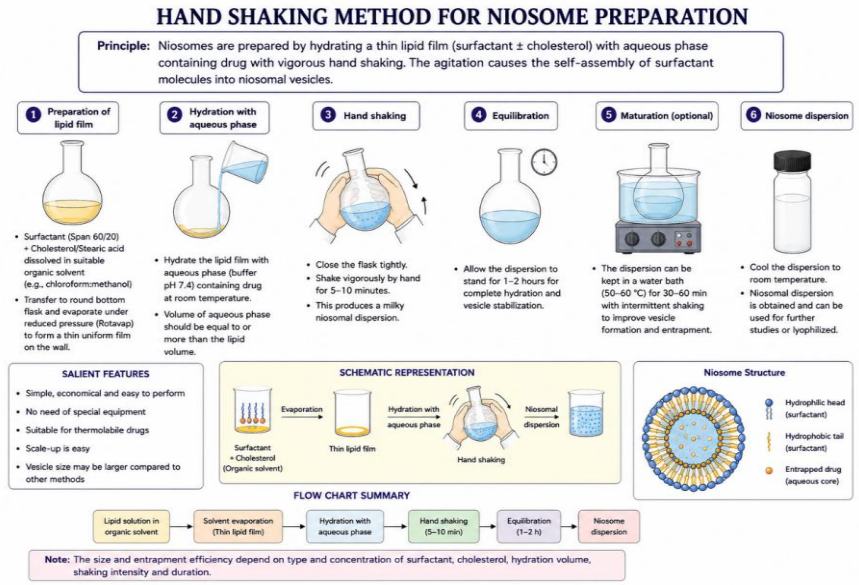
In this method cholesterol and non-ionic surfactant are dissolved in diethyl ether. This mixture is then injected through a 14 guage needle into an aqueous solution of drug maintained at 60°C. The ether solution is evaporated to room temperature giving

single layered vesicles having particle size in the range of 50-1000nm. The advantage of this method includes control of size, which can be obtained by controlling the size of the needle whereas limited solubility of materials in ether and difficulty to remove ether from the final formulation are the disadvantages of this method.



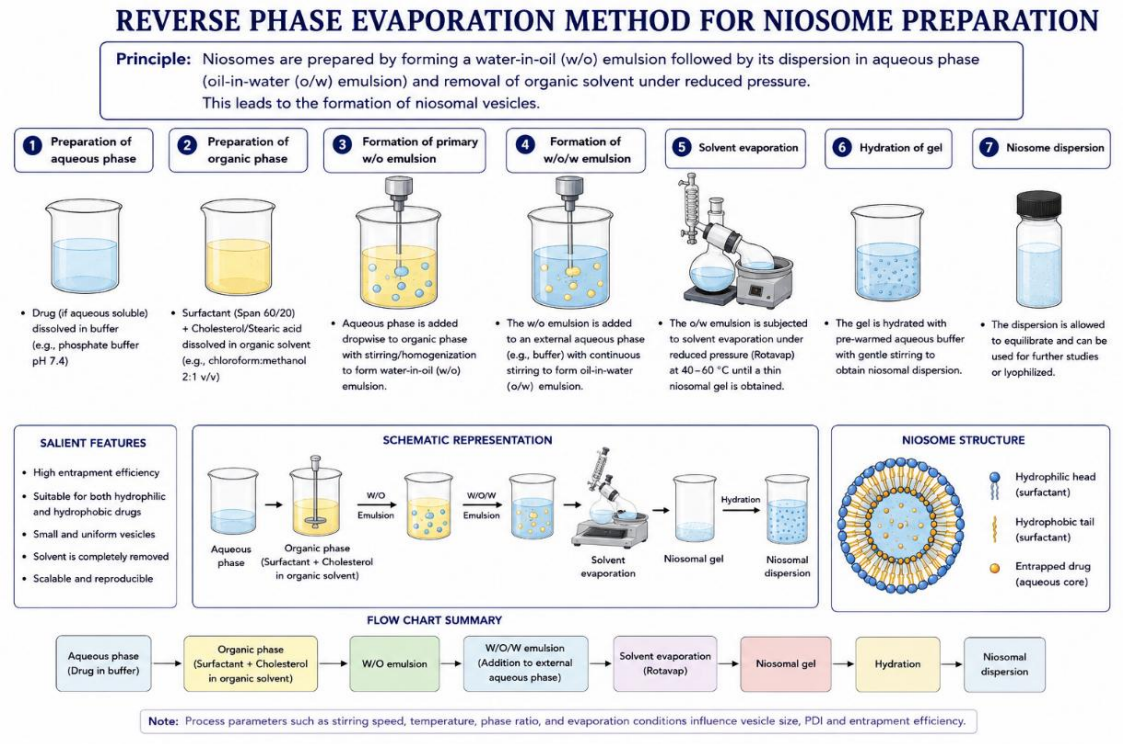

## 2. Hand shaking method

In this method the vesicle forming agents, that is cholesterol and surfactant are dissolved in volatile organic solvent such as methanol, diethyl ether or chloroform in a round bottom flask. The solvent is then evaporated using rotary evaporator which leaves a thin film of solid mixture on the wall of the flask. This dried film of surfactant is hydrated using an aqueous solution of drug with gentle agitation giving milky niosomal dispersion. This is one of the most common methods forming multi lamellar niosomes.



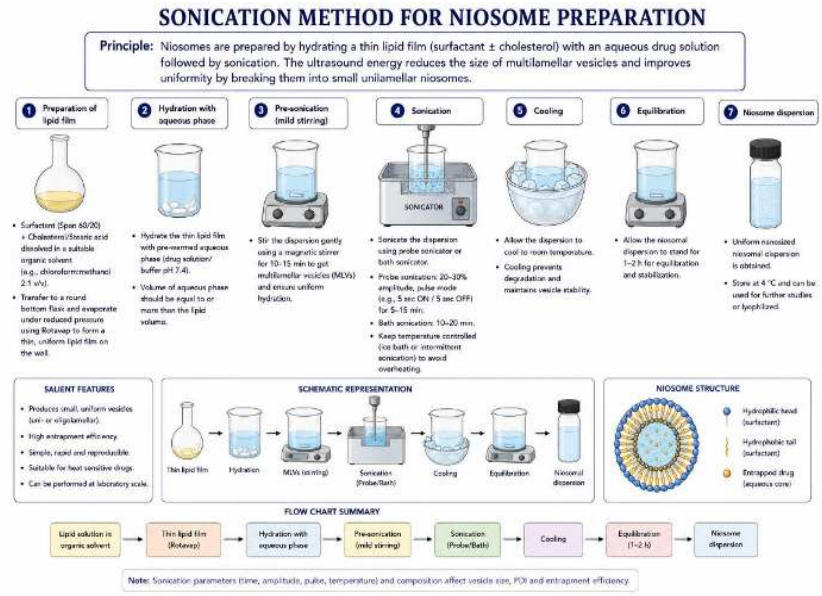
### 3. Reverse phase evaporation method

In a mixture of organic solvent (ether and chloroform), equal ratios (1:1) of cholesterol and surfactant is dissolved. To the above phase an aqueous phase containing the drug is added giving a mixture of two phases. This phase is sonicated at 4-5°C to form a clear gel which is further sonicated after the addition of phosphate buffer saline. The organic phase is removed using rotary vacuum evaporator at 40°C and at reduced pressure. This results in a viscous suspension of noisome which is diluted with phosphate buffer saline at 60°C for 10 minutes to yield niosomes.



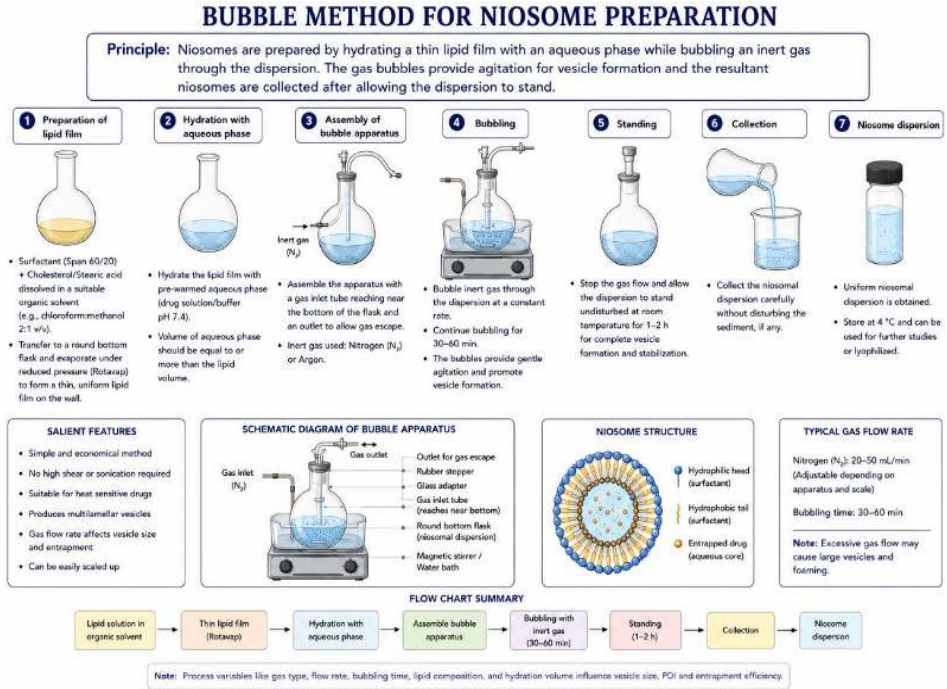

### 4. Sonication

An aqueous solution of drug in buffer is added to the mixture of surfactant and cholesterol. This mixture is added to a 10ml glass vial which is probe (titanium probe) sonicated for 3minutes at 60°C to yield niosomes. This method is also used to form SUV's from MLV's.



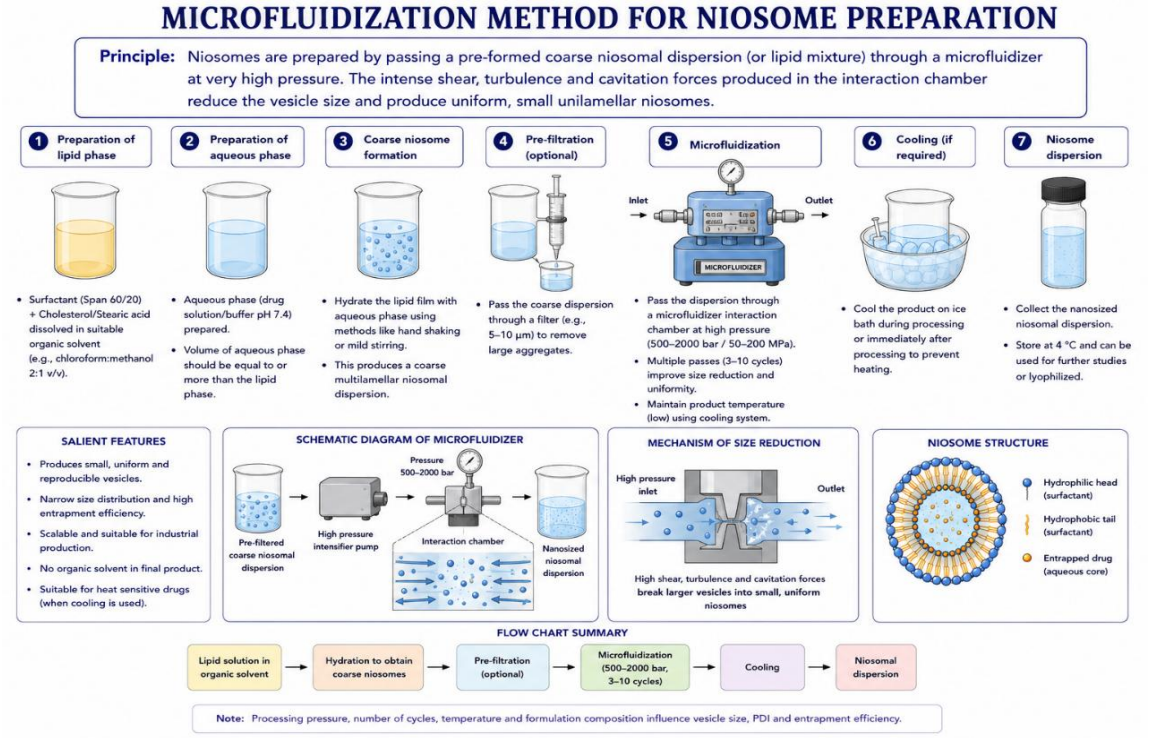

## 5. Bubble method

This method has the advantage of preparing niosomes in one step without the use of organic solvent. In this method, all the vesicle forming components were dispersed in aqueous solution of drug and mixed for 15 sec using a high shear homogenizer. This homogenous dispersion was placed in a round bottom flask which had three necks. First neck was water cooled reflex. Second neck was thermometer to check the temperature and third neck was to provide the nitrogen supply. After homogenization the dispersion was immediately bubbled using a continuous stream of nitrogen gas bubbles giving niosomes, having mean particle size between 0.2 and 0.5 µm.



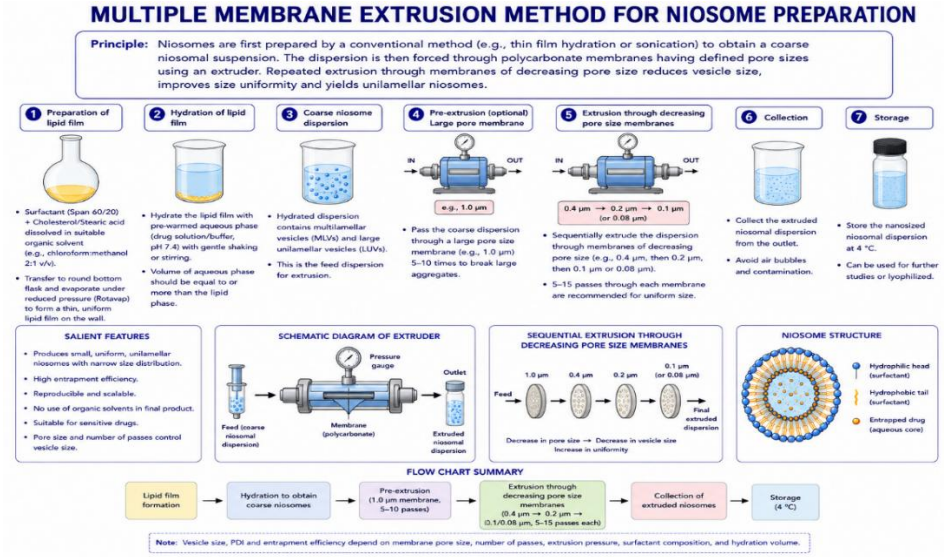

## 6. Micro fluidization method

In this method two fluidized streams were made to interact at high velocities in micro channels within an interaction chamber. Due to the effect of high speed and energy small uniform uni-lamellar niosomes were formed. This was known as submerged jet principle which was found to have better reproducibility as compared to other methods.



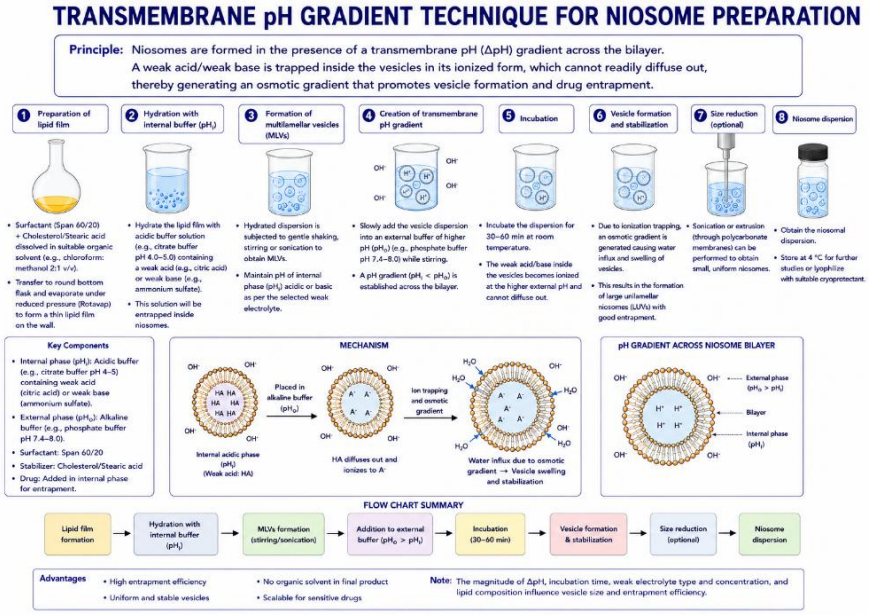

## 7. Multiple membrane extrusion

A mixture of surfactant, cholesterol and di-acetyl phosphate was dissolved in chloroform after which the solvent was evaporated using rotary evaporator leaving a thin film. This film was then hydrated using an aqueous solution of drug. The suspension formed was then extruded through polycarbonate membrane which was placed in series for up to 8 passages giving niosomes.



## 8. Transmembrane pH gradient technique

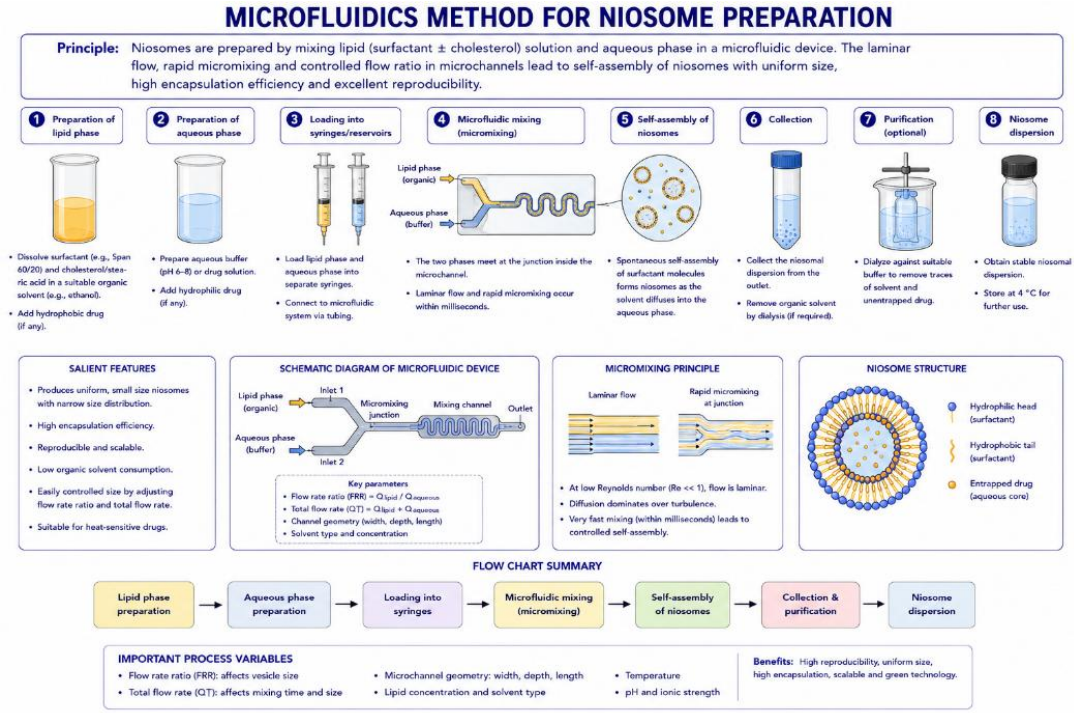
In this technique the surfactant and cholesterol placed in a round bottom flask and dissolved using an organic solvent such as chloroform. The solvent evaporated under reduced pressure thereby leaving a thin film on the walls of the flask. This film was hydrated by vortex mixing using citric acid having pH 4.0 which leads to the formation of multi lamellar vesicles. These vesicles were then frozen and thawed 3 times after which they were subjected to sonication thus forming niosomal suspension to which an aqueous solution of drug was further added. The pH of the aqueous drug sample was then increased to 7-7.5 using a base, usually disodium hydrogen phosphate so that a pH gradient was created across the niosomal membrane. In order to obtain niosomes this mixture was heated for 10 minutes at 60°C. The neutral pH gave mixture of protonated (membrane impermeable) and un-protonated (membrane permeable) forms of drug of which the un-protonated form of the drug crossed the niosome membrane and became protonated after entering the acidic medium thus getting trapped into the vesicle. This diffusion across the bilayer continued until the interior and exterior concentrations of drug attained equilibrium.




## 9. Microfluidics Method

The microfluidics method is gaining interest as a novel production technology for nanoparticles, particularly niosomes. The idea of the microfluidics method originated in 1947 and the first use of microfluidic channels was in the 1990s. The adoption of microfluidic technology also occurred in the 1990s. The production of niosomes can be carried out using microfluidics devices. It consists of side channels alongside central channels. Both the organic and aqueous phases are pumped in different channels under pressure at a specific flow rate to an ice-filled interaction chamber. The niosomes are usually collected from the mixing channels using a glass vial. Uniform, small size, and cholesterol-niosomes are produced using this method. Based on Noelia et al. (2020), the niosomes produced by the microfluidics method are smaller in diameter compared to those produced by the thin film hydration method. Other advantages that can be observed when using the microfluidics method are as follows: the control of the input variables, minimal chemical usage, and

the possibility of scaling up noisome production. Microfluidics enables the controlled synthesis of small and uniform niosomes through precisely monitored flow rates of aqueous and organic phases in microchannels. This technique is perfect for generating vesicles with minimum dispersity in size and cholesterol-free formulations, which are important for targeting active drug delivery. The flow rate ratio directly affects particle size and stability. Recent developments have shown great promise regarding the encapsulation of biomolecules such as mRNA and siRNA, making the technique highly relevant to gene therapy.

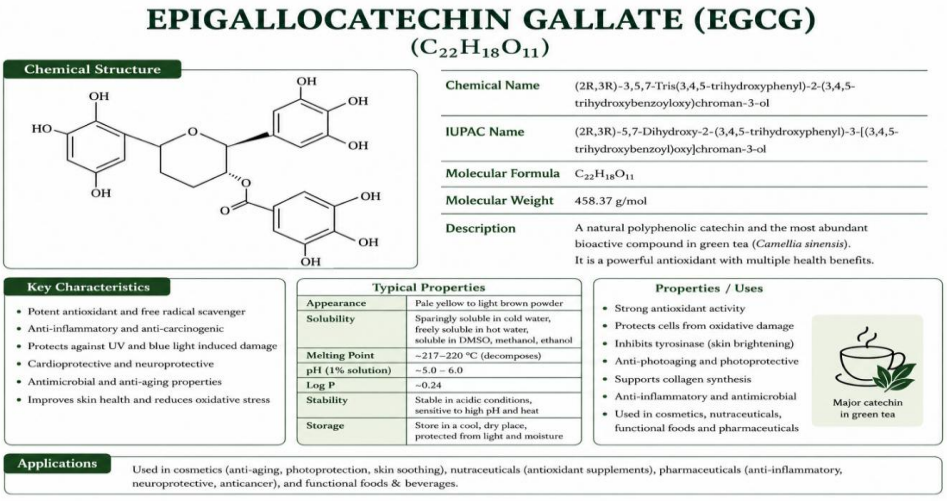



## 1.8 Major Active Marker Compound

**Epigallocatechin gallate (EGCG)** is considered the principal active constituent.

Approximate concentration:

- 40–60% of total catechins.



### 1.8.1 Pharmacological Activities

Green tea possesses several pharmacological properties:

- Antioxidant
- Anti-inflammatory
- Anti-photoaging
- Anti-wrinkle
- Anti-melanogenic
- Anti-acne
- Antimicrobial
- Anti-carcinogenic
- UV protective
- Blue-light protective
- Free radical scavenging
- Collagen protective
- Anti-aging

### 1.8.2 Mechanism of Action

Green tea polyphenols act through multiple mechanisms:

- Scavenges reactive oxygen species (ROS)
- Inhibits lipid peroxidation
- Suppresses inflammatory cytokines
- Reduces oxidative stress
- Protects collagen and elastin
- Inhibits matrix metalloproteinases (MMP-1 and MMP-9)
- Activates endogenous antioxidant enzymes such as superoxide dismutase (SOD) and catalase.[29]

### 1.8.3 Role in Anti-Blue Light Cosmetics

Green tea protects skin from blue light exposure by:

- Reducing intracellular ROS production
- Preventing oxidative DNA damage
- Protecting mitochondria
- Reducing inflammation
- Preventing premature aging
- Maintaining skin barrier integrity
- Decreasing melanin overproduction

### 1.8.4 Advantages in Niosomal Formulation

Green tea is suitable for niosomal delivery because:

- Catechins have poor skin penetration in conventional formulations.
- Niosomes improve encapsulation of polyphenols.
- They enhance stability against oxidation.
- They provide sustained drug release.
- They improve skin penetration and retention.
- They reduce degradation of EGCG.
- They increase bioavailability.

### 1.8.5 Therapeutic Uses

- Anti-aging creams
- Anti-wrinkle gels
- Sunscreens
- Anti-pigmentation products
- Acne formulations
- Skin-brightening cosmetics
- Wound healing formulations
- Antioxidant skincare
- Blue-light protection formulations

### 1.8.6 Storage

Store in:

- Well-closed, airtight container
- Cool, dry place
- Protected from direct sunlight and moisture
- Temperature below 25°C

### 1.8.7 Safety Profile

Green tea extract is generally considered safe for topical use.

Possible adverse effects:

- Mild skin irritation (rare)
- Allergic reactions in sensitive individuals
- Oxidation on prolonged exposure to air and light if not properly formulated.

### 1.8.8 EGCG DETAILS

The major active constituent of green tea, Epigallocatechin gallate (EGCG), has

| Property | Value |
|---|---|
| Chemical name | (−)-Epigallocatechin-3-gallate (EGCG) |
| Molecular formula | $C_{22}H_{18}O_{11}$ |
| Empirical formula | $C_{22}H_{18}O_{11}$ |
| Molecular weight | 458.37 g/mol |
| Drug | Green tea extract |
| Empirical formula | Not applicable (complex herbal extract) |
| Major active constituent | EGCG |
| Molecular/Empirical formula of EGCG | $C_{22}H_{18}O_{11}$ |

### 1.8.9 Mechanism of Action

EGCG exerts its pharmacological effects by:

- Neutralizing free radicals through hydrogen donation.
- Activating the **Nrf2/ARE** antioxidant pathway, increasing antioxidant enzymes such as superoxide dismutase (SOD), catalase, and glutathione peroxidase.
- Inhibiting the **NF-κB** signaling pathway, thereby reducing inflammation.
- Suppressing matrix metalloproteinases (MMPs), which protects collagen and elastin from degradation.
- Reducing lipid peroxidation and DNA damage caused by oxidative stress.
- Modulating signaling pathways such as **MAPK**, **PI3K/Akt**, and **AMPK**.

**Relevance to Anti-Blue Light Niosomal Gel**

For a **green tea-loaded niosomal gel**, EGCG:

- Protects skin cells from blue light-induced oxidative stress.
- Reduces intracellular ROS generation.
- Prevents premature skin aging and wrinkle formation.
- Helps maintain collagen integrity.
- Reduces hyperpigmentation by decreasing oxidative stress and inflammation.
- Enhances skin protection when delivered via niosomes due to improved stability and penetration.

### 1.9 EVALUATION PARAMETERS:

**1. Drug content**

Niosomal suspension equivalent to 10mg taken in a volumetric flask of 100ml & volume was made up by phosphate buffer pH 7.4[56], after that 1ml of this mixture was diluted to 10ml by phosphate buffer 7.4 & the % drug content was calculated or observed at using UV spectrophotometer.

**2. Drug Entrapment efficiency**

Entrapment efficiency of niosomes was determined by exhaustive dialysis method. The measured quantity of niosomal suspension was taken into a dialysis tube to which osmosis

cellulose membrane was securely attached on one side.[57] The dialysis tube was suspended in 100ml phosphate buffer (pH 7.4), which was stirred on a magnetic stirrer. The unentrapped drug was separated from the niosomal suspension into the medium through osmosis cellulose membrane. At every hour entire medium (100ml) was replaced with fresh medium (for about 4-5h) till the absorbance reached a constant reading indicating no drug is available in unentrapped form.[57] The niosomal suspension in the dialysis tube was further lysed with propane-1-ol and estimated the entrapped drug by UV spectrophotometric method at 274nm. The entrapment efficiency was calculated using following equation. Amount of entrapped drug.

$$\% \text{ Entrapment Efficiency} = \frac{\text{Total amount of Drug - Free amount of Drug}}{\text{Total amount of Drug}} \times 100$$

### 3. Zeta potential

The zeta potential is an essential parameter in the characterization of the colloidal stability of systems such as niosomes. It represents the difference in electrical potential between the particle surface and the diffuse layer of ions surrounding it, known as the electric dual layer. This potential, expressed in millivolts, indicates the degree of electrostatic repulsion between particles with similar surface charges. High values (positive or negative) suggest greater stability, as they decrease the probability of niosome aggregation or flocculation, which is crucial for maintaining their structural integrity and functionality in pharmaceutical or biomedical applications.

### 4. Polydispersity Index (PDI)

Polydispersity index (PDI) is a measure of heterogeneity in particle size within a sample of niosomes. This parameter is derived from DLS analysis and provides information on the size distribution present. A PDI close to 0 indicates a monodispersed sample, while higher values (>0.3) reflect greater variability in vesicle size. In physicochemical terms, polydispersity can also be interpreted as the ratio between average molecular weight by mass (Mw) and average molecular weight by number (Mn), indicating the structural uniformity of the system in niosomal formulations, a low PDI is desirable to ensure controlled and reproducible release of the active ingredient.

### 5.Fourier Transform Infrared Spectroscopy (FTIR)

Fourier transform infrared spectroscopy (FTIR) is a useful technique for structural characterization of niosomes, especially for confirming the presence of functional components such as surfactants, cholesterol, and encapsulated drugs. This technique operates in the infrared region of the electromagnetic spectrum, corresponding to wavelengths of 8 × 10-5 to 1 × 10-2 cm (energies of 4.6 to 46 kJ/mol). By applying the Fourier transform, a signal from the time domain is converted into a representation in the frequency domain, facilitating the identification of characteristic bands of molecular vibrations. Only vibrations that cause a change in the molecular dipole moment are active in IR, allowing the detection of specific functional groups present in the nosomal formulation .

### 6. In Vitro Drug Release

It can be determine by membrane diffusion technique. A dialysis sac is washed and soaked in distilled water. The vesicle suspension is pipetted into a bag and sealed. The bag containing the vesicles is placed in 200ml of buffer solution with constant shaking at 25 c or 37 c. [56]At various time

intervals, the buffer is analyzed for the drug content by an appropriate assay method.

**7.Stability Studies**

Optimized formulation preserved at refrigerated temperature & room temperature for 30days. After 30days shape, % drug remaining & % entrapment efficiency of vesicles were measured. The results were compared with the initial shape, % drug remaining & % entrapment efficiency of both samples.

**2.0 Conclusion:**

The literature reviewed demonstrates that green tea (*Camellia sinensis*) extract is a promising natural ingredient for topical dermatological and cosmeceutical applications due to its potent antioxidant, anti-inflammatory, and photoprotective properties, primarily attributed to catechins, especially epigallocatechin-3-gallate (EGCG). These bioactive compounds effectively neutralize reactive oxygen species, reduce oxidative stress, and protect skin cells from environmental damage. However, the therapeutic potential of green tea extract is limited by poor physicochemical stability, susceptibility to oxidation, and inadequate penetration through the stratum corneum. Several studies have demonstrated that niosomes, composed of non-ionic surfactants, are effective vesicular carriers capable of enhancing the stability, encapsulation efficiency, controlled drug release, and skin permeation of bioactive compounds. These advantages make niosomes a promising delivery system for improving the topical efficacy of herbal extracts. Despite significant advances in niosomal drug delivery, most studies have focused on antioxidant activity, wound healing, or ultraviolet (UV)-induced photodamage, whereas research specifically targeting blue light-induced skin damage remains limited. Therefore, the development of a green tea extract-loaded niosomal gel represents a promising approach to improve topical delivery and provide enhanced protection against blue light-induced oxidative stress, inflammation, photoaging, and other environmentally induced skin damage.

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