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A Breif Review on Formulation of Sunscreen from Nelumbo Nucifera Rhizome Extract

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ABSTRACT
The last chapter in the monograph on over-the-counter (OTC) sunscreen products. The term "sun block" will no longer be used on sunscreen labels, and there will be three levels of sun protection: minimum, moderate, and high. There will also be a new SPF category of 30+ for products with SPF values higher than 30, uniform, and streamlined labelling for all sunscreens. A small investment in prevention resulted in significant cost reductions from disease. The FDA has published its final directives for the sunscreen labelling. The definitive monograph revises the preliminary.

Nelumbo nucifera is one of two existing species of aquatic plants in the family Nelumbonaceae, sometimes known as sacred lotus, Laxmi lotus, Indian lotus, or simply lotus. It has been recorded in the most famous medicinal book in China for more than 400 years. Different part of plant (leaves, seeds, flower, and rhizome) can be used in traditional system of medicine. In traditional system of medicine, the different parts of plant is reported to possess beneficial effects as in for the treatment of pharyngopathy, pectoralgia, spermatorrhoea, leucoderma, smallpox, dysentery, cough, haematemesis, epistaxis, haemoptysis, haematuria, metrorrhagia, hyperlipidaemia, fever, cholera, hepatopathy and hyperdipsia. The pharmacological studies have shown that N.nucifera posseses various notable pharmacological activities like anti-ischemic, antioxidant, anticancer, antiviral, antiobesity, lipolytic, hypocholestemic, antipyretic, hepatoprotective, hypoglycaemic, antidiarrhoeal, antifungal, antibacterial, anti-inflammatory and diuretic activities[1]. The antioxidative ability of rhizome (Lotus roots) knot (LRK) and whole rhizome (LR) extracts was examined in comparison to commonly utilised antioxidants derived from plant material. Using the stable radicals 1-diphenyl-2-picrylhydrazyl and 2,20-azino(3-ethylbenzothioazolino-6 sulfonate), the activity of radical scavengers was evaluated spectrophotometrically. measured by the transient carbon-centered 1-hydroxyethyl radical (produced in a Fenton-type reaction) being trapped by electron spin resonance (ESR).[2]

I. INTRODUCTION

Common names for Nelumbo nucifera include lotus and sacred lotus. It is a perennial aquatic herb that is a member of the Nelumbonaceae family [3, 4]. The plant's roots continue to be rooted in the muddy water body bottoms. The leaves have a diameter of 60 cm and float on the water's surface. While the water chinquapin, N.lutea, is present in eastern and southern North America, the lotus, N.nucifera, is spread throughout Asia and Australia [5]. The aquatic plant species N. nucifera needs a lot of room and direct sunlight to grow and thrive. In India, it is frequently referred to as Kamala or Padma. The rhizomes of the lotus plant are thick, creeping, and yellow; the fruits are green. Typically, leaves are big and have both[6]This plant is not only opulent and beautiful, but it also contains powerful astringent and cooling characteristics, making it an excellent source of herbal medicine. In South East Asia, where its seeds and leaves are often consumed, the lotus has significant religious significance. Because of this, it is called a sacred lotus [7]. The by-products of lotus seed processing, as well as the seeds themselves, are widely consumed throughout Asia, Oceania, and America. There are a lot of physically active ingredients in it [8]. Due to the presence of polyphenols in N. nucifera, this plant has antioxidant activity that is advantageous to many different aspects of health [9, 10]. In China, dried, shelled lotus seeds are the most common forms of sale. These contain several alkaloids, including neferine, etc. These are occasionally offered as a raw snack option [11]. This The ability to control the temperature of the blossom within a specific range is one of this plant's unique characteristics

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[12]. Long viability duration is a feature of ts seeds [13].

History

Early synthetic sunscreens were first used in 1928. The first major commercial product was brought to market in 1936, introduced by the founder of L'Oreal, French chemist Eugène Schueller. The earliest form of sunscreen was created by Franz Greiter in 1938 and then Benjamin Green in 1944 who used a mixture of cocoa butter and red veterinary petroleum to protect his skin from the sun. Shortly afterwards, Franz Greiter branded his formula Piz Buin while Mr. Green marketed his as Coppertone Suntan Cream. In the United States, one of the first sunscreen products to become popular was invented for the military by Florida airman and pharmacist Benjamin Green in 1944. This came about because of the hazards of sun overexposure to soldiers in the Pacific tropics at the height of World War II(14,15) . © 2021 JETIR June 2021, Volume 8, Issue 6 www.jetir.org (ISSN-2349-5162) JETIR2106263 Journal of Emerging Technologies and Innovative Research (JETIR) www.jetir.org b860 Franz Grieter also credited with the term "Sun Protection Factor," better known as "SPF." Greiter was climbing an Appalachian mountain range when he was burnt to a crisp by the brutal UV rays(16). Subsequently, the relationship between UV light and skin ageing as well as skin cancer was investigated in more detail; for instance, through the development of a photoageing concept by Albert Kligman in 1986, or the relationship between tanning and development of skin cancer reported by the WHO in 2007.Clothing, scarves, and shade were early methods of protecting skin from the sun(3). However, applying products to the skin for additional protection also .

0the skin from sun damage. Health experts advise everyone, regardless of skin color, to use sunscreen with an SPF of at least 30. Although dark-skinned people won't get sunburned as quickly, they will still burn and are still susceptible to sun-induced damage—such as sun spots and wrinkles—and cancer(17)

Role of Sunscreens in Photoaging

The concept of a topical photoprotective product has been around since the times of the ancient Egyptians in 4000 BC, but the first commercial sunscreens were not available until the 1920–1930s [18,19]. At that time, understanding of UV radiation was limited and focused mainly on UVB protection. With the increasing popularity of sunscreen over the years, the concept of standardization of photoprotection against UVB was introduced [18]. SPF was recognized by the FDA in 1978 as the standard for measuring sun protection [18].

UV-induced erythema is mostly attributed to UVB, with a minor contribution by UVA2. The concept of SPF, an assessment using UV-induced erythema as an endpoint, as a sole measurement of sun protection persisted for many decades despite advances in the study of UVR suggesting that UVA may play a significant role in photoaging [18, 20, 21]. In 1992, the UVA star rating system was created by The Boots Company in the UK but was not widely implemented [18]. Although other methods of evaluating the efficacy of UVA filters have been proposed, the FDA currently uses critical wavelength (CW) determination. With this method, sunscreen products whose 90% UV absorbance occurs at ≥ 370 nm are allowed to be labeled as "broad spectrum" [22]. In Europe, the International Organization Standardization 24443 guidelines use a minimum ratio of UVA protection factor to SPF of 1:3 for all marketed sunscreens [23]. In a study of 20 sunscreens tested against the FDA guidelines and the ISO 24443 guidelines, 19 of 20 sunscreens met the CW requirements set by the FDA, whereas only 11 of 20 sunscreens met the ISO 24443 standard [22]. To address this disparity, the FDA proposed a new rule on sunscreens in 2019 that specifically highlighted a requirement for a UVA1 (340–400 nm) to UVA and UVB (290–400 nm) ratio of ≥ 0.7; however, the FDA has not yet made a final decision [24]. Clearly, there exists further need for global standardization to help protect and guide consumers.

In recent years, tinted sunscreens have become more prevalent as a means of protection against VL. Most FDA-approved compounds for UV protection do not adequately protect against VL because compounds must be opaque to filter VL [25]. Zinc oxide and titanium dioxide can protect against VL but only when they are pigmentary grade and not micronized. Tinted sunscreens incorporate combinations of iron oxides and pigmentary titanium dioxide to offer VL protection and utilize the different colors of iron oxides and pigmentary titanium dioxide to improve color match on people of all Fitzpatrick skin types [25, 26]. It should be noted that iron oxides are not considered to be UV filters so are listed under "inactive ingredients" on sunscreen product

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packages, whereas pigmentary-grade titanium dioxide and zinc oxide are FDA-approved inorganic filters. However, the exact efficacy of specific tinted sunscreens for VL protection has been largely unregulated as no standards or guidelines for VL protection yet exist. A method for VL protection factor has been recently suggested using in vivo assessment in melano-competent subjects [32, 27].

There is good evidence that daily photoprotection and daily sunscreen use plays an important role in the prevention of photoaging [28,29]. In a study of 46 patients randomly selected to use vehicle or sunscreens with UVA and UVB protection daily for 24 months, a significant histological difference in solar elastosis was observed in the vehicle versus treatment group [30]. Furthermore, in a study of 12 subjects in which each subject was exposed to one minimal erythemal dose of simulated solar radiation to three areas of buttock skin (unprotected skin, vehicle, and day cream with UVA and UVB protection) and control (no exposure), the unprotected skin demonstrated significant melanization, increased stratum corneum and stratum granulosum thickness, elevated expression of tenascin, reduced type I procollagen, and slightly increased lysozyme and alpha-1 antitrypsin, which were all mitigated by the day cream–sunscreen combination [31]. Not only have sunscreens been shown to prevent photoaging but evidence also suggests that they may play a role in the reversal of extrinsic aging. In a prospective study, 32 subjects were asked to apply daily broad-spectrum photostable sunscreen (SPF 30) for 52 weeks. At the end of the study, significant improvements in skin texture, clarity, and mottled and discrete pigmentation were observed, with 100% of subjects showing improvement in skin clarity and texture [31]. However, further research into the molecular mechanism of sunscreen's effects on the reversal of chronologic aging must be performed.

Effect of UV radiation on skin

UV rays can enter the skin and interact with keratinocytes and fibroblasts, two types of skin cells. Senescence-associated secretory phenotype, which is secreted by senescent cells, includes cytokines, chemokines, growth factors, and matrix metalloproteinases (MMPs). The kind and dosage of UV light affect how premutagenic photoproducts develop. While 8-hydroxy-2-deoxyguanine (8-OHdG) is one of the most popular indicators for the estimation of DNA damage from UVA, cyclobutane dimers Py (CPD) are mostly induced by UVB.

One of the most harmful effects of prolonged exposure to UV radiation on the skin is DNA damage, which also directly contributes to photoaging and photocarcinogenesis. The quantity of energy absorbed by base pairs in the DNA chain determines the different damaging mechanisms that UVB and UVA have on DNA molecules.[32] Direct UVB radiation exposure to cellular DNA causes distinctive changes in the nucleic chain structure, such as the creation of CPD and pyrimidine base transversals.18 Like UVB, UVA radiation can cause DNA damage in the form of CPD, pyrimidine (6-4) pyrimidone photoproducts, damage to DNA bases, and base transitions.[33,34]

Through an inflammatory response and indirectly through the produced oxidative stress, UVA exposure harms skin cells directly. The development of the most prevalent and highly carcinogenic DNA adduct, 8-hydroxy-2'-deoxyguanosine (8-OHdG), which is regarded as a trustworthy marker for oxidative DNA damage, and peroxidation of polyunsaturated fatty acids (PUFA) in the epidermal membrane are both triggered as a result.[35]

Keratinocytes experience an inflammatory response when exposed to UVB rays on the skin, which activates the protein kinase R signal transduction pathway, blocking this signal transduction route. To shield the cell from UV rays, a long non-coding RNA called nc886 inhibits the signal transduction pathway involving protein kinase R.

Maintaining homeostasis in skin structures requires the intracellular cleaning system known as autophagy. When reproducible ageing of human facial fibroblasts occurs in the case of skin ageing, the fundamental degree of autophagy rises. Additionally, UVA and UVB both cause autophagy in fibroblasts [36] and human keratinocytes, respectively. The ageing reaction of these cell types can only be postponed by the autophagic process, which is not able to totally eliminate it.[37,38]UV-induced ROS generation encourages autophagy, which controls the body's response to oxidative stress brought on by sun radiation. Increased levels of oxidised phospholipids, oxysterols, and cholesterol in epidermal cells as a result of UVA exposure serve as a cue for keratinocytes to initiate autophagy. By eliminating oxidised molecules and reducing the antioxidative reaction in different cell types, autophagy plays many roles in the response to oxidative stress brought on by UVA radiation. It

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has been demonstrated that the autophagy-related gene adaptive protein p62, as well as the autophagic activators p53 and Sestrin2 (SESN2), which can trigger autophagy through 5' adenosine monophosphate-activated protein kinase (AMPK) signalling, are regulated by UVA.[29]Endo et al.'s experiment (2020) revealed that repetitive UVA radiation adversely impacts the autophagy process in fibroblasts because of changes in lysosomal function.30 The molecular processes driving defective autophagy, such as decreased lysosomal acidity and lower expression of cathepsins B, L, and D, prevent intracellular breakdown in UVA-treated fibroblasts. This shows that the primary cause of skin photoaging is an abnormality in the autophagic mechanism. However, it is still unclear what is causing the recurrent UVA radiation fibroblasts to have lysosomes that are fundamentally defective.

Sunburn cells (SBC), which are keratinocytes that undergo apoptosis, are formed in the epidermis as a result of repeated exposure to UVB light.[40] Keratinocyte damage UVB damage to DNA causes signals to be released, which in turn triggers the production of inflammatory response mediators like the cytokines IL-1, IL-6, and TNF-.33 The UVRAG and p53-associated gene AMPK autophagy activator is directly induced by UVB. Starts the transcription of AMPK, SESN2, Tuberous Sclerosis Complex 2 (TSC2), and UVRAG to activate autophagy after being stabilised by UVB p53.[39]

Advantage of herbal extract sunscreen

1. Easily available.
2. No side effect.
3. No special equipment needed for preparation.
4. Renewable resources.
5. Botanical ingredients are easily available.
6. They are inexpensive.

II. CONCLUSION

A crucial part of sun protection is the application of sunscreen. UV radiation exposure is linked to a lower risk of a number of skin problems and malignancies when used regularly and appropriately. Patients also need to be warned not to rely only on using sunscreen. Consequently, it might be saidthat there is a large market for sunscreen chemicals, whether they are synthetic, natural, or a combination of both, since people are more conscious of the need to protect themselves from harmful UVA and UVB rays.

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