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POTENTIAL OF MEDICINAL PLANTS IN VITILIGO MANAGEMENT: A REVIEW ON PHYTOCHEMICAL STRATEGIES: A NARRATIVE REVIEW

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ABSTRACT: Vitiligo is a skin condition that cause skin to lose color as melanocytes die. While pathogenesis of vitiligo remains not understood, factors such as oxidative stress, cellular response, immune response have been identified as key contributors to melanocytes destruction. Conventional treatments, including synthetic drugs like corticosteroid, vitamin D3 analogue, immune suppressants, and phototherapy, are often associated with side effects and varying efficacy. Ongoing research has demonstrated the potential of herbal remedies like alternative or adjunctive therapies in managing vitiligo. Several plant-based compounds, including flavonoids, antioxidants, and other bioactive molecules, have shown promise in modulating immune responses, reducing oxidative stress, and promoting melanocytes regeneration. Herbal drugs, such as Ginkgo biloba, curcumins melo, are being explored for their potential benefits in treating vitiligo. Moreover, vitamin supplements, particularly those rich in antioxidants like vitamin D, C, AND B12, have been suggested to support the therapeutic process by enhancing skin health and protecting melanocytes from oxidative damage. These abstract aims to review the current understanding of vitiligo's pathogenesis, the role of herbal treatments, and their comparison to synthetic drugs, highlighting the potential benefits and challenges of integrating herbal medicine into vitiligo management. To conduct this review, an extensive literature search was conducted using relevant publication published and indexed in Google scholar, Elsevier and science direct were studied.

INTRODUCTION: In the course of second century the word Vitiligo was first used by census [1]. Vitiligo is the most typical ailment of blanched or whitened, it is global wide spread presence vary from 0.06 – 2.28% [2, 3]. It is distinguished by the non-appearance of pigment in the skin, mislaying of melanocyte [4].

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Melanocytes are present in various tissues such as hair follicles, skin, eyes, inner ear, heart brain, *etc* [5]. Melanocytes provides an essential function in the complex process called melanogenesis, as it creates and spreads melanin [5-8]. Melanogenesis is detect congenitally but it is affected by external as well as internal factors [9].

Though the precise trigger of vitiligo is unknown, an autoimmune theory is intriguing and backed by a number of data [10]. A higher amount of hydrogen peroxide in vitiligo patients and a higher level of superoxide dismutase pursuit support the biochemical concept that the damage to

melanocytes is caused by a variation in oxidative stress [10]. Another view is the melanocytorragy theory, which holds that damage cell binding results in trans epidermal loss of melanocytes with autoantigen disclosure and immune system stimulation, causing melanocyte damage or injury [11]. Ultimately, the confluence theory declare that the combination of various pathway is important for development of vitiligo including genetic background, vernability to environmental change, altered epidermal microenvironment, intrinsic melanocyte defect and an autoimmune response [12, 13].

Epidemiology: Vitiligo effect about 0.5%-2% of population in all over the world and the preference has all the lay side of being equivalent among females and males, children are influenced similarly, non-segmental vitiligo grows in every age, but commonly occurs between the age of 10 years and 30 years. Although, detecting from one epidemiological study show that at the age of 40 years, the 50% of people develop vitiligo. However vitiligo can occur at any ages, it is increasing in vigorous and moderately aged people. An examination detailed that the mean age was 18.9 [14].

Pathophysiology of Vitiligo:

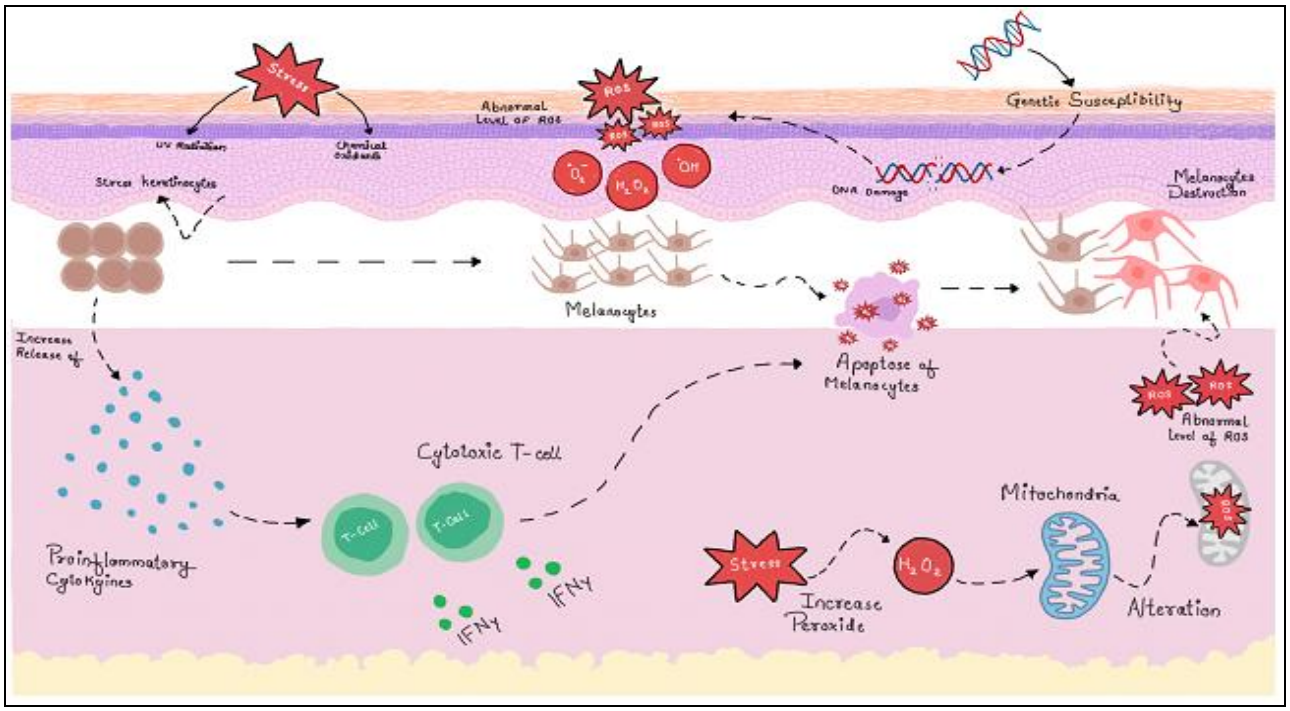


FIG. 1: PATHOPHYISIOLOGY OF VITILIGO

The diagram represents the pathogenesis of vitiligo, a skin condition distinguished by the loss of pigment cells (melanocytes).

Fundamental of the diagram: **Fig. 1**.

TRIGGERS:

Phenolic Compound: Some chemicals can destroy melanocytes

Trauma: Physical injury can trigger the onset of vitiligo.

Ultraviolet Radiation: Too much sun exposure can destroy melanocytes

Oxidative Stress:

Increase in Superoxide Dismutase: By increasing superoxide dismutase, the body's antioxidant defense system tries to prevent reactive oxygen species.

Decrease Catalase and Glutathione: Due to imbalance in the redox status, there is decrease in antioxidant enzymes such as catalase and Glutathione.

Cellular Response:

FOXO3A Polymorphism: Genetic diversity in the FOXO3A gene can affect the vulnerability to oxidative stress and vitiligo. Increase intransient

receptor potential melastatin 2: This Channel Protein associated in calcium influx causes further cellular stress and damage.

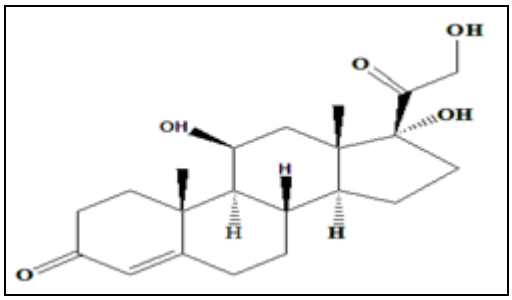
Autophagy and Apoptosis: The melanocyte which are Destroyed undergo either self- digestion and programmed cell death.

Immune Response: Damage- Associated Molecular patterns: The melanocyte which is died produce DAMPS, such as HSP70 which activate the immune system.

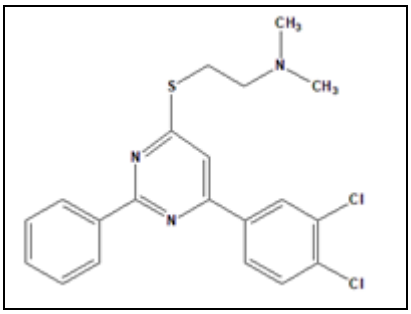
Innate Immunity: Immune cell such as natural killer cells are operate and release inflammatory cytokines like IL-6 and IL-8. Interferon Gamma: This cytokine also contributes to inflammation and help in melanocytes damage.

The Synthetic Drug which are used in Treatment of Vitiligo:

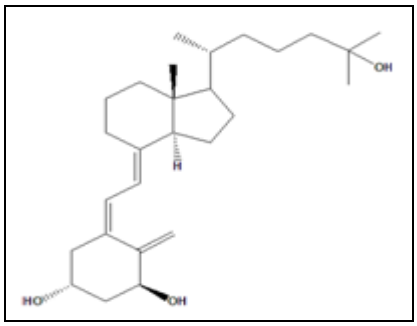
Corticosteroid: These cure vitiligo by regulating and eliminating the inflammatory like the first line drug of topical corticosteroid is used to treat vitiligo. It gives it therapeutic effect mostly in sun exposed area rather than acral zone.



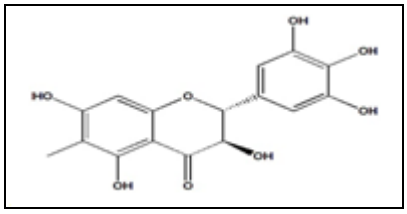
Calcineurin Inhibitors: It mostly targets the head and neck region of the body. It also involve tacrolimus and pimecrolimus in the concentration of 0.03% and 1%, which has fewer side effects and no chances of atrophy. It can be used twice a day, for an average 6 month. In this treatment, also sun exposed area is preferred for effectiveness.



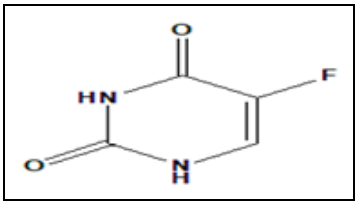
Vitamin D 3 Analogue: They not alone used in treatment of vitiligo; it involves used of supplement. When we apply ointment, the dosage is used for 4 weeks and when we apply cream, for 8 weeks, 30% of the body receives therapy owing toover100g each week of a mixture of 0.05% betamethasone and 0.005% calcipotriol [15].



Pseudocatalase / Superoxidedismutase: In vitiligo, oxidative stress and hydrogen peroxide play crucial role. In the epidermis of the wound there is an accumulation of high level of H2O2. These hydrogen peroxide deactivates the catalase by inhibiting the tyrosine, which are toxic to melanocytes [16]. By using topical superoxide dismutase gel, no observation is seen [17]. Alshiyab *et al.*[18] conducted a study that balance the effectiveness of topical superoxide dismutase gel and tacrolimus 0.1% ointment while treating children's localized vitiligo [19].



Fluorouracil (5-FU): Basically, premalignant and malignant skin lesion are treated mainly with the help of 5- gluorouracil. To treat vitiligo hyper pigmentation therapy is used with it [20]. The mechanism of action of it involves stimulation of follicular melanocytes with migration and also by increasing number of melanosomes in keratinocytes [21, 22].



Herbal Drugs: Herbal drug is those drug which are known as plant material or herbalism and obtain from the plant parts such as leaves, stem, etc to treat many diseases [23]. These are the oldest form of health care which are used by man from long time [24]. There are many herbal drugs which offer or promise to treat various diseases [25]. As herbs are obtained from natural resources, it is quite safe and effective than synthetic drug [26]. For the past few decades, herbal drugs are very popular as compare to allopathic medicine as it treat many diseases such as depression, anti-inflammatory, analgesic, hepatoprotective, *etc* [27].

List of Various Herbal Drug used in Vitiligo Treatment:

Using Capsaicin to Demonstrate Biologic and Toxic Effect and Treat Vitiligo: Capsaicin is obtained from fruit of capsicum plant, generally natural alkaloid having chemical formulatrans-8-methyl-N-vanillyl-6-nonenamide. The family of capsaicin include vanilloid, zingerone, eugenol [28]. Capsaicin showed a large number of altered cells with a biologic and poisonous effect on non-neuronal cells. This study sought to evaluate capsaicin's cytotoxic mode of action on human skin fibroblasts in culture. In order to quantify the change in cell survival, growth factor, receptor antagonist, and antioxidant were additionally pretreated with capsaicin with normal newborn human fibroblasts for the MTT experiment. EGF (10 ng/ml), bFGF (10 ng/ml), and capsazpine (10µm) all increased fibroblasts, but cycloheximide (1µg/ml) suppressed them. When 200µm capsaicin is given to fibroblasts, chromatin condensations were observed at 12 h, and cell survival rate was reduced to 25–50% at 24 h. Toxic effect of capsaicin is not prevented by vanilloid receptor antagonist, capsazepine and ruthenium red and its cytotoxicity is increased by 10µm capsazepine. In contrast to bFGF (10 ng/ml), EGF (10, 100ng/ml) enhanced the cytotoxicity of capsaicin. Neuropeptides, substance P(1,10nm) and CGRP (1, 10nm), and a structural analogue to capsaicin, tyrosine (0.3–1.2mm) did not affect the cytotoxicity. However, antioxidants Trolox (100µm) and ascorbic acid (0.1, 0.3 mm) reduced the capsaicin cytotoxicity. Of calcium- modulating agents, nifedipine, a Ca^{2+} channel blocker (10,20 µm) and cyclopiazonic acid, a Ca^{2+} -ATPase inhibitor in ER (10µm) did not influence the cytotoxicity, but BAPTA/AM as a chelator for cytoplasmic free calcium ion (10µm) significantly decreased capsaicin cytotoxicity. Unlike cycloheximide, a protein synthesis inhibitor, z-VAD-FMK, a non-specific caspase inhibitor, prevented the capsaicin cytotoxicity. The DNA ladder and TUNEL-positive cells were observed from the capsaicin-treated fibroblasts and Western blot revealed caspase-3 activity. Thus, capsaicin-induced cytotoxicity on human skin fibroblasts is more likely to suggest the mechanism of apoptotic pathway where antioxidants may play a role to prevent it [29]. Capsaicin is the active part of the chili peppers, plants of the genus capsicum. It provides anti-inflammatory and antioxidants property to treat vitiligo [30].

Using Green Tea Polyphenols to adapt immune response and Vitiligo Treatment: Since, ancient time, Green Tea Polyphenols are used as medicine which are extract from green tea leaves. Epigallocatechin -3- gallate, is the main composition of it that provides anti-inflammatory, anti-oxidant and immune modulator property [31]. Polyphenols can regulate the function of T cells, which play a crucial role in adaptive immune responses. While terpenoids and other natural compounds like alkaloids and glycosides also possess immunomodulatory properties, their mechanisms of action may be more specific or limited compared to polyphenols. Polyphenols are abundant in sources. As crucial secondary metabolites in plants, their widespread distribution is closely tied to the roles in plant physiology, encompassing defence mechanisms, antioxidants, and influencing plant coloration. This study investigates the potential role of polyphenolic compounds in treating vitiligo, a dermatological condition characterized by the loss of skin pigmentation, for which effective treatment is currently lacking. Renowned for their antioxidative and anti-inflammatory properties, polyphenolic compounds are believed to positively impact the pathogenesis of vitiligo by modulating multiple molecular targets and signaling pathways. This study systematically reviews the potential mechanisms by which polyphenolic compounds may affect the treatment of vitiligo, thereby providing a scientific basis for the development of novel therapeutic strategies and medications [32]. It can be taken systemically as well as topically [33]. It

is used to treat vitiligo by inhibiting oxidative stressof the melanocyte unit [34].

Curcumin: Inhibit Pro-inflammatory and Treat Vitiligo: It is a polyphenol derived from the *Curcuma longa* species. Curcumin is used to treatment diseases such as it provides anti -oxidant, anti - proliferative, anti-viral, anti- bacterial activity [35]. Moreover, curcumin may inhibit many pro-inflammatory molecules, including IFNγ, which play a critical role in the pathogenesis of vitiligo [36]. The ability of curcumin in inhibiting IFNγ was already demonstrated in psoriasis but not demonstrated in vitiligo [37], even if curcumin was able to minimize melanin content and tyrosinase activity in a dose-dependent manner via suppressing melanogenesis in an *in-vitro* model [38]. Curcumin's low bioavailability is the primary factor limiting its potential use. In order to improve its bioavailability, many experimental techniques are being used. For example, modified curcumin inhibited melanogenesis by triggering the extracellular signal-regulated protein kinase (ERK) pathway [39]. Supplementing with curcumin is an effective alternative treatment for anxiety and depression symptoms, enhancing the quality of life for people with long-term illnesses [40]. However, there is currently conflicting information regarding curcumin's benefits on vitiligo; consequently, more research is needed to advocate its usage in clinical practice for the treatment of vitiligo. Currently, nb-UVB with tetrahydrocurcumide cream is used to treat vitiligo. For the treatment of vitiligo phototherapy is also used twice a week for 12 weeks, which show better results than nb-UVB [41].

***Pyrostegia venusta*: An Antioxidant and Anti-Inflammatory Natural Vitiligo Treatment:** It is commonly found in southern Brazil, which are used topically to treat vitiligo. It is effective against antioxidant, anti -inflammatory, *etc* [42]. Botanical material and extract obtention Leave of *P. venusta* were collected in May 2013 in the city of Colombo (coordinates: S 25°19´31´´), Paraná, Brazil. The specie was identified by Osmar dos Santos Ribas from Municipal Botanic Museum of Curitiba (MBM). A voucher specimen was deposited at MBM in Curitiba, Paraná, Brazil. Leaves of *P. venusta* (100 g) were air dried (40°C, forced ventilation), powdered and extracted with ethanol: water (70:30, v/v) by turbolysis (Ultra Turrax Biotrona BA80, Kinematica AG) at room temperature for 2 h. HE was filtered through a filter paper (Adventecno.1) followed by solvent reduction to 1/6 of the initial volume under reduced pressure, lyophilized and kept at −20 °C. Before pharmacological assays, the lyophilized HE was dissolved in water or non-ionic cream to produce different pharmaceutical forms [43].

Animals: Experiments were performed on Swiss (female) and C56BL/6 (female and male) mice, weighing 25–35 g. All animals were housed at 22±2 °C under a 12 h light/12 h dark cycle, with access to food and water *ad libitum*. The animals were allowed to adapt to the laboratory for at least 48 h before testing. Swiss mice were obtained from Universidad Federal do Paraná (UFPR, Curitiba, Brazil). C56BL/6 mice were obtained from Universidad Federal do Paraná (UFPR, Curitiba, Brazil) and Universidad Federal de Santa Catarina (UFSC, Florianopolis, Brazil). Experiments were performed after approval by the Institutional Ethics of Universidad Federal do Paraná (Protocol number 738, 2013) and were carried out in a ccordance with the current guidelines for the care laboratory animals [44].

Quercetin as a Protective agent to treat Vitiligo: Quercetin is a polyphenolic flavonoid that occurs in fruits like apples and cranberries, vegetables like onions and asparagus, and herbs like dill, cilantro, and *Camellia sinensis* (black tea) [45]. In addition to its anti-cancer and neuroprotective effects, quercetin appears to be helpful in vitiligo patients [46]. Previous study demonstrated that quercetin can change the tyrosinase inhibition observed in human epidermal melanocytes and could potentially reduce oxidative stress-induced endoplasmic reticulum (ER) growth [47]. Like other flavonoids, quercetin additionally promotes melanogenesis by increasing intracellular tyrosinase activity [48] and melanin in melanoma cells in a manner that is dose-dependent [49]. Flavonoids affect melanogenesis by stimulating the MITF, and this helps in controlling the expression of a large number of melanogenic enzymes, including tyrosinase, dopachrome tautomerase (DTC, also known as tyrosine-related protein 2 or TYRP-2), and tyrosine-related protein 1 (TYRP-1) [50]. Quercetin may be useful in the treatment of mental diseases, as shown by Samad *et al.*, who evaluated

the effects of parenteral dosage in mice with anxiety and depression. In contrast to anxiety and depression, it not only enhanced memory retention but also regulated the serotonergic and cholinergic neuronal transmission of mice. Moreover, quercetin's antioxidant effects enable it to impact the modifications in the expression of heme oxygenase-1 (HO-1), protein kinase B (Akt), phosphoinositide 3-kinase (PI3K), and Nrf2 that occur in depression [51].

***Ginkgo biloba*: A Potential Vitiligo Treatment And has Anxiolytic Properties:** *Ginkgo biloba* has anxiolytic properties, which prevent the spread of vitiligo lesions [52].

Methods: 12 participants 12 to 35 years old were recruited to a prospective open-label pilot trial and treated with 60 mg of standardized *G. biloba* two times per day for 12 weeks. Among the requirements for sustainability were successful recruiting, 75% orgreater retention, effectiveness and lack of serious adverse reactions. Effectiveness was assessed using the Vitiligo Area Scoring Index (VASI) and the Vitiligo European Task Force (VETF), which are validated outcome measures evaluating the area and intensity of depigmentation of vitiligo lesions. Other outcomes included photographs and adverse reactions. Safety was assessed by serum coagulation factors (platelets, PTT, INR) at baseline and week 12.

Results: In order to make it easy to recruit the necessary sample size, the upper age restriction was reduced from 18 to 35 years old after two months of recruiting. Eleven participants was adhered to the protocol at least 85% of the time during the experiment performed. The overall VASI score, which ranges from 0 (no depigmentation) to 100 (full depigmentation), increased by 0.5 (P=0.021) from 5.0 to 4.5. Every participant's vitiligo stopped advancing, and the average repigmentation of vitiligo lesions was 15%, according to the total VASI. During baseline and week 12, the VETF total vitiligo lesion area dropped from 5.9 to 5.6 by 0.4% (P = 0.102). From 6.6 to 5.8, the VETF staging score increased by 0.7 (P = 0.101), and from 2.7 to -1.2, the VETF spreading score improved by 3.9 (P < 0.001). PTT, INR, or platelet count weren't affected in an extent that was statistically significant [52].

***Polypodium leucotomos*: Known for Photoprotective Properties:** The tropical fern *Polypodium leucotomos*, known as "Calaguala," which belonging to the Polypodiaceae family. Psoriasis, atopic dermatitis, or other [54, 55] skin disorders are treated by using its extracts, which are known for their multiple properties including photoprotective properties [53]. *Polypodium leucotomos* has been used as therapy for vitiligo patients receiving phototherapy in recent years. An intriguing study provides how oral *Polypodium leucotomos* combined with PUVA therapy increased repigmentation more than photo chemotherapy alone. Same results were obtained by a separate study using the combination of oral *Polypodium leucotomos* and nb-UVB as opposed to the single Phototherapy [56, 57]. A randomized study also compared the effectiveness of *P. leucotomos* when combined with NB-UVB for the treatment of generalized vitiligo. Patients with generalized vitiligo (N=57) were randomized to receive combination therapy with twice-weekly NB-UVB and once- daily *P. leucotomos* 480mg (N=29) or NB-UVB phototherapy alone (N=28) for up to six months. The addition of oral *P. leucotomos* to NB-UVB resulted in 40-percent re- pigmentation versus 22 percent for NB-UVB alone. Patients treated with combination therapy showed significantly higher re-pigmentation throughout the study ($p<0.0005$). There are no published studies to date regarding the efficacy of oral *P. leucotomos* without adjunctive light therapy. There was no significant difference in the energies (joules) used in patents receiving oral *P. leucotomos* in any of the studies [58-60].

***Picrorhiza kurroa* Enhances Phototherapy and Melanogenesis for Vitiligo Treatment:** Ayurvedic medicine has the use of herbal products like *Picrorhiza kurroa* for treating the vitiligo. The extract from khellin having hepatoprotective properties, *Picrorhiza kurroa* can be called as "Kutki" or "Kutaki." Of late, some work have shown that this herbal extract also possesses antioxidant and immune-modulating properties. A recent research look into the potential of *Picrorhiza kurroa* when used along the side of phototherapy for vitiligo treatment. The herbal extract was given orally twice a day for a period of three months. Simultaneously, patients underwent treatment with methoxsalen photochemotherapy.

The combined use of these therapies has shown improved results in terms of repigmentation [61]. Another Ayurvedic herbal remedy used in vitiligo treatment is extracted from plants in the Anacardiaceae family, known as anarchic [62]. This topical treatment seems to function as a photosensitizing agent, further data and research are important.

***Cucumis melo* known for its Superoxide Dismutase Properties:** *Cucumis melo*, also called as "muskmelon," it is a herb that belongs to a member of the family Cucurbitaceae or the genus Cucumis. The extract from antioxidant-rich Cucumis melo is well-known for its superoxide dismutase (SOD) activity, which is necessary in halting the oxidative stress-induced degradation of melanocytes, specialy in the early stages of vitiligo. Recently ongoing research has been conducted to evaluate the effectiveness of a topical formulation containing catalase and *Cucumis melo* superoxide dismutase (SOD) for the treating vitiligo [63-64].

The gel was commonly applied to skin lesions in each of these investigations, and then the lesions were subjected to either artificial narrow band UVB or natural UV radiation. While the formulation has been proven to be safe, individuals who got only phototherapy did not show a significantly greater degree of repigmentation. The use of an alternative topical formulation that blends phenylalanine, acetyl cysteine, and *Cucumis melo* extract is more appealing and desired. When used using nb-UVB target phototherapy, this gel has been known to be safe and effective, improving skin lesions' repigmentation [65].

Baicaleinlow the Level of ROS and Treat Vitiligo: Asian traditional medicine makes considerable use of a flavonoid which has been isolated from the roots of *Scutellaria baicalensis* Georgi [66]. It has anti-cytotoxic, anti-tumor, and anti-inflammatory effects [67] likewise, baicalein's antioxidant properties have attracted attention recently, particularly for its ability to lower the levels of reactive oxygen species (ROS) produced by certain chemicals [68] or ultraviolet (UV) rays [69]. Baicalein has potent antioxidant properties because it may oxidatively burn the three hydroxyl (OH) groups at positions 5, 6, and 7 in its chemical structure, reducing ROS [70]. Its strong antioxidant qualities are made even stronger by the persistent semiquinone radicals it generates [71]. The apoptosis that H_2O_2-induced PIG1 cells endure may be accelerated *via* the mitochondrial pathway. In a laboratory experiment that replicated H_2O_2-induced oxidative stress, baicalein offered protection against H_2O_2-induced oxidative stress and mortality in PIG1 cells. In addition, it produced cytochrome c, decreased the Bax/BcI-2 ratio, and maintained mitochondrial membrane potential. The main procedure involves the regulation of the p38MAPK pathway and the activation of mitochondria-dependent caspase. Liu *et al.* (2012) discovered that baicalein exhibited the strongest protective effect at a concentration of 40 μM. Baicalein assisted the passage of Nrf2 from the cytoplasm to the nucleus by raising the expression of Nrf2 and its downstream effector HO-1 in human vitiligo melanocytes (PIG3V) exposed to hydrogen peroxide. It indicates that the Nrf2 signaling pathway [72] is essential for baicalein's protective effects on melanocytes. In addition, baicalein displays antioxidant activities in keratinocytes [73], suggesting that creating a topical cream using baicalein to treat vitiligo might be a practical strategy.

Kaempferol (No Cytotoxic Effect for Vitiligo Therapy): This common type of flavonoid is mostly obtained from *Kaempferia galanga* L.'s rhizome [74]. It also seen in wide varieties of fruits, vegetables, and beverages, involving tea, hazelnuts [75]. Cardiovascular illnesses, cancer, immunologic dysfunction, diabetes, oxidative stress, and other medical conditions can all be treated with kaempferol [76].

On B16F10 cells, kaempferol does not significantly affect cytotoxicity at doses of 16–32 μm for a 24-hour period [77]. An extract from *Baccharoides anthelmintica* (L.) *Moenchknown kaliziri* is showing useful as treating vitiligo [78]. The main active ingredients in the *Baccharoides anthelmintica* (L.) Moench[79] extract is kaempferol. The melanin available in hmvii cells significantly increased following 7 days of kaempferol treatment at 1, 5, 10, and 20 μm [80]. By promoting the development of the tyrosinase protein, it increased tyrosinase activity in mouse buccal hair follicles and HMVII cells.

Utilizing the SDTNBI method along with experimental validation, kaempferol significantly enhanced tyrosinase activity and the gene expression associated with melanin biosynthesis in B16F10 cells, effective in facilitating melanin production.

Vitexin: Multifaceted Flavonoid with Cancer and Skin Disorder Therapeutic Potential: A naturally occurring flavonoid, vitexin (apigenin-8-C-β-D-glucopyranoside) hasfound in a various kind of medicinal plants, including cristalina Vanderpl [81].

Multiple pharmacological behavior, including anti-inflammatory, antiviral, anticancer, and antihypertensive properties [82], were shown by it. Vitexin also known for its anti-oxidant properties [83]. By stimulating the MAPK-Nrf2/ARE pathway, which involved a decrease in IL-1β production, Vitexin was able to enhance cell proliferation and also prevent hydrogen peroxide-induced death in tests utilizing hydrogen peroxide-induced human melanocyte PIG1. In addition, it reduced the levels of IL-17A, Bax, caspase-3, and ROS while enhancing the expression of p53, Bcl-2, Nrf2, HO-1, NQO-1, and SOD [84].

Sesamin: A type of lignan present in oil and seeds of *Sesamum indicum* L., has demonstrated various biological effects beneficial to the human body [85]. Reports indicate that sesamin possesses high antioxidant properties [86]. Furthermore, its also has anti-nociceptive and anti-inflammatory effects have been documented [87]. It has been shown to enhance to cotrienol levels in the skin, thereby reducing the likelihood of sunburn and tumor occurrence [88], mitigating UVB-induced skin erythema, alleviating skin inflammation, and protecting against wrinkle development and light damage. Within the concentration range of 1–10 μM, sesamin stimulates melanin synthesis in a dose-dependent manner. Sesamin's activation of the cAMP/PKA signaling pathway leads to a boost in tyrosinase and MITF expression, which in turn upregulates the CREB gene and promotes the synthesis of melanin. New developments in transdermal drug delivery methods are expected to help the transformation of lignin into an oral remedy for vitiligo [89].

TABLE 1: LIST OF VARIOUS HERBAL DRUGS USED IN THE TREATMENT OF VITILIGO

Herb	Part used	Major constituents	Mechanism of action	Ref.
Psoralea corylifolia	Seeds	Psoralen, bergapten, bakuchiol	Melanogenesis stimulator/ photosensitizer	90
Ammi visnaga	Seeds	Khellin, visnagin	Melanocyte stimulant	91
Ginkgo biloba	Leaves	Bilobalide, kaempferol	Immune- modulatory	92
Camellia sinensis	Leaves	Epigallocatechin-3-gallate	Anti- inflammatory	93
Aloe vera	Leaf pulp	Aloin, emodin	Anti- inflammatory, wound healing	94
Polypodium leucotomos	Whole plant	Caffeic acid, ferulic acid	Photoprotective	95
Piper nigrum	Fruit	Piperine	Enhances melanocyte proliferation	96
Nigella sativa	Seeds / oil	Thymoquinone, nigellone	Immunomodulatory	97
Curcuma longa	Rhizome	Curcumin, demethoxycurcumin	Potent antioxidant	98
Scutellariabaicalensis	Roots	Baicalein, wogonin	Antioxidant (Nrf2/ho-1 activation)	99
Silybum marianum	Seeds	Silymarin (silybin, silychristin)	Free- radical scavenger	100
Zingiber officinale	Rhizome	Gingerols, shogaols	Nutritional adjunct	101
Glycyrrhiza glabra	Roots	Glycyrrhizin, liquiritin	Some topical depigmentation effects	102
Rosmarinus officinalis	Leaves	Rosamarinic acid, carnosol	Antioxidant, anti-inflammatory	103
Pongamia pinnata	Seeds / oil	Pongamol, karanjin	In topical skin remedies	104
Berberis vulgaris	Bark/ roots	Berberine	Immunomodulatory activity	105
Baicalea	Roots / leaves	Baicalein, baicalin	Nrf2 activation, apoptosis reduction	106
Quercetin	Bulbs/ leaves	Quercetin, rutin	Protects melanocytes from oxidative damage	107
Emblica officinalis	Fruits	Ascorbic acid, tannins	Supports melanocyte health	108
Moringa oleifera	Leaves / Seeds	Flavonoids, phenolics	Skin supportive properties	109
Eclipta alba	Whole plant	Wedelolactone, ecliptal	Anti-oxidant	110
Polypodium leucotomos	Leaf extract	Caffeic, ferulic & p- coumaric acids	Photoprotective,adjunct to UV therapies	111
Amaranthus / Beet & spinaches	Leaves/ vegetables	Carotenoids, folates	Nutritional antioxidant support- reduce oxidative stress	112

Pueraria	Roots	Puerarin, isoflavones	Cell- protective effects in experimental models	[113]
Fenugreek	Seeds	Diosgenin, flavonoids	Anti-inflammatory	[114]
Tribulus terrestris	Fruits	Saponins, steroidal saponin	Potentially through activating the PDE/Camp/PKA pathway	[115]
Polygonum multiflorum	Roots	Emodin, rhein	Stimulates melanogenesis	[116]
Cinnamomum verum	Bark	Cinnamic acid, cinnamaldehyde	Tyrosinase inhibition, regulation of Gene Expression	[117]
Syzygium aromaticum	Flower bud	Eugenol, tannins	Influence on signaling pathway	[118]
Elettaria cardamomum	Seeds	Terpenes, flavonoids	Melanocyte- Restorative properties	[119]
Echinacea purpurea	Roots / aerial	Alkamides, cichoric acid	Immunomodulatory effects	[120]
Salvia officinalis	Leaves	Rosmarinic acid, essential oils	Inhibit pro- inflammatory markers	[121]
Thymus vulgaris	Leaves	Thymol, carvacrol	Reducing pro-inflammatory cytokines & promoting IL-10	[122]
Melissa officinalis	Leaves	Rosmariic acid	Photoprotection, melanogenesis promotion	[123]
Valeriana officinalis	Roots	Valerenic acids	Potentially influencing GABAergic & glutamatergic system	[124]
Passiflora incarnata	Aerial parts	Alkaloids, flavonoids	Neuroprotection	[125]
Withania somnifera	Roots	Withanolide D, withaferin A	Melanocyte protection & regeneration	[126]
Smilax spp.	Roots	Saponins, flavonoids	Antioxidant	[127]
Rehmannia glutinosa	Roots	Catalpol, acteoside	Reducing oxidative stress through antioxidant activity	[128]
Salvia miltiorrhiza	Roots	Tanshinones, salvianolic acids	Reducing autoimmune- mediated melanocyte destruction	[129]
Bupleurum Chinese	Roots	Saikosaponins	Interferon- gamma pathway	[130]
Cuscuta chinensis	Whole plant	Flavonoids, alkaloids	Regulation of Apoptosis, mitochondrial protection	[131]
Epimedium (Horny goat weed)	Aerial parts	Icariin	Modulating pathways like Nrf2/HO-1, MAPK to protect melanocytes from oxidative stress	[132]
Aconite	Roots (processed)	Alkaloids	Interacting with voltage- gated sodium channels to cause persistent cell depolarization	[133]
Picrorhiza kurroa	Roots	Picrosides 1 & 2	Repigmentation, inhibition of inflammatory cells	[134]
Bacopa monnieri	Leaves	Bacosides	Enhance cognitive function	[135]
Milk thistle	Seeds	Silymarin complex	Modulation of T- cells	[136]
Eldeberry	Fruits/ flower	Anthocyanins	Upregulation of Nrf2 pahway	[137]
Astragalus membranaceus	Roots	Astragalosides	In TCM adjuncts	[138]
Nettle	Leaves	Flavonoids	NF- Kb inhibition	[139]

Recent Advancement in Treatment of Vitiligo:

Emerging Therapeutics Targeting Janus-Activated Kinase (Jak) Signaling: It consists of various family such as JAK1, JAK2, JAK3, TYK2, which is important in JAK/STAT pathway, show pleiotropic effect on transducing various extracellular signals involved in regulating proliferative signaling, differentiation, migration, *etc* [140].

No inhibitor is present against dermatological issue, although ruxolitinib and tofacitinib are used to treat myelofibrosis and rheumatoid arthritis. This medication is proved useful to treat vitiligo [141].

Wntsignaling and its Agonist: Wnt signaling is necessary for melanocyte migration, proliferation, and differentiating in vitiligo suffering patient [142], and oxidative stress can halt these processes [143]. Along with its downstream enzymes [144], it additionally has a role in MITF activation. Intradermal injection of IWR-1 inhibits the number of melanocytes. A substance which suppresses small interfering RNA and beta-catenin activation against Wnt7-alpha [145]. This proof that inhibition of Wntsignalling can be used to treat vitiligo [146].

Emerging Therapeutics Targeting Micronas: It belongs to the class of non-coding RNA molecules,

which involve in mRNA expression regulation through degradation of mRNA translation [147]. Studies which are performed previously state that miRNAs were involved with genetic polymorphism, immune response, oxidative stress, *etc* [149-153].

CONCLUSION: Ultimately, a promising strategy for addressing this difficult skin condition involves investigating natural bioactive compounds as potential treatments for vitiligo. The management of vitiligo can take various forms through the use of natural bioactives derived from plant extracts and phytoconstituents. These naturally occurring bioactives offer various pharmacological advantages, including antioxidant, anti-inflammatory, and immunomodulatory effects.

The substances highlighted in this review, ranging from Afzelin to berberine (BRB), target different facets of vitiligo's pathophysiology through their distinct mechanism of action. Beyond enhancing the overall condition of individuals with vitiligo, these bioactive compounds have the potential to restore skin pigmentation. They facilitate this by initiating melanogenesis, scavenging free radicals, modulating inflammation, and promoting re-pigmentation.

Further research is essential to gain a comprehensive understanding of the mechanisms of these natural compounds and to assess their safety for use in vitiligo. Clinical trials with larger sample sizes and longer durations are needed to confirm and ensure the repeatability of findings from preclinical studies. The exploration of natural bioactive compounds as potential therapies for this challenging skin disorder represents an intriguing and promising area of research that could enhance the quality of life for those affected by vitiligo.

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