**A Review on Vitiligo: its Causes, Mechanism and Treatment**

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**Abstract**

Vitiligo is a complex skin disorder characterized by white macules. Our goal is to present an overall view of the current remedies widely adopted for the management of vitiligo. Some people experience itching before the appearance of a new patch. Around 0.5% to 2% of the world's population has vitiligo. It affects people of any age, more than half of whom develop it before the age of 20 years; white macules in the skin due to the destruction of melanocytes in the epidermis. Various hypotheses are proposed to explain the pathomechanisms occupied in this disease and studies have exposed the participation of autoimmune processes in the pathogenesis of vitiligo. Cellular and humoral immunities are implicated in the development of vitiligo. There are many treatments to cure vitiligo such as use of corticosteroid creams, PUVA (psoralen and ultraviolet A light), narrow band UVB (ultraviolet B), various surgical techniques and vitamin D analogues. These treatments are subjected for undesired side effects but some herbal and natural treatments act against the immune system with no side effects.

**Keywords:** Vitiligo; Mechanism; Melanocyte; Herbal Drugs; Vitiligo Diets

**Introduction**

Vitiligo also known as leucoderma is the most common chronic pigmentary disorder affecting 1-4% of the world population [1]. Approximately 50% of the cases have the onset of the disease prior to the age of 20 years and 25% prior to the age of 14 years [2]. Vitiligo is also known as leucoderma [3]. When skin whiteness happens due to cut, accident or a burn then it is termed leucoderma but normally happening patches can be termed vitiligo [4]. It includes the selective destruction of melanocytes of the basal layer of the epidermis and/or occasionally the hair follicle resulting in white patches on the skin, the mucous membranes and/or white hair [5]. These white patches tend to become progressive with time [6]. Although vitiligo is usually not harmful medically and causes no physical pain, its emotional and psychological effects can be devastating, [7] particularly if the condition develops on the visible areas of the body (such as face, hands, arms and feet) or on the genitals [8,9].

**Causes of Vitiligo**

There are many theories and opinions about the origin of vitiligo, which includes

- Autoimmunity i.e. due to frequent association with other autoimmune disease such as thyroid, and type I diabetes, presence of anti-melanocyte antibodies, and a response to immunosuppressant therapy.
- Cytotoxicity i.e. the possibility that metabolites formed during melanin synthesis may destroy melanocytes.
- Neural i.e. chemical mediators released at nerve endings might destroy melanocytes or inhibit melanin production, excess free radicals might be toxic for melanocytes.
- Genetic i.e. (NALP1) that cause an individual to be susceptible for developing vitiligo.
- Triggering i.e. some event triggers the destruction of the pigment cells (sunburn, trauma, pregnancy etc.).
- Regular intake of odd food combination, regular stress, and due to some lifestyle problems [2].

**Process of Melanogenesis**

Melanocytes are cells which produce the pigment melanin, which is present in the matrix of the hair follicle of the basal layer of epidermis. Tyrosinase is required for melanin production. Tyrosinase is the rate limiting enzyme of melanogenesis, catalyzes the hydroxylation of L-tyrosine to DOPA and the oxidation of DOPA to DOPAquinone. If cysteine or glutathione is available, it reacts with DOPAquinone to form cysteinyl DOPA and the benzo-thiazine derivatives of pheomelanin. As cysteine is diminished, DOPAquinone cyclizes into DOPAchrome. TYRP-2 catalyzes the tautomerization of DOPAchrome to 5,6-dihydroxyindole-2-carboxylic acid (DHICA), which is later oxidized to DHICA-melanin subunits. The oxidation of DHICA to eumelanin is thought to be catalyzed by TYRP-1. Without TYRP-2 the carboxylic acid moiety of DOPAchrome is suddenly lost to form 5,6-dihydroxyindole (DHI). DHICA...

Figure 1: Normal skin Vs Vitiligo skin

Figure 2: Process of melanogenesis within epidermal melanosomes
in combination with DHI comprise subunits of eumelanin [1,10].

**Symptoms and Signs of Vitiligo**

Vitiligo can affect melanocytes in the hair roots, resulting in white eyelashes and white hair within the pale skin patches. These are often symmetrical and usually increase in size with time. In symmetrical vitiligo, the commonest sites to be affected are the fingers and wrists, the axillae and groins and the body orifices such as the mouth, eyes and genitalia. In vitiligo skin there is no surface change and usually no redness. As the pigment cells are destroyed, sometimes a ‘trichrome’ appearance of a white centre with an intermediate, pale area around it is found. When vitiligo affects only the genital areas, it can be difficult to exclude lichen sclerosis, which sometimes can coexist with vitiligo. Patients with vitiligo often develop autoimmune thyroid disease or other autoimmune diseases and a history of autoimmune disease in a family member is obtained in 32% of patients [4].

**Classification of Vitiligo**

Vitiligo is classified according to the distribution, pattern and extent of de pigmentation.

There are two large subtypes of vitiligo, segmental vitiligo (S) and non-segmental vitiligo (NS). According to another classification three types are identified i.e., localized, generalized and universal vitiligo [1].

**Localized Vitiligo**

Localized vitiligo has three types

- **Focal vitiligo:** Limited to one or few areas
  - Does not progress
  - Not clearly in a segmental distribution
- **Segmental vitiligo:** Unilateral and asymmetric in distribution
  - Only one side of the body is affected
  - Common in children
- **Mucosal vitiligo:** Mucous membranes are affected

**Generalized Vitiligo**

Generalized vitiligo has three types

- **Acrofacial vitiligo:** Occurs on the parts away from the centre of the body
- **Vulgaris vitiligo:** Scattered patches
  - Widely distributed
- **Universal vitiligo:** Nearly complete de pigmentation

**Home Remedies for Vitiligo**

Vitiligo may also be treated using certain herbal remedies, some of which are mentioned below.

- Turmeric mixed with mustard oil has proved useful in vitiligo. Approximate 500 gm of turmeric should be pounded and soaked in 8 liters of water at night. It should be heated in the morning till only one liter of water is left, and then be strained and mixed with 500 ml of mustard oil. This mixture should be heated up to
only the oil is left. It should then be strained and preserved in a bottle. The mixture should be applied on the white patches every morning and evening for 2-3 months.

- Take about 25 grams of radish seeds and ground them into a powdery substance. Add 2 tsp vinegar and make a fine paste. Apply this paste on the affected area and wash off when dry.
- Soak psoralea seeds in ginger juice for 3 days. Thereafter, dry them and grind to make a fine powder. Have 1 gram of this powder, with a glass of milk, for about a month. Can also apply this powder on white patches.
- Another method would be to soak psoralea and tamarind seeds in water for 3-4 days. Dry them well and grind to form a paste. Apply this paste on the affected area on a daily basis.
- Mix red clay and ginger in equal parts. Apply this on the affected area and wash off when dry.
- Fill a copper vessel with drinking water and leave it overnight. Have this water, on an empty stomach, every morning.
- Take a handful of dry pomegranate leaves and grind them into a fine powder. Have about 8 grams of this powder every morning and evening, with a glass of water.
- Neem is very effective in treating leucoderma. Try to drink a glass of neem juice every day.
- Grind black gram and add water, to form a paste. Apply this paste on the affected area, washing off after it dries, for about 4 to 5 months. It is an effective remedy for treating leucoderma.
- Holy basil leaves provide evidence to be beneficial in treating white patches. Use raw basil leaves or make a decoction out of Holy basil's leaves and stem, along with water [2].

**Reported Herbs that are Claimed to be Used in the Condition of Vitiligo**

Ayurveda, a Sanskrit term meaning 'science of life,' is said to be the most ancient system of medicine in widespread practice today. In Ayurvedic classical literature, such as the CharakaSamhita, vitiligo also known as 'white leprosy' is called svitraorkilasa. Following plants or herbs are used in vitiligo [2,11].

**Mechanism of Vitiligo**

Autoimmune Mechanism Causing Vitiligo

The correct etiology of vitiligo is unknown, but four main theories exist to clarify it: The Autoimmune hypothesis, The Neural hypothesis, The Self destruct hypothesis, and The Growth Factor Defect hypothesis. None of these mechanisms are conclusively proven. It is believed that Vitiligo is a polygenic trait and that a Convergence theory, combining elements of different theories across a spectrum of expression is the most accurate etiology. In a number of recent studies, strong evidence in favor of the autoimmune hypothesis has been obtained. The autoimmune model shows that melanocytes death occurs through irregular immune system destruction of pigment cells. Both Cellular and Humoral immunity helps in the degradation of melanocytes. The association of vitiligo with other known autoimmune disorders such as Addison's Disease, Hashimoto's Thyroiditis, Pernicious anemia and Alopecia areata also endorses the autoimmune theory of disease [13,14].

Antigens yielded by Melanocytes (including those released from defected Melanocytes) can be detected by Antigen specific immune effector cells including Cytotoxic T-cells, T-Helper cells and B-cells. Recognition of Melanocyte Antigens might also arise through cross reaction from immune responses to other cell types or contagious agents. The presence of Lymphocytes as well as Auto antibodies significantly decreases the number of living Melanocytes, explaining the role of cellular immunity by Antibody dependent cellular cytotoxic reaction. A continuous increase of LMIF (Leukocyte Migration Inhibition Factor) by activated T-cell sand circulating Igs (Immunoglobulin) and immune complexes became as a result of T-Cell mediated B-cell activation during Vitiligo. The crucial antigen recognized by these auto antibodies is Tyrosinase. Other melanocyte differentiation antigens that are

<table>
<thead>
<tr>
<th>Sr. No.</th>
<th>Herbs biological name</th>
<th>Family</th>
<th>Common name</th>
<th>Part used</th>
<th>Treatment support</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Plumbago indica</td>
<td>Plumbaginaceae</td>
<td>Chitraka</td>
<td>Root and bark</td>
<td>Vitiligo skin</td>
</tr>
<tr>
<td>2</td>
<td>Psoralea corylifolia</td>
<td>Leguminosae</td>
<td>Bavchi, Bakuchi</td>
<td>Seeds, root, leaves</td>
<td>vitiligo</td>
</tr>
<tr>
<td>3</td>
<td>Pipernigrum</td>
<td>Piperaceae</td>
<td>Pepper, Kali mirch, Maricha</td>
<td>Seeds</td>
<td>Repigmentation, Penetration enhancer</td>
</tr>
<tr>
<td>4</td>
<td>Nigellastiva</td>
<td>Ranunculaceae</td>
<td>Kalonji</td>
<td>seeds and oil</td>
<td>Vitiligo skin</td>
</tr>
<tr>
<td>5</td>
<td>Ginkgo biloba</td>
<td>Ginkgoaceae</td>
<td>Ginkgo</td>
<td>Dried Leaves</td>
<td>Repigmentation</td>
</tr>
<tr>
<td>6</td>
<td>Picrorhizakurroa</td>
<td>Scrophulariaceae</td>
<td>Indian Gentian, Kutki</td>
<td>Dried Rizomes</td>
<td>Repigmentation</td>
</tr>
<tr>
<td>7</td>
<td>Zingiber officinalis</td>
<td>Zingiberaceae</td>
<td>Ginger</td>
<td>Dried Rizomes</td>
<td>Bowel Strengthening</td>
</tr>
<tr>
<td>8</td>
<td>Ammimajus</td>
<td>Apiaceae</td>
<td>Ammi</td>
<td>Fruits</td>
<td>Pigmentation enhancer</td>
</tr>
<tr>
<td>9</td>
<td>Tribulusterrestris</td>
<td>Zygophyllaceae</td>
<td>Gokhru</td>
<td>Fruit powder</td>
<td>Repigmentation</td>
</tr>
<tr>
<td>10</td>
<td>Azadirachtaindica</td>
<td>Meliaceae</td>
<td>Neemru</td>
<td>Leaves</td>
<td>Repigmentation</td>
</tr>
</tbody>
</table>

Table 1: Reported herbs that are claimed to be used in the condition of vitiligo
recognized are Gp100/Pmel17, and TRP1 (Tyrosinase Related Proteins 1) and TRP2 (Tyrosinase Related Proteins 2). These antigens localize primarily to the Melanosomes, where it is a well known fact that Antibody mediated killing needs membrane expression of the target antigens. If the immune system raises Antibodies or Cytotoxic T-Cells to harm melanocytes, the way of action the cells take against the melanocyte scan be a direct induction of apoptosis against melanocytes or Ig induced Complement [14,15].

Cell mediated immunity

In Cell mediated immunity, after processing of Antigens by APC (Antigen Presenting Cells), Antigenic peptides are presented to the TCR (T-Cell Receptors) of CTLs (Cytotoxic T-Lymphocytes) in the context of MHCI (Major Histo compatibility Complex Class I) molecules. Majority of these T-cells also expressed the cytotoxic effector molecules Perforin and Granzyme. CD8+ CTL plays an important role in immune mediated destruction of autologous melanocytes.

However, cellular cytotoxicity against melanocyte differentiation antigens may not be solely dependent on MHC ClassI restricted T-cells. The in vivo activation of such CD4+ CTL may occur when professional APC, after endocytosis of dying melanocytes, migrate to skin draining lymph nodes to present peptides derived from Melanosomal Antigens. However, actual killing of Melanocytes by MHC Class II restricted CTL also requires that target cells present endogenous melanosomal proteins in the context of MHC Class II molecules. Cognate help (via Cytokine production) by Antigen specific T-Helper cells, in the context of Antigenic peptides presented on MHC Class II molecules, is required for a long lasting Cytotoxic T-cell response against melanocytes that can lead to their destruction. The major cytokines involved in cognate help include IL2R (Interleukin 2 Receptor), IL6 (Interleukin 6) and IL8 (Interleukin 8). IL6 can induce the expression of ICAM1 (Intercellular Adhesion Molecule1) on melanocytes, which helps Leukocyte Melanocyte interaction, leading to immunological damage of the pigment cells. IL8 may attract neutrophils to vitiligo lesions, which could lead to the amplification of inflammatory reactions and the destruction of Melanocytes [16,17].
Humoral Immunity

The Humoral immune reaction has been worsened through the detection of circulating antibodies. The antigen specific membrane IgM of B-Cells captures antigens. The production and secretion of antigen specific antibodies by B-Cells are also dependent on cognate help (via Cytokine production) by antigen specific T-helper cells. Anti-melanocyte antibodies can destroy pigment cells by either antibody dependent Complement mediated damage or ADCC (Antibody Dependent Cell mediated Cytotoxicity) [18].

Autoimmune hypothesis of Melanocyte declination is further exhalted by the current clinical practice of vitiligo management, all of the non-surgical vitiligo treatments with specified efficacy are based on immuno suppression. Thus, further investigations in Vitiligo etiology would based on finding the exact conditions that trigger and sustain this melanocyte specific autoimmune response. Establishing the triggers that awaken this latent mechanism in vitiligo will not only help patients with this autoimmune disease, but may also be of considerable importance for melanoma therapy, where an effective immune response against melanocyte differentiation antigens is lacking [19].

Treatments of Vitiligo

Conventional treatments available for vitiligo, indicated topical mono therapy for mild to moderate vitiligo. Current treatment options for vitiligo include medical, surgical and additional treatments. Medical treatment targets the immune system and helps to arrest the spread of de pigmentation. In cases of stable vitiligo, re pigmentation can be achieved by dermato-surgical techniques and additional treatments include use of cosmetics. Both surgical and medical treatments have their own limitations. Additional treatments only cover the patch and can be used along with surgical and medical treatments.

**Topical Treatments**

Topical treatments for vitiligo include

**Corticosteroid Creams**

These can be used for vitiligo on trunk and limbs for up to 3 months. Potent steroids should be avoided on thin skinned areas of face especially eyelids, neck, armpits and groin.

**Calcineurin Inhibitors**

Pimecrolimus cream and Tacrolimus ointment can be used for vitiligo affecting eyelids, face, neck, armpits and groin.

**Psoralen-Light Therapy**

(UVA or UVB) can be combined with Psoralen. This drug makes the skin more susceptible to UV light. As the skin heals and normal coloration sometimes returns. Treatment may need to be repeated two or three times a week for 6 - 12 months [20].

Psoralen increases the risk of sunburn and skin damage, as a result increasing the risk of skin cancer further down the line.

**Calcipotriene (Dovonex)**

Calcipotriene is a form of vitamin D used as a topical ointment. It may be used in combination with corticosteroids or light treatment. Side effects can include rashes, dry skin and itching [21].

![Figure 6: Treatment of Vitiligo](image-url)
Phototherapy

Phototherapy refers to treatment with ultraviolet (UV) radiation. Options consist of

- Whole body or localised broadband or narrowband (311 nm) UVB
- Excimer radiation UVB (308 nm) or targeted UVB for small areas of vitiligo
- Oral, topical, or bathwater photo chemotherapy (PUVA)[21]

Phototherapy probably works in vitiligo by 2 mechanisms.

- Immune suppression- preventing destruction of the
- Stimulation of cytokines (growth factors)

Treatment is usually given twice weekly for a trial period of 3 to 4 months. If repigmentation is observed, treatment is continued until repigmentation is complete or for a maximum of 1-2 years.

- Phototherapy is unsuitable for extremely fair skinned people.
- If repigmentation is observed, treatment is constant until repigmentation is complete or for a maximum of 1-2 years.
- Treatment times are generally concise. The aim is to cause the treated skin to appear very slightly pink the following day.
- It is important to avoid burning (red, blistered, peeling, itchy or painful skin), as this could cause the vitiligo to get worse.

Systemic Therapy

Systemic treatments for vitiligo include

- Oral minocycline, a tetracycline antibiotic with anti-inflammatory properties
- Mini-pulses of oral steroids for 3 to 6 months
- Subcutaneous afamelanotide (α-melanocytestimulating hormone) [22]

It is anticipated that monoclonal antibody biologic agents will be developed to treat vitiligo.

Surgical Treatment of Stable Vitiligo

Surgical treatment for stable and segmental vitiligo requires removal of the apex layer of vitiligo skin by shaving, dermabrasion, sandpapering or laser and replacement with pigmented skin removed from a different site.

Techniques include

- Non cultured melanocyte-keratinocyte cell suspension transplantation.
- Blister grafts, formed by suction or cryotherapy
- Punch grafting
- Split skin grafting
- Cultured auto-grafts of grown in tissue culture
- Tattooing: The pigment is implanted into the skin. It works best around the lips, especially in people with darker skin [23].

Depigmentation Therapy

When the affected area is well-known covering 50% of the body or more depigmentation can be an option. Depigmentation reduces the skin color in unaffected areas in order to match the whiter areas. Depigmentation is achieved by the use of strong topical lotions, creams or ointments, like monobenzone, mequinol or hydroquinone. This treatment is permanent and can make the skin more fragile.

Long exposure to the sun must be avoided. Depigmentation can take about 12 to 14 months depending on factors including the depth of the original skin tone. Laser treatment (eg 755 nm Q-switched alexandrite or 694 nm Q-switched ruby) have also been used successfully to depigment small areas of vitiligo.

<table>
<thead>
<tr>
<th>S.No.</th>
<th>Class</th>
<th>Drugs</th>
<th>Chemical structure</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Natural</td>
<td>Piperine</td>
<td><img src="image1" alt="Chemical structure of Piperine" /></td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>(piper nigrum)</em></td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Psoralen</td>
<td><img src="image2" alt="Chemical structure of Psoralen" /></td>
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<tr>
<td></td>
<td></td>
<td><em>(Psoraleacorylifolia)</em></td>
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<tr>
<td></td>
<td>Hormones</td>
<td>Corticosteroids-</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>2.</td>
<td></td>
<td>1. Triamcinolone</td>
<td></td>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. Hydrocortisone</td>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>3. Clobetasol propionate</td>
<td></td>
</tr>
<tr>
<td>3.</td>
<td>Vitamin</td>
<td>Vitamin D analogues</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>- Calcipotriol</td>
<td></td>
</tr>
<tr>
<td>4.</td>
<td>Synthetics.</td>
<td>Monobenzone,</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mequinol,</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>or hydroquinone</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Calcineurin inhibitors –</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pimecrolimus</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Tacrolimus</td>
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</tr>
</tbody>
</table>

**Table 2: Drugs uses in treatment of vitiligo**

Vitiligo Diet

Vitiligo Diet - Food Items to Avoid
Following is the list of food items which avoiding in our diet. These are generally suggested by many people for controlling Vitiligo.

Vitiligo Diet - Food Items to Intake
Following is the list of food items which adding to our diet. This list also again complied on the basis of recommendations given by people looking to cure Vitiligo.

<table>
<thead>
<tr>
<th>S.N.</th>
<th>Fruits</th>
<th>Vegetables</th>
<th>Milk Products</th>
<th>Non-Vegetarian Food Items</th>
<th>Other Food Items</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Orange</td>
<td>Brinjal</td>
<td>Milk</td>
<td>Fish</td>
<td>Junk Food</td>
</tr>
<tr>
<td>2.</td>
<td>Gooseberry</td>
<td>Red Sorrel</td>
<td>Curd</td>
<td>Red Meat</td>
<td>Chocolates</td>
</tr>
<tr>
<td>3.</td>
<td>Custard Apple</td>
<td>Parsley</td>
<td>Buttermilk</td>
<td>Beef</td>
<td>Coffee</td>
</tr>
<tr>
<td>4.</td>
<td>Guava</td>
<td>Papaya</td>
<td></td>
<td></td>
<td>Soda bi carb</td>
</tr>
<tr>
<td>5.</td>
<td>Prunes</td>
<td>Lemon</td>
<td></td>
<td></td>
<td>Carbonated Drinks</td>
</tr>
<tr>
<td>6.</td>
<td>Cashew nuts</td>
<td>Tomato</td>
<td></td>
<td></td>
<td>Oily and spicy food items like Pickles</td>
</tr>
<tr>
<td>7.</td>
<td>Water melon</td>
<td>Tamarind</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8.</td>
<td>Melon</td>
<td>Garlic</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Table 3: Vitiligo Diet – Food Items to Avoid

<table>
<thead>
<tr>
<th>S.N.</th>
<th>Vegetables</th>
<th>Fruits</th>
<th>Other Food Items</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Radish</td>
<td>Mangoes</td>
<td>Wheat</td>
</tr>
<tr>
<td>2.</td>
<td>Beet Root</td>
<td>Grapes</td>
<td>Pure Ghee</td>
</tr>
<tr>
<td>3.</td>
<td>Carrot</td>
<td>Walnut</td>
<td>Red Pepper</td>
</tr>
<tr>
<td>4.</td>
<td>French Beans</td>
<td>Apricot</td>
<td>Bengal Gram</td>
</tr>
<tr>
<td>5.</td>
<td>Ridge and Bitter Gourd</td>
<td>Dates</td>
<td>Jaggery</td>
</tr>
<tr>
<td>6.</td>
<td>Fenugreek</td>
<td>Papaya</td>
<td>Pistachio Nut</td>
</tr>
<tr>
<td>7.</td>
<td>Spinach</td>
<td></td>
<td>Almond</td>
</tr>
<tr>
<td>8.</td>
<td>Drumsticks</td>
<td></td>
<td>Potato</td>
</tr>
<tr>
<td>9.</td>
<td>Onion</td>
<td></td>
<td>Chillies</td>
</tr>
</tbody>
</table>

Table 4: Vitiligo Diet – Food Items to Intake

Conclusion
In the conclusion it can be said that, Vitiligo is a disorder of skin, in which white macules, develops on the skin due to the loss of functioning melanocytes. We have a number of treatment options for the disease but there is no satisfactory treatment for this disease. There is a need to find the exact pathogenesis for the disease for the development of effective treatment. The results in the disease in term of re pigmentation are occurred with the use of herbal treatment along with irradiation of the de pigmented skin. Surgical treatment can be considered if the other treatments failed.

References


