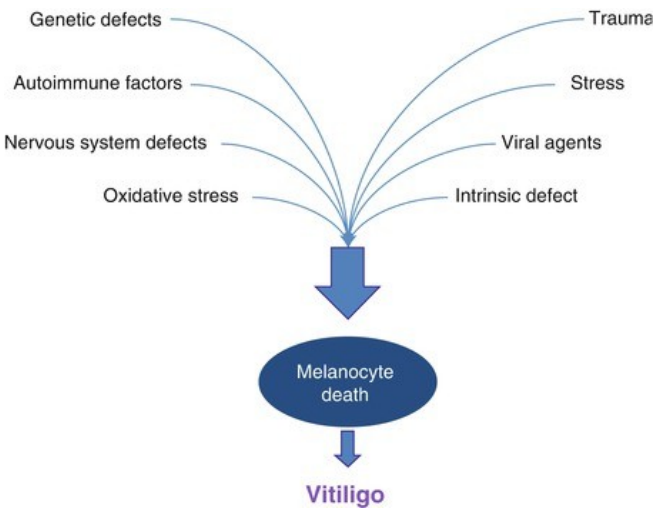
Vitiligo is a skin condition that causes the skin to lose its color or pigment. This causes the skin to appear lighter than the natural skin tone or turn white. Areas of the skin that lose their pigment are called macules if they're less than 1 centimeter wide, or patches if they're larger than 1 centimeter. The amount of affected skin varies for each person diagnosed. Some experience a few depigmented areas, while others have widespread loss of skin color.

Vitiligo is a complex condition that occurs when the body's immune system destroys melanocytes. Melanocytes are skin cells that produce melanin, the chemical that gives skin its color, or pigmentation. It usually starts with a few small white macules or patches that gradually spread over the body. It typically begins on the hands, forearms, feet and face, but can develop on any part of the body. Sometimes, larger patches continue to widen and spread but can stay in one place for years.

Oxidative Stress

The pathogenesis of the condition results from the interaction of genetic components, metabolic factors linked to cellular oxidative stress, melanocyte adhesion to the epithelium, and immunity, which culminate in aggression against melanocytes. In individuals with vitiligo, melanocytes are more sensitive to oxidative damage, leading to increased expression of proinflammatory proteins. It is hypothesized that oxidative stress inflicts cell damage by inducing apoptosis in melanocytes.



Innate and Adaptive Immunity

Dendritic cells, macrophages, and NK cells are found in the lesional skin of patients with vitiligo characterizing an activation of the innate immune response. The oxidative stress that occurs in the melanocyte, as mentioned above, can cause damage to the melanocytes and is possibly the autoimmunity trigger in vitiligo.

The activation of innate immunity triggered by damage to melanocytes ~~affected by oxidative stress~~ promotes cytokine secretion and antigen presentation, resulting in the adaptive immune system activation, in which autoreactive T-cells amplify damage to melanocytes in vitiligo-affected skin. Cytotoxic CD8+ T-cells are necessary and sufficient for the destruction of melanocytes, acting as an effector arm of autoimmunity. The lesions are caused by effector CD8+ T-lymphocytes in the initial or active phase of the disease and by recirculating and resident memory CD8 + T (TEM) lymphocytes in the stable phase.

CD4+ T-regulatory cells (Tregs) act to maintain tolerance to their own tissues by suppressing the activity of T-effector cells. In vitiligo, there is a Treg dysfunction, although it is not known exactly whether due to the inability to migrate to the skin, decreased numbers, or activity suppression.

Wellness Recommendation

In Traditional Chinese Medicine, autoimmunity and T cell dysfunction is tied to Spleen Damp. The recommendation for vitiligo includes Vitiligo A and B formulas. Vitiligo A is an internal formula that helps clear Spleen Damp in the skin to relieve the autoimmune condition. It also helps restore the damaged

pigment production process. Herbal ingredients in Vitiligo A have been shown to modulate the essential deficiency by promoting blood circulation and darken the skin color by balancing melanin cell proliferation and pigmentogenesis. Ingredients also have been shown to have anti-inflammation and anti-oxidant effects. Vitiligo B is an externally applied formula that nurtures the skin to help repair the damage to the melanocytes and promote melanocytes regeneration. Herbal ingredients in Vitiligo B have strong Kidney and Spleen Yang effects and have been used to treat vitiligo externally in various traditional medical systems.

Patients can respond to Vitiligo A and B after 1-2 months. At this time, the patient's loss of pigment in random areas should merge into one larger area and new skin can form from the edge toward the center area. 3-6 months is recommended for significant improvement.

References:

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