

# Dopamine Dynamics in Dermatology and Behavioural Science

**Kotala Sreedevi**

*Dermatologist & Cosmetologist, Sreeguru Heart & Skin Clinic, Pallikaranai, Chennai, Tamilnadu, India*

**\*Correspondence to:**

Sreedevi K  
Dermatologist & Cosmetologist  
Sreeguru Heart & Skin Clinic  
Pallikaranai, Chennai  
Tamilnadu, India

**Received:** September 02, 2020

**Accepted:** November 12, 2020

**Published:** November 15, 2020

**Citation:** Sreedevi K. 2020. Dopamine Dynamics in Dermatology and Behavioural Science. *J Addict Sci* 6(2): 34-36.

**Copyright:** © 2020 Sreedevi. This is an Open Access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC-BY) (<http://creativecommons.org/licenses/by/4.0/>) which permits commercial use, including reproduction, adaptation, and distribution of the article provided the original author and source are credited.

Published by United Scientific Group

## Abstract

In this case studies, we are informing the importance of dopamine in various skin diseases. Stress and depression aggravates skin diseases. It leads to change in dopamine levels in skin. Positive attitude, increasing happiness or making the patient comfortable give good results in skin diseases.

## Keywords

Skin structure, Dopamine, Psoriasis, Atopic dermatitis, Vitiligo

## Introduction

In our dermatology practice, we come across different patients with dermatological and psychological problems.

Before understanding about dopamine receptors, we have to understand about skin anatomy and physiology. There are two main kinds of human skin. Glabrous skin (non-hairy skin) seen in palms and soles and hair bearing skin.

The layers of skin are epidermis, dermis and subcutaneous tissue. The epidermis is further divided into 5 layers [1]. *Stratum corneum*, *Stratum lucidum*, *Stratum granulare*, *Stratum spinosum*, *Stratum basale*. The superficial epidermis is stratified squamous epithelium. The cells of epidermis are keratinocytes, melanocytes, langerhans'cells and merkel cells. Main components of dermis are collagen, elastin and cellular components are fibroblasts, mast cells and histiocytes.

Skin has very rich nerve supply [2]. The sensory nerves originate from nerve trunks in the dermis enter the epidermis, then divide distally to eventually end in small enlargements, near the surface of the skin and in deeper areas.

Free nerve endings are the most common nerve endings in skin, and they extend into the middle of the epidermis. Free nerve endings are sensitive to painful stimuli, to hot and cold, and to light touch. Meissner's corpuscles respond to touch and low-frequency vibration.

The dermis contains nerve endings, sweat glands and, sebaceous glands, hair follicles, and blood vessels. The nerve endings sense pain, touch, pressure, and temperature. Some areas of the skin contain more nerve endings than others.

The exposed mucous membranes of the lips, the anal mucous membrane, and the external genital organs form the most densely innervated parts of the body [3]. Though there is no specific categorization, both "free" nerve endings and unencapsulated nerve endings of myelinated axons are found within the dermis of those areas [4].

The epidermis does not contain blood vessels; instead, cells in the deepest layers are nourished by diffusion from blood capillaries that are present in the upper layers of the dermis [5]. Diffusion provides nourishment and waste removal from the cells of the dermis as well as for the cells of the epidermis.

The outer layer of skin, the epidermis, provides waterproofing and serves as a barrier to infection [6]. The middle layer of skin, the dermis, contains blood vessels, nerves, and glands that are important for our skin's function. The inner layer of the skin, the subcutis, contains fat that protects us from trauma.

The hypodermis is beneath the dermis which is beneath the epidermis [7]. It is used mainly for fat storage. The hypodermis consists primarily of loose connective tissue. It contains larger blood vessels and nerves than those found in the dermis.

Cell to cell communication is critical for the survival of an organism. Cells can communicate through a process called signal transduction pathway. When sending a signal, different molecules, such as hormones, can bind to a receptor on or inside the cell membrane, leading to chemical reactions in the cell ultimately reaching the target. Dopamine is secreted in keratinocytes. D1 receptors are present in dermis of the human plantar skin, lower epidermal layers and subcutaneous tissue [8]. Human keratinocytes also have the capability to utilize L-tyrosine to synthesize L-DOPA, subsequently resulting in the production of dopamine, norepinephrine, and epinephrine.

In psoriasis there is increased keratinocytes because of thick scales. Due to trauma or stress also, these scales will increase. Dopamine also released much from D1 receptors of keratinocytes [9]. So, dopamine levels are high in psoriatic patients. If we reduce stress, they will recover soon. Psoriasis is a chronic inflammatory disease mediated by the immune system with increased proliferation of keratinocytes. The exact cause is unknown but as a multifactor, such as infection, trauma and psychological stress have been thought to play a role.

Dopamine excess in skin causes T. cell activation and cell mediated immunity altered leading to infection or disease [10]. Even Though the disease is chronic in nature, if we show proper attention and empathy towards patient it leads to normal secretion of dopamine from brain and skin.

## Case Report 1

A male patient aged 50 years came with psoriasis. A part from silvery white scales, itching, bleeding spots after removing thick scales, the patient appeared very gloomy and had low self-esteem. It took one year to completely clear his 90% involved skin. Apart from medication, improving his self-esteem, cheering the patient helped a lot in recovery. The medicines used in this patient are

1. Aloe vera, squalene, vitamin E combination moisturiser, whole body application after bath morning and night.

2. Clobetasol propionate 0.05% and salicylic acid 3% combination cream application on the affected skin morning

and night, 10 mins after applying moisturiser.

3. Trimethoxy psoralen tablets morning at 8 am orally, after two hours (10 am) exposure to sun rays (ultraviolet A rays) for few seconds and gradually increasing the sun exposure time to a maximum of 15 mins done. This is called PUVA (Psoralen ultraviolet A) therapy. Once in two days PUVA therapy given.

4. Vitamin D and calcium tablets. oral tablet once daily at night.

5. Coal tar shampoo. Overnight application to scalp, next day morning showering was advised. Once in three days he has to do like this.

## Case Report 2

A male patient aged 5 years came with atopic dermatitis, a condition where skin show dryness, itching, papular eruptions associated with nasal allergy. The patient is anxious, feeling bad to see his skin and told that his classmates and teachers are asking about this. He was treated with the following medication.

1. Cetyl alcohol moisturizer, full body application after bath morning and evening.

2. Mometasone cream application at morning on the affected skin.

3. Tacrolimus cream. 0.03 percent cream application at night on the affected skin.

4. Syrup cetirizine 2.5 ml oral at night advised.

Along with that boosting his self-esteem, making the child more comfortable in the class helped a lot.

## Case Report 3

A female patient aged 18 years suffered from acrofacial vitiligo. The chalky white patches caused more embarrassment to the patient and her parents. They are worried about her future. Her inferiority complex was nullified by counselling. Then medication was started. As there are different modalities of treatment which ever suitable to her was given. She completed her treatment within 14 months and repigmentation through-out the patches was seen. She is more confident.

Skin type's four to six means light brown, dark brown, black respectively. These types of skin are mostly seen in Asians and Africans. The pigment loss is more visible [table 1](#).

Dopamine synthesis begins with the amino acid phenylalanine, and proceeds sequentially through tyrosine, DOPA, and then dopamine. Tyrosine hydroxylase is the rate-limiting enzyme in this pathway. Another important enzyme is DOPA (Dihydroxyphenylalanine) decarboxylase, which decarboxylates DOPA to form dopamine.

DOPA plus tyrosine show a prominent melanogenic activity. Vitiligo, a de-pigmentary disorder of the skin and hair

characterized by selective destruction of melanocytes, has been reported to show increased levels of DA (Dopamine) with the onset and progression of the disease [11-23].

**Table 1:** Classification of sun-reactive skin types.

Skin type	Sun sensitivity	Pigmentary response
1	Very sensitive, always burn easily	Little or no tan
2	Very sensitive, always burn	Minimal tan
3	Sensitive, burn moderately	Tan gradually (light brown)
4	Moderately sensitive, burn minimally	Tan easily (brown)
5	Minimally sensitive, rarely burn	Tan darkly (dark brown)
6	Insensitive	Deeply pigmented (Black)

## Discussion

Dopamine reaches skin via nerve endings. Dopamine is secreted by keratinocytes. These keratinocytes are present in epidermis of skin. Skin has motor, sensory and autonomic nerves. Nerve endings. The skin has very rich blood supply in dermis and partly in sub cutaneous tissue. Epidermis receives blood from blood in dermis by diffusion.

The dopamine reaches skin through nerve endings excess dopamine is observed in blood of vitiligo patients. It is toxic to melanocytes. Lucchi et al, in pigment cell research mentioned in their study about higher levels of catecholamines in early stage of vitiligo.

Dopamine receptors are identified in B. cells, nature killer cells and on T. cells and melanocytes. Dopamine impairs keratinocyte migration through beta 2 adrenergic receptors. Dopamine enhances angiogenesis and wound healing.

## Conclusion

There is a strong relation between dopamine and skin. Skin diseases are causing anxiety, mood changes and depression. In future excess dopamine in vitiligo patients can be extracted and utilized in reward deficiency syndrome. The medicines used in reward deficiency syndrome are also helpful for increasing the well-being of skin patients.

## References

- Burns T, Breathnach S, Cox N, Griffiths C. 2004. Rook's Text book of Dermatology. <https://doi.org/10.1002/9780470750520>
- <https://courses.lumenlearning.com/boundless-biology/chapter/somatosensation/> [Accessed on: November 12, 2020]
- <https://www.merckmanuals.com/> [Accessed on: November 12, 2020]
- [https://en.wikipedia.org/wiki/Cutaneous\\_nerve](https://en.wikipedia.org/wiki/Cutaneous_nerve) [Accessed on: November 12, 2020]
- [https://med.libretexts.org/Bookshelves/Anatomy\\_and\\_Physiology](https://med.libretexts.org/Bookshelves/Anatomy_and_Physiology) [Accessed on: November 12, 2020]
- <https://lumenlearning.com/courses/> [Accessed on Nov 12, 2020]
- [https://en.wikipedia.org/wiki/Subcutaneous\\_tissue](https://en.wikipedia.org/wiki/Subcutaneous_tissue) [Accessed on Nov 12, 2020]
- Tammaro A, Cavallotti C, Gaspari AA, Narcisi A, Parisella FR, et al. 2012. Dopaminergic receptors in the human skin. *J Biol Regul Homeost Agents* 26(4): 789-795.
- Keren A, Gilhar A, Ullmann Y, Zlotkin-Frušić M, Soroka Y, et al. 2019. Instantaneous depolarization of T cells via dopamine receptors, and inhibition of activated T cells of Psoriasis patients and inflamed human skin, by D1-like receptor agonist: *Fenoldopam*. *Immunology* 158(3): 171-193. <https://doi.org/10.1111/imm.13109>
- Park ES, Kim SY, Na JI, Ryu HS, Youn SW, et al. 2007. Glutathione prevented dopamine-induced apoptosis of melanocytes and its signaling. *J Dermatol Sci* 47(2): 141-149. <https://doi.org/10.1016/j.jdermsci.2007.03.009>
- Roosterman D, Goerge T, Schneider SW, Bunnett NW, Steinhoff M. 2006. Neuronal control of skin function: the skin as a neuroimmunoendocrine organ. *Physiol Rev* 86(4): 1309-1379. <https://doi.org/10.1152/physrev.00026.2005>
- Reimann E, Kingo K, Karelson M, Reemann P, Loite U, et al. 2012. Expression profile of genes associated with dopamine pathway in vitiligo skin biopsies and blood sera. *Dermatology* 224(2): 168-176. <https://doi.org/10.1159/000338023>
- Nicolae I, Ene Nicolae CD, Schipor S, Tampa M, Matei C, et al. 2013. Dopamine-chemical mediator in atopic dermatitis. *Revista de Chimie* 64(10): 1201-1206.
- Cucchi ML, Frattini P, Santagostino G, Orecchia G. 2000. Higher plasma catecholamine and metabolite levels in the early phase of nonsegmental vitiligo. *Pigment Cell Res* 13(1): 28-32. <https://doi.org/10.1034/j.1600-0749.2000.130106.x>
- Iyengar B, Misra RS. 1987. Reaction of dendritic melanocytes in vitiligo to the substrates of tyrosine metabolism. *Acta Anat (Basel)* 129(3): 203-205. <https://doi.org/10.1159/000146400>
- Kennedy WR, Wendelschafer-Crabb G. 1993. The innervation of human epidermis. *J Neurol Sci* 115(2): 184-190. [https://doi.org/10.1016/0022-510x\(93\)90223-1](https://doi.org/10.1016/0022-510x(93)90223-1)
- Keren A, Gilhar A, Ullmann Y, Zlotkin-Frušić M, Soroka Y, et al. 2019. Instantaneous depolarization of T cells via dopamine receptors, and inhibition of activated T cells of psoriasis patients and inflamed human skin, by D1-like receptor. *Immunology* 158(3): 171-193. <https://doi.org/10.1111/imm.13109>
- Wardhana M, Windari M, Puspasari N, Suryawati N. 2019. Role of serotonin and dopamine in psoriasis: a case control study. *Open Access Maced J Med Sci* 7(7): 1138-1142. <https://doi.org/10.3889/oamjms.2019.267>
- Kurian MA, Li Y, Zhen J, Meyer E, Hai N, et al. 2011. Clinical and molecular characterisation of hereditary dopamine transporter deficiency syndrome: an observational cohort and experimental study. *Lancet Neurol* 10(1): 54-62. [https://doi.org/10.1016/S1474-4422\(10\)70269-6](https://doi.org/10.1016/S1474-4422(10)70269-6)
- Daubner SC, Le T, Wang S. 2011. Tyrosine hydroxylase and regulation of dopamine synthesis. *Arch Biochem Biophys* 508(1): 1-12. <https://doi.org/10.1016/j.abb.2010.12.017>
- Lowes MA, Kikuchi T, Fuentes-Duculan J, Cardinale I, Zaba LC, et al. 2008. Psoriasis vulgaris lesions contain discrete populations of Th1 and Th17 T cells. *Journal of Investigative Dermatology* 128(5): 1207-1211. <https://doi.org/10.1038/sj.jid.5701213>
- Shome S, Rana T, Ganguly S, Basu B, Choudhury SC, et al. 2011. Dopamine regulates angiogenesis in normal dermal wound tissues. *PLoS One* 6(9): e25215. <https://doi.org/10.1371/journal.pone.0025215>
- Park ES, Kim SY, Na JI, Ryu HS, Youn SW, et al. 2007. Glutathione prevented dopamine-induced apoptosis of melanocytes and its signaling. *J Dermatol Sci* 47(2): 141-149. <https://doi.org/10.1016/j.jdermsci.2007.03.009>