www.ijarp.com ISSN 2456-9992 Page: 01-14
International Journal Advanced Research Publications
Review Article
Volume: 02
Issue: 05

A TRANSITION OF NEVI MOLE TO MALENOCYTES (SKIN CANCER)

*Priya Sharma, Keshav Garg, Sanjiv Duggal

Global College of Pharmacy, Kanpur Khui, Anandpur Sahib, Punjab, India 140117

Article Received: 16 April 2026, Article Revised: 06 May 2026, Published on: 26 May 2026

***Corresponding Author: Priya Sharma**
Global College of Pharmacy, Kanpur Khui, Anandpur Sahib, Punjab, India 140117
DOI: https://doi-doi.org/101555/ijrpa.2402

ABSTRAC

Melanocytic nevi (moles) are common, benign proliferation of pigment-producing melanocytes, arising from genetic and environmental factors (UV exposure). They typically appear as symmetric, well-demarcated, colored spots (brown, pink, blue) during childhood or adolescence. Even though they are usually benign, moles can act as indicators or precursor lesions to melanoma, thus monitor them is crucial. Melanocytic nevi are benign skin lesion; they come in a variety of sizes and colors. A person with dysplastic nevus syndrome typically has 100 or more moles, some of which are larger than normal or abnormal. This condition is mostly inherited. This frequently increases the chance of developing melanoma, a dangerous kind of skin cancer. Melanocytes that have undergone transformation give rise to melanoma, a skin cancer. UV-induced DNA damage is partly responsible for melanoma's greatest mutational burden of any malignancy. Radiation therapy is used to eradicate any cancer cells that remain after surgical resection of localized melanoma, which is "curable." Immunotherapies that inhibit immunological checkpoints and targeted treatments that target elements of the MAPK signaling cascade have demonstrated impressive clinical outcomes [2].

INDEX TERMS: Nevi moles, malenoma, skin cancer, MAPK, melanocytes, dysplastic nevus.

INTRODUCTION TO NEVI MOLE AND MALENOMA

Nevi (moles) are common, generally benign skin growths formed by clusters of pigment-producing cells called melanocytes. It is also known as nevus. A melanocytic nevus, sometimes referred to as a mole, is a widespread, mostly benign, pigmented skin growth

made up of clusters of melanocytes. The majority of which range in hue from brown to black and are frequently round with defined borders, show up by the age of 20. They are usually benign, but the ABCDE method is an indication of cancer, such as asymmetry, irregular borders, color changes, or growth. Melanocyte is a pigment and proliferates in clusters. Congenital nevi are those that exist from birth. The majority are acquired, developing in childhood and adolescence as a result of genetics and sun exposure. People who have a family history of atypical moles, have fair skin, or have many moles (10-45) are more vulnerable.

If a mole exhibits ABCDE alterations, it should be examined:

Asymmetry: One side does not resemble the other due to asymmetry.
Border: Not well defined, scalloped, or irregular.

Color: Various hues or uneven tones.

Diameter: A pencil eraser or more than 6 mm.

Evolving: Changing in terms of size, shape, color, or symptoms like scabbing, bleeding, or itching.

The majority of congenital nevi moles is harmless and may only need to be watched for the emergence of skin cancer. Melanoma, an aggressive kind of skin cancer, is more likely to occur in those with large congenital nevi. Nevus cells are present in this kind of melanocytic tumor. A mole, which is mostly composed of a type of cell called a melanocyte, can be either subdermal (found beneath the skin) or a pigmented growth on the surface.

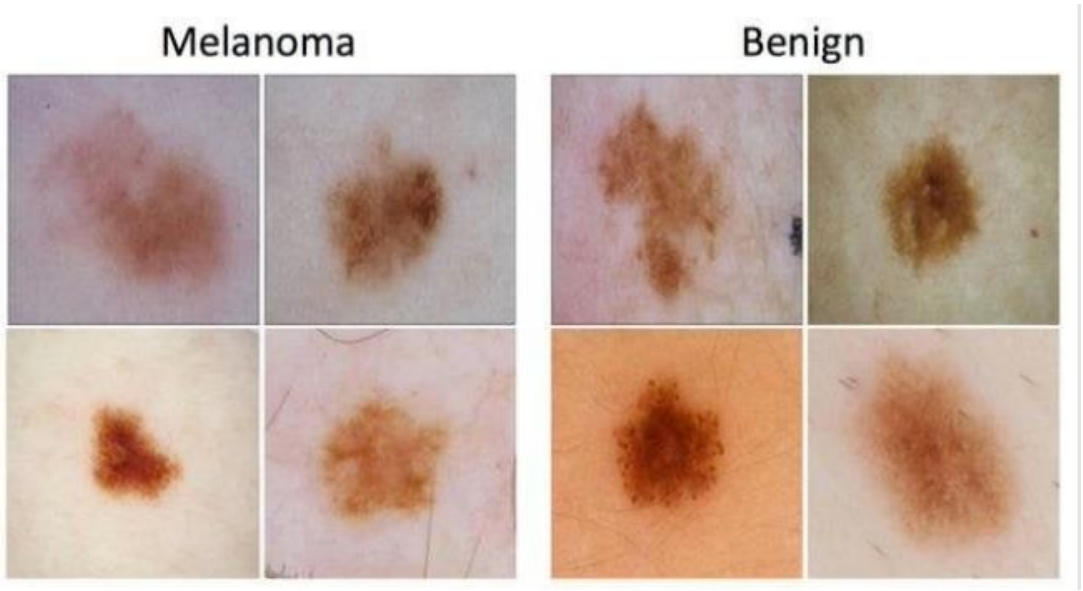


Fig 1.1: Melanocytic nevis (a benign stage of skin cancerous form).

It is associated with significant morbidity and mortality. A significant fraction of melanomas is associated with precursor lesions, benign clonal proliferations of melanocytes called nevi. Melanocytes, the cells that give skin its typical color, are believed to proliferate as a result of the abnormality. Certain parts of the body have aberrant skin pigmentation when melanocytes are created at a very high rate because they gather in clusters rather than dispersing uniformly.

A person with dysplastic nevus syndrome typically has 100 or more moles, some of which are larger than normal or abnormal. This condition is mostly inherited. This frequently increases the chance of developing melanoma, a dangerous kind of skin cancer. Compared to regular moles, dysplastic nevi have a higher risk of developing into cancer as shown in figure 2. Having more than 50 normal moles also raises the chance of melanoma, even though dysplastic nevi are widespread and many people have a few of these atypical moles.

However, dysplastic nevi are a risk factor for melanoma, and an individual's risk of acquiring melanoma increases with the number of dysplastic nevi they have According to research, a person with more than five dysplastic nevi is almost ten times more likely to develop melanoma than a person without any. [1]

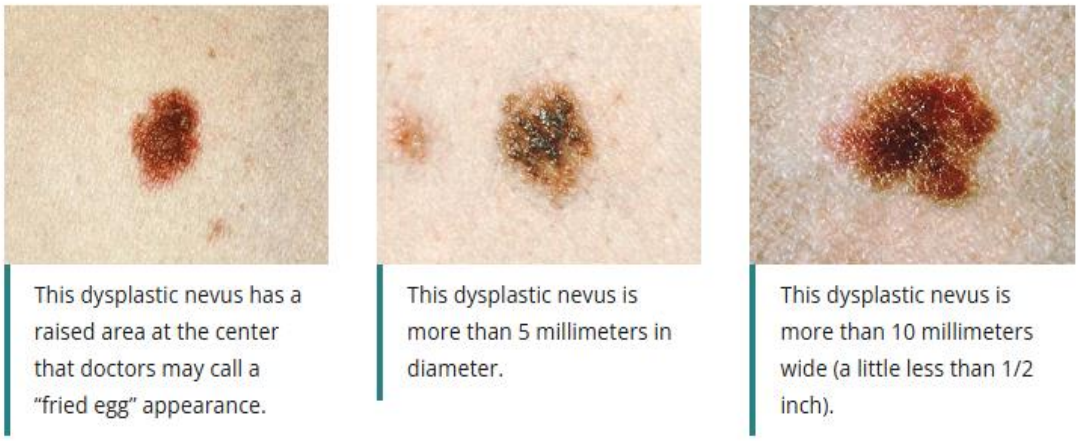
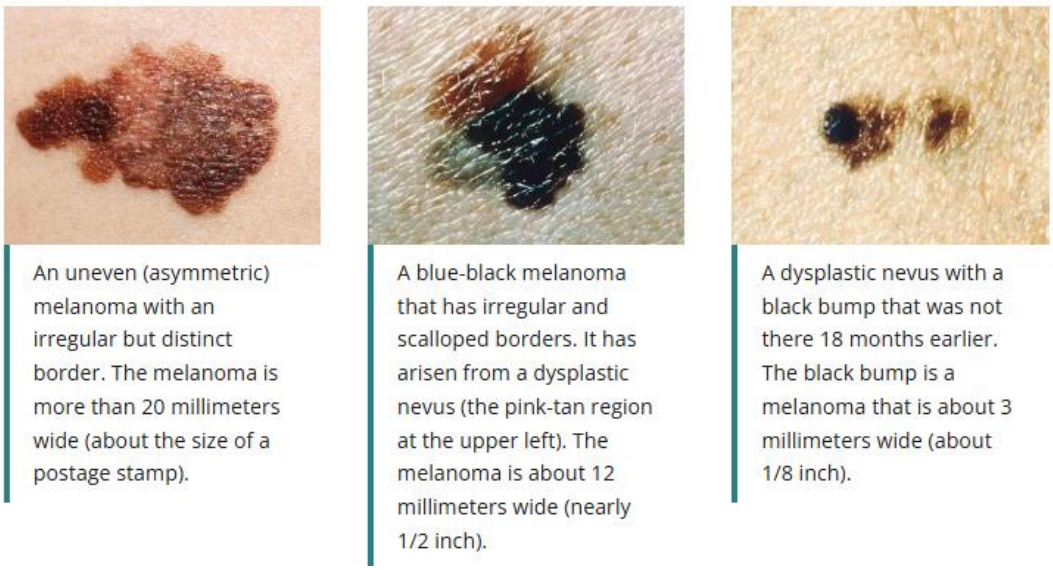


Fig 1.2: Dysplastic Nevus- Atypical mole



An uneven (asymmetric) melanoma with an irregular but distinct border. The melanoma is more than 20 millimeters wide (about the size of a postage stamp).

A blue-black melanoma that has irregular and scalloped borders. It has arisen from a dysplastic nevus (the pink-tan region at the upper left). The melanoma is about 12 millimeters wide (nearly 1/2 inch).

A dysplastic nevus with a black bump that was not there 18 months earlier. The black bump is a melanoma that is about 3 millimeters wide (about 1/8 inch).

Fig 1.3: A Malenoma (Serious cancer of skin).

LITERATURE REVIEW

Melanoma neoplasms have a strong tendency to spread, particularly to the brain, lungs, and lymph nodes Organs such the pancreas, bones, small intestine, and adrenal glands can develop metastases. While adjuvant therapies, chemotherapy, immunotherapy, and the use of vaccines are complementary choices for tumors in advanced stages, surgical excision remains the primary therapeutic method for early-stage melanoma.

Transition of Primary Malenoma to Metastatic Malenoma

Melanoma arises from aberrant melanocyte proliferation in the basal layer of the epidermis. The two types of pigments found in melanosomes of melanocytes are **pheomelanin (yellow/red) and eumelanin (brown/black).** The skin's main photoprotective pigment is eumelanin, whereas pheomelanin damages DNA by producing reactive oxygen species when exposed to UV light. By binding to the Melanocortin 1Receptor (CM1R), the melanocyte-stimulating binding hormone (α-MSH) increases the production of eumelanin by raising intracellular levels of cyclic adenosine monophosphate (cAMP) and activating the cAMP-response-element-binding protein (CREB).

When assessing suspected pigmented lesions, they observe for alterations in newly formed skin lesions and consider pigmentary variations, asymmetry, ill-defined margins, ulcer formation, nodules in the growth of pre-existing lesions, and pigment loss as potential indicators of melanoma.

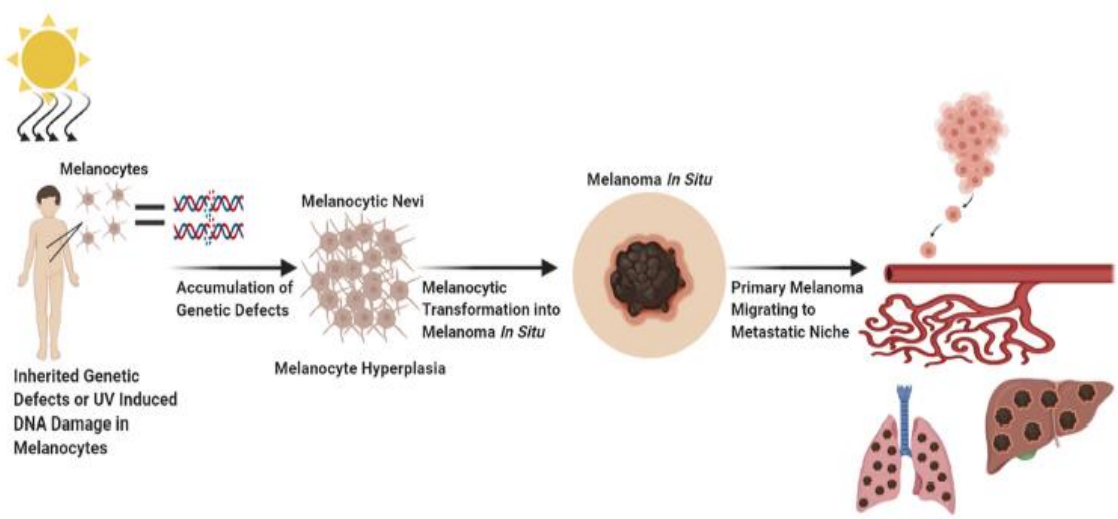


Fig. 2.1: Mechanism of transition of malenoma to metastatic malenoma.

All of these mutations contribute to different elements of melanocytic neoplasia, however some are regarded as driver mutations since they are likely to start the early stages of tumor formation, growth, and dissemination, as well as melanocytic transformation. Melanocyte hyperplasia and nevi formation are caused by an initiating driver mutation that is first acquired by a normal melanocyte.

BRAF mutations are frequently observed in melanocyte nevi (26–28). NRAS mutations can occasionally be discovered in nevi, particularly congenital nevi, but BRAF and NRAS mutations are often mutually exclusive.

Staging Phase of Malenoma

1. *Primary stage i and ii:* The T scale's key classification criteria are tumor thickness and ulceration (Fig. 1). Based on tumor thickness, primary melanomas are classified into four substages (T1–T4), with further sub-staging if ulceration is present. Based on tumor thickness and sub-stage in the presence of ulceration (stages I and II), primary melanomas are divided into four sub-stages, T1–T4. Individuals with stage III lymph node metastases, the lymph nodes' clinical state (palpable or not), and the primary prognostic indicators are the quantity of metastatic lymph nodes. The spread of melanoma beyond the epidermis and local lymph nodes is a characteristic of stage IV melanoma.

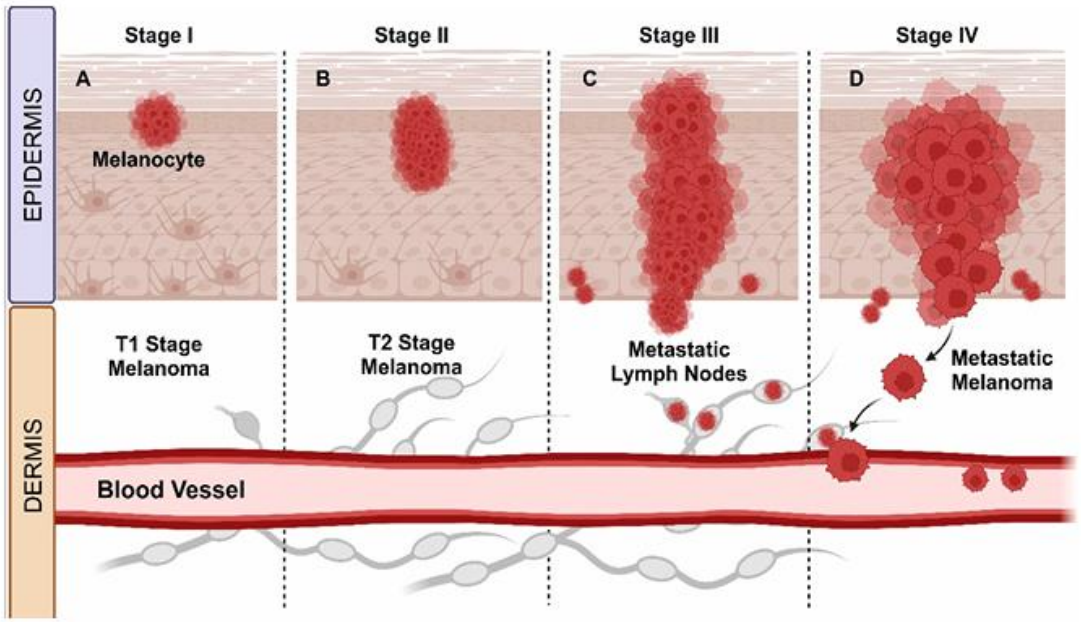


Fig 2.1: Stages of Malenoma (**A**) In first stage, the tumor is thin and less than 1 mm thick. (**B**) In stage II, the tumor is between 1 and 2 mm thick. (**C**) Presence of lymph node metastases. (**D**) Distant metastases are present.

Stage III and IV: The spread of cancer beyond the skin and lymph nodes to distant organs and tissues through distant metastases is a characteristic of stage IV malenoma. Melanoma can spread to the liver, lungs, brain, bones, or other organs at this advanced stage, which greatly complicates treatment and related issues. The prognosis and available treatments are significantly impacted by stage IV, which is typically regarded as an advanced stage of the disease.

Real World Insights

a) *Ana Paula De et al (2024)* published a study on skin malignancies, melanoma is known to be the most prevalent and aggressive malignant neoplasia. When melanocytes, which make pigment, proliferate abnormally in the basal layer of the epidermis, melanoma results. Pheomelanin (yellow/red) and eumelanin (brown/black) are the two types of pigments found in melanosomes which originates from melanocytes. The skin's main photoprotective pigment is eumelanin, whereas pheomelanin produces reactive oxygen species when exposed to UV light, which damages DNA [3].

b) *Nazeer Hasan et al (2023)* added that if skin cancer is not detected at an early stage, it is predicted to increase significantly over the next 20 years, posing a threat to the healthcare

system worldwide. Drug resistance, clinical success, and the discovery of innovative drugs are significant obstacles, despite the fact that it is curable at an early stage. The etiology of skin cancer, the process of cell proliferation, the factors influencing cell development, and the mechanism of drug resistance must all be understood in order to close the gap and provide an effective treatment. Understanding the structural diversity of skin cancers, current treatments (phytocompounds, chemotherapy, radiotherapy, photothermal therapy, surgery, combination therapy), molecular targets linked to cancer growth and metastasis, and a focus on nanotechnology-based methods for downregulating the harmful disease are the main topics of this article.

c) *Radomir M Slowinski et al (2022)* published in his article that melanin may be required for the malignant transformation of melanocytes and protects against the development of skin malignancies, including cutaneous melanoma. This demonstrates the intricate function that melanogenesis plays in the development of melanoma, which is characterized by the chemical characteristics of melanin and the nature of producing pathways like eu- and pheomelanogenesis. Pheomelanin, which is less photostable, can create a mutagenic environment following exposure to short-wavelength UVR, but eumelanin is thought to offer radioprotection and photoprotection by functioning as an effective antioxidant and sunscreen.

d) *Kevinn Eddy et al (2021)* concluded that UV-induced DNA damage is partly responsible for melanoma's greatest mutational burden of any malignancy. Radiation therapy is used to eradicate any cancer cells that remain after surgical resection of localized melanoma, which is "curable." Immunotherapies that block immunological checkpoints and targeted therapies that target components of the MAPK signaling cascade have demonstrated impressive clinical responses; however, most patients eventually develop resistance to these treatments due to disease relapse. An overview of melanocytic transformation into malignant melanoma and important molecular events that take place during this evolution are presented in this article.

e) *Sarah A Weiss et al (2019)* explored about genuine improvement in overall survival (OS) within a randomized phase III trial was not seen until anti-CTLA-4 (ipilimumab) was developed, despite numerous trials over decades using vaccines, cytokines, and cell therapies showing significant responses in a small subset of patients with metastatic disease. Anti-PD-1-based treatments (nivolumab, pembrolizumab) either alone or in combination with ipilimumab showed additional improvements in OS for metastatic illness.

The development and validation of predictive biomarkers in the metastatic setting, enhanced prognostic and predictive biomarkers for the adjuvant setting, comprehending the mechanisms of and reducing toxicity, and optimizing the duration of therapy are further objectives.

Detection test for Malenoma.

Sr. No	Test Method	Primary Use in Melanoma	Key Details & Strengths	Limitations
1	**Dermoscopy**	Early Detection (Skin)	Handheld magnifier (dermatoscope) used by dermatologists to identify, atypical patterns/structures of pigmented lesions.	Requires expertise; limited to surface/upper skin layers.
2	**Sonography (Ultrasound)**	Nodal/Local Staging	Real-time assessment of superficial lymph nodes. Superior for staging regional lymph nodes	User-dependent; poor visualization of deep or bone-covered areas
3	**PET-CT scan**	Distant Staging/Recurrence	Combines functional (PET) and anatomical (CT) data. Best for detecting widespread metastasis (soft tissue, lymph nodes, viscera).	Limited sensitivity for lesions <5–6 mm or brain lesions; high cost.
4	**CT scan**	Nodal/Visceral Staging	Imaging is generally reserved for thicker melanomas (> 4mm) Provides detailed cross-sectional images of the chest, abdomen, and pelvis	Uses ionizing radiation; less sensitive for small nodules compared to PET
5	**MRI Scan**	Brain/Soft Tissue Staging	MRI scans for skin cancer are primarily used to determine if advanced melanoma or other skin cancers have spread (metastasized) to internal organs, l	Longer acquisition time; higher cost; not ideal for whole-body imaging.

Future perspectives

Future perspectives on the transition of nevi (moles) to melanoma focus on enhancing early detection, understanding the molecular mechanisms behind this rare transformation. While most nevi remain benign, roughly 30%–50% of melanomas are associated with a pre-existing nevus, making this transition a critical area of research. Future research will focus on Identifying the precise molecular events that overcome the senescence (growth arrest) of common nevi.

This includes studying the role of mutations in tumor suppressor genes, such as \(CDKN2A\) and \(PTEN\). Studies suggest that nevi can activate immune surveillance, but that immune evasion plays a key role in the progression to melanoma. Future therapies may focus on enhancing the immune system's ability to recognize and eliminate these early-stage malignant cells.

Early detection greatly improves the prognosis of patients with melanoma. Owing to the cumulative nature of oncogenic mutations in melanocytic nevi, a fine-grained early morphologic footprint should be detectable by an algorithm trained with prospective data: Differentiated melanocytes of the skin are described to have long cell life cycles and are unable to undergo mitosis .Thus, melanocytic nevi may accumulate many mutations during their life cycle which are involved in various signalling cascades regulating proliferation (e.g. BRAF and NRAS), replication (TERT), cell cycle control (CDKN2A), metabolism (PTEN and KIT), apoptosis (TP53).

REFERENCES

1. National Cancer Institute Surveillance, Epidemiology, and End Results Program. Cancer Stat Facts: Melanoma of the Skin; 2025Available online: https://seer.cancer.gov/statfacts/html/melan.html.
2. Kevin Eddy, Raj Shah, Suzie Chen, Decoding Malenoma Development and Progression: Identification of Therapeutuc Vulnerabilities, 2021; 10: 1-13.
3. Ana Paula et al, Cutaneous Malenoma: An Overview of Physiological and Therapeutic Aspect and Biotechnological Use of Serine Protease Inhibitor; 2024, 29(16) https://doi.org/10.3390/molecules29163891
4. Nazeer Hasan et al, Skin Cancer: Understanding the Journey of transformation from conventional to Advanced treatment approaches, 2023 Oct 6;22(1):168. doi: 10.1186/s12943-023-01854-3
5. Radomir M Slominski et al, Malenoma, Melanin and Melanogenesis: The Yin and Yang Relationship 2022; 14:12:842496 doi: 10.3389/fonc.2022.842496.
6. Sarah A Weiss, Jedd D Wolchok, Mario Sznol, Immunotherapy of Malenoma: Facts and Hopes 2019; 25(17):5191-5201 doi: 10.1158/1078-0432.CCR-18-1550.
7. Culp MB, Lunsford NB. Melanoma among non-Hispanic Black Americans. *Preventing Chronic Disease* 2019;16:E79.
8. Rigel DS, Russak J, Friedman R. The evolution of melanoma diagnosis: 25 years beyond the ABCDs. *CA: A Cancer Journal for Clinicians* 2010; 60(5):301–316.

9. Siegel RL, Miller KD, Fuchs HE, Jemal A. Cancer statistics, 2022. *CA: A Cancer Journal for Clinicians* 2022; 72(1):7–33.
10. Sargen MR, Cahoon EK, Yu KJ, et al. Spectrum of non keratinocyte skin cancer risk among solid organ transplant recipients in the US. *JAMA Dermatology* 2022; 158(4):414–425.
11. Rollan MP, Cabrera R, Schwartz RA. Current knowledge of immunosuppression as a risk factor for skin cancer development. *Critical Reviews in Oncology/Hematology* 2022; 177:103754.
12. Bafounta ML, Beauchet A, Aegerter P, Saiag P. Is dermoscopy (epiluminescence microscopy) useful for the diagnosis of melanoma? Results of a meta-analysis using techniques adapted to the evaluation of diagnostic tests. Arch Dermatol. 2001;137:1343–1350. doi: 10.1001/archderm.137.10.1343.
13. Kittler H, Pehamberger H, Wolff K, Binder M. Diagnostic accuracy of dermoscopy. Lancet Oncol. 2002;3:159–165. doi: 10.1016/s1470-2045(02)00679-4.
14. Forsea AM, Del Marmol V, de Vries E, Bailey EE, Geller AC. Melanoma incidence and mortality in europe: New estimates, persistent disparities. Br J Dermatol. 2012;167:1124–1130. doi: 10.1111/j.1365-2133.2012.11125.x.
15. Erdmann F, Lortet-Tieulent J, Schüz J, Zeeb H, Greinert R, Breitbart EW, Bray F. International trends in the incidence of malignant melanoma 1953-2008-are recent generations at higher or lower risk? Int J Cancer. 2013;132:385–400. doi: 10.1002/ijc.27616.
16. Lallas A, Apalla Z, Chaidemenos G. New trends in dermoscopy to minimize the risk of missing melanoma. J Skin Cancer. 2012;2012:820474. doi: 10.1155/2012/820474. Tromme I, Sacré L, Hammouch F, Legrand C, Marot L, Vereecken P, Theate I, van Eeckhout P, Richez P, Baurain JF, Thomas L, Speybroeck N. Availability of digital dermoscopy in daily practice dramatically reduces the number of excised melanocytic lesions: Results from an observational study. Br J Dermatol. 2012;167:778–786. doi: 10.1111/j.1365-2133.2012.11042.x.
17. Argenziano G, Albertin i G, Castagnetti F, De Pace B, Di Lernia V, Longo C, Pellacani G, Piana S, Ricci C, Zalaudek I. Early diagnosis of melanoma: What is the impact of dermoscopy? Dermatol Ther. 2012;25:403–409. doi: 10.1111/j.1529-8019.2012.01482.x.
18. Calin MA, Parasca SV, Savastru R, Calin MR, Dontu S. Optical techniques for the noninvasive diagnosis of skin cancer. J Cancer Res Clin Oncol. 2013;139:1083–1104. doi: 10.1007/s00432-013-1423-3.
19. Puig S, Malvehy J. Monitoring patients with multiple nevi. Dermatol Clin. 2013;31:565–577. doi: 10.1016/j.det.2013.06.004.
20. Chen L, Dusza S, Grazzini M, Blum A, Marghoob A. Redefining the number needed to excise. Australas J Dermatol. 2013;54:310–312. doi: 10.1111/ajd.12039. Salerni G, Terán T, Puig S, Malvehy J, Zalaudek I, Argenziano G, Kittler H. Meta-analysis of digital dermoscopy follow-up

of melanocytic skin lesions: A study on behalf of the international dermoscopy society. J Eur Acad Dermatol Venereol. 2013;27:805–814. doi: 10.1111/jdv.12032. [DOI] [PubMed] [Google Scholar]

21. .Nakahara S., Fukushima S., Okada E., Morinaga J., Kubo Y., Tokuzumi A., Matsumoto S., Tsuruta-Kadohisa M., Kimura T., Kuriyama H., et al. MicroRNAs that predict the effectiveness of anti-PD-1 therapies in patients with advanced melanoma. J. Dermatol. Sci. 2020;97:77–79. doi: 10.1016/j.jdermsci.2019.11.010. [DOI] [PubMed] [Google Scholar]
22. Jones N., Nonaka T. Circulating miRNAs as biomarkers for the diagnosis in patients with melanoma: Systematic review and meta-analysis. Front. Genet. 2024;15:1339357. doi: 10.3389/fgene.2024.1339357. [DOI] [PMC free article] [PubMed] [Google Scholar]
23. Gaponova S., Patutina O., Sen'kova A., Burakova E., Savin I., Markov A., Shmendel E., Maslov M., Stetsenko D., Vlassov V., et al. Single Shot vs. Cocktail: A Comparison of Mono- and Combinative Application of miRNA-Targeted Mesyl Oligonucleotides for Efficient Antitumor Therapy. Cancers. 2022;14:4396. doi: 10.3390/cancers14184396. [DOI] [PMC free article] [PubMed] [Google Scholar]
24. Arnold M., Singh D., Laversanne M., Vignat J., Vaccarella S., Meheus F., Cust A.E., de Vries E., Whiteman D.C., Bray F. Global Burden of Cutaneous Melanoma in 2020 and Projections to 2040. JAMA Dermatol. 2022;158:495–503. doi: 10.1001/jamadermatol.2022.0160. [DOI] [PMC free article] [PubMed] [Google Scholar
25. Siegel RL, Miller KD, Fuchs HE, Jemal A. Cancer statistics, 2022. CA: A Cancer Journal for Clinicians 2022; 72(1):7–33. [PubMed Abstract]
26. Sargen MR, Cahoon EK, Yu KJ, et al. Spectrum of nonkeratinocyte skin cancer risk among solid organ transplant recipients in the US. JAMA Dermatology 2022; 158(4):414–425. [PubMed Abstract]
27. Rollan MP, Cabrera R, Schwartz RA. Current knowledge of immunosuppression as a risk factor for skin cancer development. *Critical Reviews in Oncology/Hematology* 2022; 177:103754.
28. Dummer R., Queirolo P., Guijarro A.M.A., et al. Atezolizumab, vemurafenib, and cobimetinib in patients with melanoma with CNS metastases (TRICOTEL): a multicentre, open-label, single-arm, phase 2 study. Lancet Oncol. 2022;23:P1145–P1155. doi: 10.1016/S1470-2045(22)00452-1.
29. Gondi V., Bauman G., Bradfield L., et al. Radiation therapy for brain metastases: an ASTRO Clinical Practice Guideline. Prac Radiat Oncol. 2022;12(4):265–282. doi: 10.1016/j.prro.2022.02.003.
30. Tang JD, Mills MN, Nakashima J, Dohm AE, Khushalani NI, Forsyth PA, Vogelbaum MA, Wuthrick EJ, Yu HM, Oliver DE, Liu JKC, Ahmed KA.J Clinical outcomes of melanoma brain metastases treated with nivolumab and ipilimumab alone versus nivolumab and ipilimumab with stereotactic radiosurgery. Neurooncol. 2024 Feb;166(3):431-440.

doi: 10.1007/s11060-023-04543-9. Epub 2024 Feb 3.PMID: 38310157

31. Wang JW, Feng YF, Liu JH.Front Immunol, CTLA-4 and PD-1 combined blockade therapy for malignant melanoma brain metastases: mechanisms, challenges, and prospects. 2025 Jul 1;16:1629879. doi: 10.3389/fimmu.2025.1629879. eCollection 2025.PMID:
32. Rosendahl C., Marozava A. A Handbook for Hunters of Skin Cancer and Melanoma. Scion Publishing Ltd.; Banbury, UK: 2019. Dermatoscopy and Skin Cancer. [Google Scholar]
33. Sainz-Gaspar L., Sánchez-Bernal J., Noguera-Morel L., Hernández-Martín A., Colmenero I., Torrelo A. Spitz Nevus and Other Spitzoid Tumors in Children—Part 1: Clinical, Histopathologic, and Immunohistochemical Features. Actas Dermosifiliogr. (Engl. Ed.) 2020;111:7–19. doi: 10.1016/j.ad.2019.02.011.
34. Trindade F.M., de Freitas M.L.P., Bittencourt F.V. Dermoscopic evaluation of superficial spreading melanoma. An. Bras. De Dermatol. 2021;96:139–147. doi: 10.1016/j.abd.2020.06.012.
35. Antisani C, Hamilton-Maikle PK, Di Segni M, Banvolgyi A, Paolino G, Gonzales S, Boostani M, Pellacani G, Paragh G, Kiss N (2025) Reddish/bluish benign lesions evaluated by dermus SkinScanner. J Ultrasound 10.1007/s40477-025-01066-z
36. Boostani M, Lallas A, Goldust M, Nádudvari N, Lőrincz K, Bánvölgyi A, Holló P, Wikonkál NM, Paragh G, Kiss N (2025) Diagnostic performance of multimodal large language models in distinguishing melanoma from nevi in clinical and dermoscopic images. JAAD Int 23(5):58–60
37. Reiter O et al. The long-term evolution of melanocytic nevi among high-risk adults. J Eur Acad Dermatol Venereol 36, 2379–2387 (2022). 10.1111/jdv.18470
38. Silva L, Martins da Cruz TM, Aparecido Gonçalves, MW, Moreira Gonçalves RN, Rocha dos Santos CR, et al. Oral blue nevus two case reports and literature review. *Revista Estomatología*. 2021 Sep 15. 29
39. ziveleka S, Georgaki M, Pettas E, Savva V, Papadopoulou E, Katafygiotis P, et al. Acquired Compound Melanocytic Nevus on the Palate of a Child: Report of a Case. *J Oral Maxillofac Res*. 2022 Jan-Mar. 13 (1):e5.
40. Dhanuthai K, Theungtin N, Theungtin N, Thep-Akrapong P, Kintarak S, Klanrit P, et al. Pigmented Oral Lesions: A Multicenter Study. *Eur J Dent*. 2022 May. 16 (2):315-319.
41. Amérigo-Góngora M, Machuca-Portillo G, Torres-Lagares D, Lesclous P, Amérigo-Navarro J, González-Cámpora R. Clinicopathological and immunohistochemical analysis of oral melanocytic nevi and review of the literature. *J Stomatol Oral Maxillofac Surg*. 2017 Jun. 118 (3):151-155.
42. Haenssle HA, Mograby N, Ngassa A, Buhl T, Emmert S, Schön MP, et al. Association of Patient Risk Factors and Frequency of Nevus-Associated Cutaneous Melanomas. *JAMA Dermatol*. 2016 Mar. 152 (3):291-8.

43. Popov H, Pavlov P, Stoyanov GS. Blue Nevi and Melanoma Arising in Blue Nevus: A Comparative Histopathological Case Series. *Reports (MDPI)*. 2025 Aug 1. 8 (3):
44. Tavares AT, Pereira A, Pimentel J, Prates M, Fonseca L, Marques MR, et al. Blue Nevus of the Hard Palate: The Importance of a Careful Examination in an Emergency Setting. *Case Rep Dermatol Med*. 2022. 2022:6329334.
45. Gellén E, Janka E, Tamás I, Ádám B, Horkay I, Emri G, et al. Pigmented naevi and sun protection behaviour among primary and secondary school students in an Eastern Hungarian, city. *Photodermatol Photoimmunol Photomed*. 2016 Mar. 32 (2):98-106.
46. Dika E, Starace M, Lambertini M, Patrizi A, Veronesi G, Alessandrini A, et al. Oral and nail pigmentations: a useful parallelism for the clinician. *J Dtsch Dermatol Ges*. 2020 Jan. 18 (1):7-14
47. Rosés-Gibert P, Perrot JL, Pérez-Anker J, Dorado Cortez C, Orte Cano C, Cinotti E, et al. Revolutionizing ex vivo Skin Imaging: 3D Characterization of Skin Tumors with ex vivo Line-Field Confocal Optical Coherence Tomography - A Pilot Study. *Dermatology*. 2025. 241 (3):230-239.
48. Deußing M. [Line-field confocal optical coherence tomography in melanocytic tumors]. *Dermatologie (Heidelb)*. 2025 Aug 18.
49. Zhang AD, Clovie J, Lazar M, Vashi NA. Treatment of Benign Pigmented Lesions Using Lasers: A Scoping Review. *J Clin Med*. 2025 Jun 5. 14 (11):
50. Alqahtani J. Nevus Spilus: A Review of Laser-Based Therapeutic Approaches. *Acta Inform Med*. 2025. 33 (2):158-161.
51. Tsai SY, Buta MR, Bojovic B, Mologousis MA, Anderson RR, Hawryluk EB, et al. Combination Laser Treatment in Procedural Management of Congenital Melanocytic Nevi. *Lasers Surg Med*. 2025 Apr. 57 (4):306-311.
52. Porrini R, Valente G, Colombo E, Cannas M, Sabbatini M. Non pigmented melanocytic nevus of the oral cavity: a case report with emphasis on the surgical excision procedures. *Minerva Stomatol*. 2013 Jan-Feb. 62 (1-2):43-9.
53. Moreno S, Soria X, Martínez M, Martí RM, Casanova JM. Epidemiology of Melanocytic Naevi in Children from Lleida, Catalonia, Spain: Protective Role of Sunscreen in the Development of Acquired Moles. *Acta Derm Venereol*. 2016 May. 96 (4):479-84.
54. Zhu G, Gordon S, Green AC, Martin NG, Duffy DL. Halving of Australian children's naevus counts 1992-2016 and change in sun behaviour. *Br J Dermatol*. 2025 Jun 17.
55. Tronnier M. Melanotische Flecke und melanozytäre Nävi. In: Plewig G, Ruzicka T, Kaufmann R, Hertl M. Braun-Falcos Dermatol. Venerol. Allergol. Springer Berlin Heidelberg, 7. Auflage, 2016.
56. Pérez ME, Bley C, Cárdenas C. Nevus of Ota, a classic presentation. Med Clin (Barc) 2019; 153: 92.

57. Müller CSL, Müller SG, Vogt T, Pföhler C. Current concepts of ectopic nodal inclusions with special emphasis on nodal nevi. JDDG J Dtsch Dermatol Ges 2021; 19: 1145–57.
58. oura E, Eliades PJ, Shannon K et al. Hereditary melanoma: Update on syndromes and management: Emerging melanoma cancer complexes and genetic counseling. J Am Acad Dermatol 2016; 74: 411–20; quiz 421–2.
59. Toussi A, Mans N, Welborn J, Kiuru M. Germline mutations predisposing to melanoma. J Cutan Pathol 2020; 47: 606–16.
60. Deinlein T, Michor C, HofmannWellenhof R et al. Die Bedeutung von Ganzkörperfotografie und sequenzieller digitaler Dermatoskopie bei der Überwachung von Patienten mit erhöhtem Melanomrisiko. J Dtsch Dermatol Ges 2020; 18: 692–8. Pampena R, Kyrgidis A, Lallas A. A metaanalysis of nevusassociated melanoma: Prevalence and practical implications. J Am Acad Dermatol 2017; 77: 938–945.e4.