Movement Disorders
CLINICAL PRACTICE

VIEWPOINT

Levodopa and Melanoma: Practical Recommendations for Parkinson's Disease—International Parkinson and Movement Disorder Society Scientific Issues Committee Viewpoint

Giorgia Sciacca, MD, PhD,[1] Inas Elsayed, PhD,[2,3] Santiago Perez-Lloret, MD, PhD,[4,5] Tiago F. Outeiro, PhD,[6,7,8] Vivek K. Unni, MD, PhD,[9,10] Jeffery H. Kordower, PhD,[11,12] Daniela Berg, MD,[13] Lorraine V. Kalia, MD, PhD, FRCPC,[14,15,16,*] on behalf of the MDS Scientific Issues Committee

The potential association between Parkinson's disease (PD), melanoma, and levodopa (L-dopa) use can be a source of uncertainty and concern in clinical practice. Several etiopathological hypotheses have been proposed, including a possible role of the protein α-synuclein that, in addition to being a major component of Lewy bodies in PD, is expressed in melanocytes.[1] However, a definitive mechanistic link between PD and melanoma has yet to be defined. Epidemiological data examining whether L-dopa treatment increases melanoma risk in PD patients have been contradictory, and a causal connection has not been established. Nonetheless, product monographs for L-dopa-containing medications continue to cite a history of melanoma as a contraindication and list malignant melanoma as a potential adverse effect. This discrepancy between regulatory language and the available evidence can create uncertainty for both clinicians and people with PD when initiating or continuing L-dopa therapy. The aim of this article is to provide practical recommendations for clinicians based on current evidence regarding the (1) risk of melanoma in PD independent of L-dopa treatment based on epidemiological evidence and shared pathobiological mechanisms, (2) potential risk of developing melanoma with L-dopa use in PD, and (3) safety of L-dopa treatment in PD patients with a history of melanoma.

Risk of Melanoma in PD

Epidemiological Studies Linking PD and Melanoma

Determining whether PD itself is associated with melanoma risk independent of dopaminergic therapy is challenging because most individuals initiate treatment soon after diagnosis. One approach has been to examine the bidirectional relationship between PD and melanoma by comparing melanoma risk after PD diagnosis with the risk of PD after a diagnosis of melanoma. This approach was used in a meta-analysis that included 24 studies comprising over 290,000 individuals with PD.[2] The pooled analysis showed that melanoma risk preceding PD diagnosis did not significantly increase compared to non-PD controls (odds ratio

[1]Department of Medical, Surgical Sciences and Advanced Technologies GF Ingrassia, University of Catania, Catania, Italy; [2]Faculty of Pharmacy University of Gezira, Wadmadani, Sudan; [3]Systems Biology Ireland, School of Medicine and Medical Science, University College Dublin, Dublin, Ireland; [4]Observatorio de Salud Pública, Calidad de Vida y Medio Ambiente, Facultad de Psicología y Psicopedagogía, Pontificia Universidad Católica Argentina, Consejo de Investigaciones Científicas y Técnicas (CONICET), Buenos Aires, Argentina; [5]Departamento de Fisiología, Facultad de Medicina, Universidad de Buenos Aires, Buenos Aires, Argentina; [6]Department of Experimental Neurodegeneration, University Medical Center Göttingen, Center for Bio-structural Imaging of Neurodegeneration, Göttingen, Germany; [7]Translational and Clinical Research Institute, Faculty of Medical Sciences, Newcastle University, Newcastle upon Tyne, United Kingdom; [8]Faculdade de Medicina e Ciências Biomédicas (FMCB), Universidade do Algarve, Faro, Portugal; [9]Department of Neurology and Jungers Center for Neurosciences Research, Oregon Health and Science University, Portland, Oregon, USA; [10]OHSU Parkinson Center, Oregon Health and Science University, Portland, Oregon, USA; [11]ASU-Banner Neurodegenerative Disease Research Center, Tempe, Arizona, USA; [12]School of Life Sciences, Arizona State University, Tempe, Arizona, USA; [13]Department of Neurology, University Hospital Schleswig-Holstein, Campus Kiel and Christian Albrechts-University of Kiel, Kiel, Germany; [14]Krembil Brain Institute, Toronto Western Hospital, University Health Network (UHN), Toronto, Ontario, Canada; [15]Division of Neurology, Department of Medicine, University of Toronto, Toronto, Ontario, Canada; [16]Tanz Centre for Research in Neurodegenerative Diseases, University of Toronto, Toronto, Ontario, Canada

***Correspondence to:** Dr. Lorraine V. Kalia, Krembil Brain Institute, Toronto Western Hospital, University Health Network (UHN), Toronto, ON, Canada; E-mail: lorraine.kalia@utoronto.ca
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[OR]: 1.09, 95% confidence interval [CI]: 0.78–1.54). In contrast, individuals with PD had a higher risk of developing melanoma compared with those without PD (OR: 2.43, 95% CI: 1.77–3.22). Similar findings were reported in a more recent meta-analysis.[3] These data do not establish an independent association between PD and melanoma, as postdiagnostic exposure to dopaminergic therapies could not be separated from disease-related effects.

Additional studies examining untreated patients or at-risk populations have not provided consistent evidence for an independent association. A population-based cohort study from Israel analyzed cancer incidence among 7125 individuals with PD using data from a health maintenance organization and a linked cancer registry.[4] Overall cancer incidence, including melanoma, did not differ from that of the reference population within a defined time window surrounding PD diagnosis. However, the analysis was limited to ~10 years around the time of diagnosis, which may have limited the ability to detect longer-term associations between PD and melanoma.

In contrast, other study designs have suggested a possible link. In a small prospective study, 65 individuals with malignant melanoma and 35 healthy controls were evaluated.[5] Compared with controls, melanoma patients exhibited increased substantia nigra hyperechogenicity and a higher nonmotor symptom burden. During 12 to 18 months of follow-up, probable or possible PD was diagnosed in 6 of 59 melanoma patients (10.2%) and 1 of 31 controls (3.2%), a difference that was not statistically significant. In the Parkinson Research Examination of CEP-1347 Trial (PRECEPT), 806 untreated early PD patients underwent regular dermatologic surveillance for a mean follow-up of 1.8 years (1467 patient-years).[6] Patients with a history of cancer, other than basal or squamous cell skin cancers, within 5 years prior to enrollment were excluded. Melanoma was diagnosed in 9 of 806 patients (1.1%), corresponding to 20.9 times the number of expected diagnoses based on external population estimates (95% CI: 9.6–39.7). Reliance on an external reference population and the relatively short follow-up limit interpretation.

An alternative epidemiological strategy to dissociate melanoma risk related to PD itself from that potentially related to L-dopa exposure is to examine for increased melanoma risk in relatives of individuals with PD. This approach was used with data from 2.3 million participants in the Utah Population Database and Utah Cancer Registry, which included 2998 with PD and 7841 with melanoma.[7] Because this genealogical resource contains family lineage information up to 15 generations, relative risks were evaluated in index patients and their first-, second-, and third-degree relatives. The relative risk of melanoma increased in individuals with PD (1.95, 95% CI: 1.44–2.59), as well as in their first- (1.23, 95% CI: 1.07–1.40) and second-degree relatives (1.12, 95% CI: 1.02–1.23), but not in their third-degree relatives (1.06, 95% CI: 0.99–1.12). Conversely, the relative risk of PD was elevated in individuals with melanoma (1.65, 95% CI 1.22–2.19) and in their first- (1.29, 95% CI: 1.12–1.48), second- (1.18, 95% CI: 1.07–1.30), and third-degree relatives (1.12, 95% CI: 1.04–1.20). This bidirectional risk in index patients and their relatives supports a shared pathobiological linkage between PD and melanoma, rather than an iatrogenic effect of L-dopa treatment, although limitations include potential incompleteness of registry data and lack of information regarding chronicity of PD and melanoma.

Taken together, methodological limitations and inconsistent findings across study designs preclude firm conclusions regarding the risk of melanoma in PD. Further well-powered studies with careful control for treatment exposure and long-term follow-up are needed to determine whether PD itself contributes directly to melanoma risk.

Shared Pathobiological Mechanisms

Although epidemiological studies do not conclusively demonstrate that PD itself increases melanoma risk independent of dopaminergic therapy, evidence of the potential bidirectional association between PD and melanoma described earlier is more consistent with shared pathobiological mechanisms than with an effect mediated only by L-dopa exposure. Supporting this possibility, several PD-associated genes, including *SNCA*, *LRRK2*, *PRKN*, *PINK1*, and *DJ-1*, are also dysregulated in cancer. Consistent genetic evidence, however, remains limited. Linkage disequilibrium score regression across large cancer cohorts and PD datasets from the International Parkinson Disease Genomics Consortium suggested possible shared genetic architecture between PD and several cancers.[8] In contrast, Mendelian randomization analyses have not supported a causal relationship between melanoma and PD.[9]

At the mechanistic level, a key intersection between PD and melanoma lies in tyrosine metabolism, as tyrosine serves as the precursor for dopamine synthesis in substantia nigra neurons and for melanin production mediated by tyrosinase in skin melanocytes.[10] Dopamine oxidation in neurons and melanogenesis in melanocytes each produce reactive intermediates and reactive oxygen species, linking these pathways to oxidative stress.[11] Dopaminergic neurons and melanoma cells share high metabolic demand and dependence on mitochondrial oxidative phosphorylation, increasing their vulnerability to reactive oxygen species.

Interestingly, α-synuclein provides an additional molecular connection. Beyond its neuronal expression, α-synuclein is expressed in melanocytes and melanoma cells, where it localizes to vesicular and melanosomal compartments and may influence intracellular trafficking and pigment organelle dynamics.[12] In melanocytes, α-synuclein interacts with tyrosinase and can influence melanin synthesis, suggesting that altered α-synuclein homeostasis could modulate melanogenesis while simultaneously sensitizing cells to oxidative injury.[13] Moreover, neuromelanin within substantia nigra neurons shares structural similarities with peripheral eumelanin and pheomelanin, including metal-chelating properties that may initially confer protection but ultimately enhance oxidative burden and inflammation through iron accumulation and redox cycling.[14] Collectively, these observations support a shared molecular framework linking dysregulated

catecholamine metabolism, α-synuclein homeostasis, and oxidative stress across neuronal and melanocytic lineages.

L-Dopa Use and Melanoma Risk in PD

Biological plausibility for an association between L-dopa and melanoma has been hypothesized because L-dopa serves as a shared intermediate in dopamine synthesis within dopaminergic neurons and melanin synthesis within melanocytes. Tyrosine hydroxylase, the enzyme that catalyzes the conversion of tyrosine to L-dopa, has been reported to be overexpressed in some melanoma cells,[15] a finding that could be interpreted as indirect support for a link between L-dopa metabolism and melanoma biology. Based on this, it has been speculated that exogenous L-dopa could enhance melanin production and thereby promote melanoma growth. Nevertheless, there is no evidence that exogenous L-dopa is incorporated into melanocytes in vivo. In vitro studies have even suggested potential toxicity of dopamine to melanocytes,[16] and high-dose L-dopa has been trialed unsuccessfully as a treatment for advanced melanoma without accelerating tumor growth.[17]

Case reports have raised concern that melanoma might be more frequent in PD patients treated with dopaminergic agents.[18–29] However, a case series of 54 PD patients with melanoma reported that 31% developed melanoma prior to initiating L-dopa therapy.[30] A literature review showed melanoma was most often diagnosed either before L-dopa initiation or after months to a few years of treatment onset,[31] a time frame inconsistent with the long latency typically required for melanoma development (10–40 years). Consistent with this, a review of 34 reported cases of melanoma in PD found that 9 were diagnosed with melanoma prior to L-dopa exposure and 23 after L-dopa initiation, with latencies ranging from 4 months to 17 years.[32]

Additional evidence derives from cohort and registry-based studies examining melanoma risk in relation to L-dopa use. A retrospective analysis of the DATATOP trial cohort, comprising ~800 patients with PD, demonstrated a higher incidence of melanoma compared with the general population but found no association between melanoma risk and L-dopa treatment.[33] Similarly, a large nationwide Danish registry study linking PD diagnoses, cancer registries, and prescription data reported that L-dopa use, cumulative dose, and treatment duration were not associated with melanoma risk.[34] Together, these studies provide strong epidemiologic evidence against a causal role for L-dopa in melanoma development.

Prospective clinical studies further support this dissociation between L-dopa exposure and melanoma risk. In a cohort of 1395 patients with idiopathic PD followed up for 5 years and evaluated by both movement disorders specialists and dermatologists, melanoma prevalence was higher than that in the general population but was independent of L-dopa treatment.[35] In another prospective study specifically designed to assess an association between L-dopa use and melanoma incidence, only 1 of 1099 patients had received L-dopa prior to the diagnosis of primary melanoma.[36]

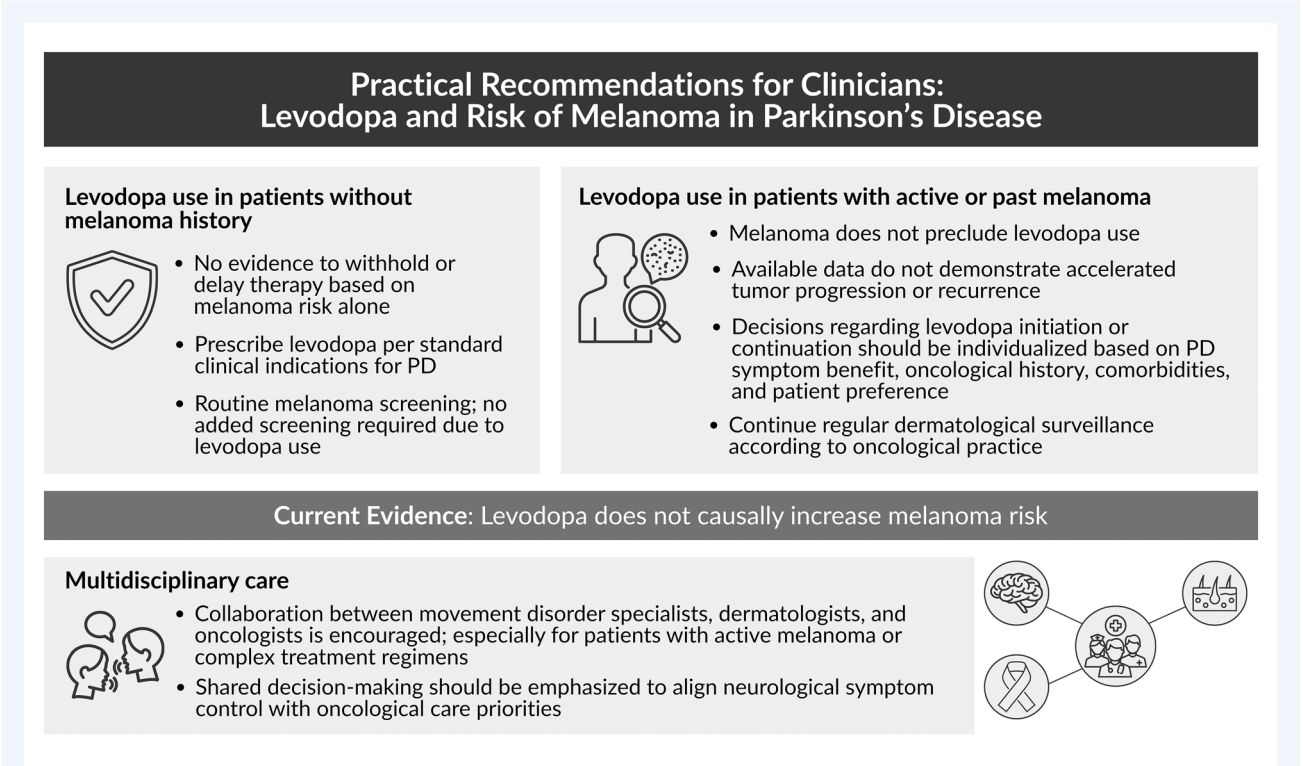

Fig 1. Summary of practical recommendations for clinicians about melanoma and Parkinson's disease.

More recent studies reinforce these conclusions. A Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA)–based systematic review found no evidence of a causal link between L-dopa and melanoma, although it recommended continued clinical vigilance due to the intrinsic melanoma risk associated with PD.[37] A large retrospective cohort study using health administrative data similarly concluded that the association between PD, melanoma, and dopaminergic therapy remains noncausal.[38] Taken together, clinical, epidemiologic, and systematic review data consistently suggest that the observed association between L-dopa therapy and melanoma is coincidental rather than causal.[6]

L-Dopa Use in PD Patients with a History of Melanoma

Several practical considerations, independent of any effect of L-dopa on melanoma itself, are relevant when prescribing L-dopa to patients with PD and melanoma or a prior history of melanoma. Concerns regarding the initiation or continuation of L-dopa in this population stem from theoretical considerations related to the shared melanin and dopamine biosynthetic pathways, as well as isolated clinical reports of hyperpigmentation and increased melanogenesis observed during L-dopa therapy.[37,39] However, there are reports of numerous PD patients with melanoma, including those with metastatic disease, who have been treated with L-dopa without evidence of accelerated tumor progression or recurrence.[32,40] Although available evidence does not support a causal association, definitive prospective safety data are lacking. Thus, based on the available evidence, the presence of melanoma or a history of melanoma does not, in itself, preclude L-dopa use in PD, although caution and close clinical monitoring remain appropriate.

Beyond considerations related to melanoma itself, L-dopa-associated adverse effects, including nausea, vomiting, orthostatic hypotension, and dyskinesia, may complicate management in patients already experiencing cancer-related symptoms or treatment-related toxicities.

Conclusions

Based on the available evidence, L-dopa treatment does not appear to causally increase melanoma risk. Nonetheless, uncertainty persists in clinical practice due to historical case reports, theoretical considerations, and regulatory language. Practical recommendations are provided to support clinicians in the management of PD in relation to melanoma risk (Fig 1).

With appropriate clinical assessment, routine dermatologic surveillance, and multidisciplinary collaboration, L-dopa can be used safely and effectively in most patients with PD, including those with melanoma or a history of melanoma. Ongoing prospective studies will be important to further refine these recommendations.

Author Roles

(1) Research Project: A. Conception, B. Organization, C. Execution; (2) Literature Review: A. Design, B. Execution; (3) Manuscript Preparation: A. Writing of the first draft, B. Review and Critique.

G.S.: 1A, 1B, 1C, 2A, 2B, 3A, 3B
I.E.: 1A, 1B, 1C, 2A, 2B, 3A, 3B
S.P.-L.: 1A, 1B, 1C, 2A, 2B, 3A, 3B
T.F.O.: 2A, 2B, 3A, 3B
V.K.U.: 2A, 2B, 3A, 3B
J.H.K.: 3B
D.B.: 3B
L.V.K.: 1A, 1B, 1C, 3A, 3B

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Data Availability Statement

Data sharing not applicable to this article as no datasets were generated or analysed during the current study. ■

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Supporting Information

Supporting information may be found in the online version of this article.

Data S1. COI_SIC_Melanoma.