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Review Article

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Segmental Vitiligo and Somatic Mosaicism: From Pathogenesis to Therapeutics

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ABSTRACT

Segmental vitiligo (SV) is a distinct clinical subtype of vitiligo characterized by unilateral, sharply demarcated depigmentation, rapid progression and early stabilization, and frequent leukotrichia. Compared with non-segmental vitiligo (NSV), SV is generally less strongly associated with systemic autoimmunity, suggesting potential differences in pathogenic mechanisms, although overlap phenotypes and mixed presentations have been reported. Accumulating clinical, epidemiological, and mechanistic evidence supports the concept that SV arises from somatic mosaicism, in which post-zygotic genetic or epigenetic alterations occur during embryogenesis and give rise to a clonally distinct melanocyte population distributed along developmental territories. Embryologic studies indicate that melanoblast migration from the neural crest, coupled with high proliferative activity during early development, creates a permissive context for mosaic mutation accumulation. The resulting melanocyte clones align with Blaschko's lines or other embryonic patterning units, accounting for the segmental distribution of SV. Beyond melanocytes, segment-restricted abnormalities in neural and vascular structures have also been reported, raising the possibility of broader neurocutaneous mosaicism, although whether these changes are primary or secondary to localized immune injury remains unresolved. Recognizing SV as a mosaic disorder has important clinical implications, particularly for early intervention, recurrence risk assessment, and the prominent role of autologous surgical therapies. Future advances will depend on multi-omics approaches to identify causal mosaic mutations, refined disease models, and long-term registries to translate developmental insights into precision management strategies.

Keywords: Mosaicism; Vitiligo

INTRODUCTION

Vitiligo is an acquired, chronic autoimmune depigmenting disorder affecting approximately 0.36% of the global population[1]. Current international consensus recognizes three major forms of vitiligo: non-segmental vitiligo (NSV), segmental vitiligo (SV), and undetermined or unclassified vitiligo[2]. NSV is typically characterized by symmetric or generalized depigmentation, a progressive disease course, and a strong association with systemic autoimmunity. In contrast, SV presents with a unilateral distribution, often stabilizes early after onset, and shows prominent follicular involvement, frequently manifested as leukotrichia[3]. Accumulating clinical, epidemiological, and mechanistic evidence—including earlier onset, lower familial aggregation, weaker association with systemic autoimmune comorbidities, and frequent leukotrichia—supports its classification as a distinct clinical subtype compared with NSV[4].

Nevertheless, both NSV and SV involve melanocyte-directed autoimmune responses, as evidenced by increased innate immune cytokines and $CD8^+$ T-cell-associated cytokines in active disease[5]. However, emerging evidence suggests that the two subtypes may differ in their initiating events and in the susceptibility of target melanocytes. In NSV, melanocytes exposed to oxidative stress and environmental triggers are thought to become immunologically vulnerable, whereas in SV, genetically predisposed or mosaic melanocytes may represent intrinsically susceptible targets.

Accordingly, while NSV has traditionally been investigated more extensively within an autoimmune framework, research efforts aimed at elucidating the distinct initiating mechanisms and segmental distribution of SV have increasingly expanded in recent years.

The skin provides an ideal model for studying such phenotypic manifestations on the body surface, which are far more readily detectable and interpretable than those arising in internal organs. In this context, growing evidence from epidemiological investigations and large-scale analyses of lesion distribution has gradually reshaped the prevailing conceptual framework of SV, leading to the emergence of somatic mosaicism as a unifying pathogenic paradigm[6-10].

Somatic mosaicism, suggesting that SV originates from post-zygotic genetic or epigenetic alterations in melanocyte lineage cells during embryogenesis[11]. These mosaic melanocytes are distributed along embryonic migration routes and form a genetically and functionally distinct melanocyte population that remains clinically silent until triggered by local immune or neurocutaneous factors[12]. This paradigm aligns SV with other mosaic skin disorders, such as nevus depigmentosus and segmental pigmentary anomalies, and provides a developmental framework for understanding its unique clinical behavior[9,10].

EMBRYOLOGIC AND DEVELOPMENTAL BASIS UNDERLYING PATTERNS OF SEGMENTAL VITILIGO

Neural crest cell development: melanocyte lineage differentiation and migration

SV exhibits a distribution pattern that closely mirrors the embryonic migration routes of melanocyte precursors. During embryogenesis, melanoblast precursors arise from the neural crest and migrate predominantly along a dorsolateral pathway toward the developing skin[13]. Evidence from somatic lineage-tracing studies indicates that the early embryonic period, characterized by rapid cell proliferation, is associated with substantially higher rates of somatic mutagenesis[14,15]. This migratory phase is also accompanied by extensive cellular proliferation, a process that inherently increases the likelihood of post-zygotic genetic mosaicism within the melanocyte lineage. The predilection of SV for lateral body regions, as opposed to the midline back, and its relatively lower frequency on the trunk compared with the lateral abdomen, further aligns with established embryonic melanoblast migration patterns[9].

In addition, a distinct subpopulation of adult melanocytes originates from precursor cells with dual Schwann cell-melanoblast potential, which migrate along a ventrolateral route. Experimental studies in murine models have demonstrated that approximately 65% of hair follicle melanocytes are derived from this alternative progenitor population[16,17]. Melanocytes arise from neural crest-derived precursors that populate both the interfollicular epidermis and the hair follicle niche. The presence of melanocyte stem cells within the hair follicle bulge region provides a potential source for perifollicular repigmentation observed in vitiligo[18,19]. Consistent with this notion, therapeutic interventions—particularly phototherapy—tend to yield more favorable outcomes when initiated early in the disease course, typically within the first six months after onset[20]. The embryologic differentiation and migration of melanocytes are demonstrated in **Fig. 1A**.

Errors occurring during melanoblast migration, proliferation, or differentiation can result in cell-specific or tissue-restricted mosaicism, leading to clonally distributed melanocyte populations. The characteristic SV distribution—often following Blaschko's lines rather than classical dermatomes—strongly supports disruption at this developmental stage[21].

Somatic mosaicism and embryonic lineage allocation

The somatic mosaicism hypothesis posits that post-zygotic mutations arising at distinct stages of embryonic development could influence both the extent and the timing of SV. Earlier mosaic events could generate a larger clone and broader segmental distribution, whereas later events may produce more localized patterns. The age at clinical onset may not directly reflect the timing of the mosaic event and may depend on secondary triggers and local immune context[22-24]. Importantly, SV shares parallel mosaic patterns with other segmental pigmentary disorders such as nevus depigmentosus, segmental lentiginosis and verrucous epidermal nevus, supporting a shared developmental origin rather than acquired systemic autoimmunity[9,25].

Cutaneous neurovascular mosaicism

Melanocytes and the nervous system share a common origin in the neural crest, and a mosaic mutation could affect both melanocytes and neural elements in a given region. Several studies have reported segment-restricted abnormalities not only in melanocytes but also in local neural and vascular compartments, raising the possibility that SV reflects a broader neurocutaneous mosaicism rather

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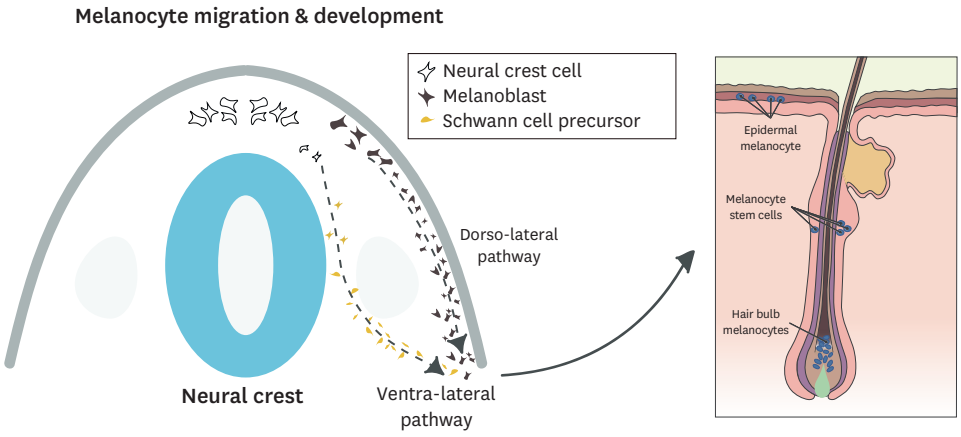

Fig. 1. Developmental origin of melanocytes and schematic illustration of melanocyte development and migration.

than a purely melanocyte-limited mosaic disorder[26-28]. However, it remains unclear whether these neurovascular changes are primary mosaic manifestations across multiple lineages or secondary to localized immune injury. Clinical studies have found focal neural abnormalities confined to SV-affected segments. The motor nerve conduction velocity in nerves supplying an affected limb was significantly slow compared to the contralateral normal side at stable status[26]. It suggests a regional demyelination or neuropathy confined to the SV segment. Notably, such changes were absent systemically, highlighting that the nervous system changes are mosaic rather than generalized. Additionally, skin blood flow studies have shown cutaneous hyperperfusion in SV patches—nearly a threefold increase in blood flow in lesional skin—along with heightened adrenergic (sympathetic) responses in the affected skin only[27]. There were no changes in circulating catecholamine levels or adrenergic receptor density systemically, indicating this hyperactivation is a local phenomenon. In a study of 140 patients, lesion distribution showed exact correspondence with underlying arterial blood supply territories, such as those of the facial artery, in 72% of cases[28]. This vascular alignment was supported by thermographic studies demonstrating statistically significant perfusion abnormalities in lesional skin, together with histological evidence of peri-arterial inflammatory infiltrates. In SV, not only melanocyte loss but also arterial perfusion patterns exhibit a segmental organization, collectively suggesting that embryonic vascular patterning may contribute to the spatial confinement and distribution of mutant melanocyte clones.

Taken together, the embryologic evidence paints SV as a cutaneous mosaic disorder: the patch reflects the clonal expansion of a mutant melanocyte population along developmental pathways, sometimes involving associated neural or vascular mosaicism in that region. Understanding this developmental basis is crucial, as it sets the stage for the localized nature of the disease and its unique behavior.

Age of onset and timing of mosaic events

The characteristically early age of onset of SV is highly consistent with a somatic mosaicism model, suggesting that pathogenic events may arise during early development[29]. However, age of onset alone cannot be considered definitive proof of mosaicism and may instead reflects the timing at which developmentally primed melanocyte clones become clinically manifest. Moreover, as SV can also present later in life, it remains uncertain whether such late-onset cases represents delayed clinical expression of somatic mosaicism acquired during early development or involve additional, distinct pathogenic mechanisms.

The alignment of SV with embryonic territories, reflected by its sharply demarcated segmental depigmentation, strongly suggests an underlying somatic mosaicism. During blastocyst and gastrulation stages, cells are allocated to different body halves and segments. The clinical manifestations of SV may reflect differences in the timing of postzygotic mosaic events, with earlier mutations predisposing to broader segmental involvement and later mutations resulting in more localized, Blaschkoid patterns[30,31]. A schematic illustration depicting how the timing of mutations influences the pattern and extent of lesions is presented in **Fig. 2**.

PATHOPHYSIOLOGY OF SEGMENTAL VITILIGO: MOSAIC MELANOCYTES MEET LOCAL IMMUNITY

Mosaic melanocyte vulnerability hypothesis

Decades of translational investigation into vitiligo have progressively refined our understanding of disease pathogenesis bringing somatic mosaicism to the forefront as a key paradigm in disease pathogenesis.

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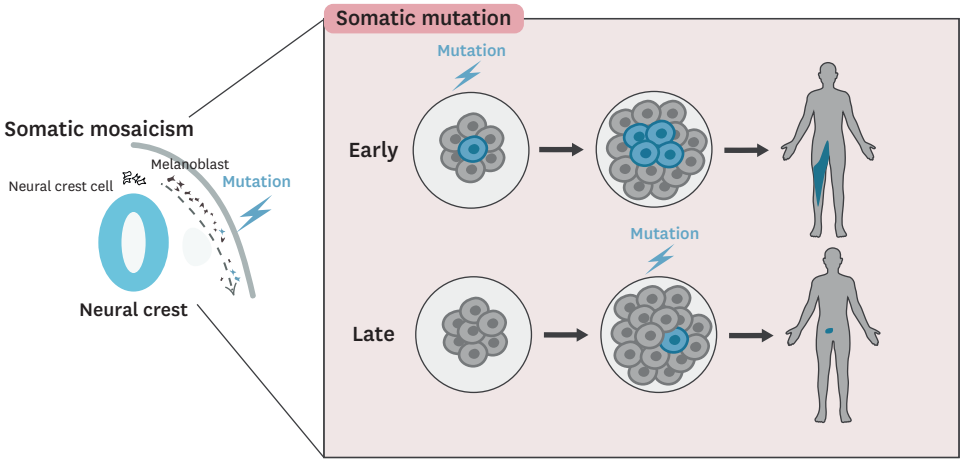

Fig. 2. Timing of genetic mutations and their contribution to somatic mosaicism.
Early postzygotic somatic mutation arising during early embryogenesis gives rise to a large clone of mutant cells, producing segmental or extensive mosaic patterns involving a substantial portion of the body. Late postzygotic somatic mutation occurring at a more advanced stage of embryonic development results in a smaller population of mutant cells, manifesting as a localized or focal mosaic phenotype.

To substantiate the mosaic hypothesis in SV, it is essential to demonstrate that melanocytes harboring mosaic alterations are intrinsically abnormal and/or exhibit heightened susceptibility to stress specifically within lesional skin, but not in nonlesional areas. In this context, Passeron et al.[32] reported that several stress-associated markers—including increased CXCL16 mRNA expression, elevated numbers of HSP70i-positive cells, and enhanced mitochondrial DNA release—were selectively enriched in lesional skin of patients with SV, while remaining unchanged in corresponding nonlesional sites[33]. These findings support the notion that mosaic alterations within the epidermis confer increased stress vulnerability in affected segments. Furthermore, the same study demonstrated that melanocytes isolated from lesional skin exhibited significantly higher expression of the pro-apoptotic chemokine receptor CXCR3B compared with melanocytes from healthy controls and from nonlesional skin of the same patients[32]. Overexpression of CXCR3B may be an intrinsic defect of mosaic melanocytes in SV, enhancing their responsiveness to chemokine signaling and contributing to the initiation of localized autoimmune responses.

The immune response appears to be highly localized and selectively directed against mosaic melanocytes. Histopathological analyses and flow cytometric studies of active SV lesions consistently demonstrate the accumulation of melanocyte antigen-specific $CD8^+$ T cells at the dermal-epidermal junction, exhibiting a pro-inflammatory effector phenotype characterized by robust interferon-γ and tumor necrosis factor-α production[34,35]. This immune response, is typically transient and confined. Unlike NSV, There is no systemic alterations in Treg frequency and function, melanoma specific autoantibodies in SV[33]. Clinically, inflammatory activity in SV generally stabilizes within 1–2 years, a course that is consistent with the notion that immune activation subsides once the vulnerable population of mosaic melanocytes within the affected segment has been eliminated[36].

Therefore, a clone of mosaic melanocytes with heightened stress sensitivity or altered antigenicity resides in the segment, setting the stage for focal melanocyte loss. This is in stark contrast to NSV, where the vulnerability is not limited to one cell clone—instead, a systemic autoimmune milieu can affect melanocytes body-wide. In SV, the melanocyte "weakness" is an inherent property of the cells in one region, likely due to that somatic mutation.

Neuro-immune-melanocyte axis

Beyond the inherent melanocyte abnormality, SV pathology involves a complex neuroimmune crosstalk. Neurovascular dysfunction could induce local immune responses and further exacerbate melanocyte vulnerability. As mentioned, studies have noted localized sympathetic hyperactivity in SV lesions—increased cutaneous blood flow and heightened responsiveness to adrenergic stimulation in the patch, without systemic cathecholamine change[27]. This suggests that the sympathetic nerves in the segment may be overactive or altered. Such localized sympathetic overdrive can influence immune activity: sympathetic nerve endings in the skin release neurotransmitters like norepinephrine (NE) and neuropeptide Y (NPY). Elevated local norepinephrine levels can modulate cytokine production and T-cell function within the skin. In patients with SV, transient surges of these neurotransmitters may be particularly deleterious within the affected segment. Increased local norepinephrine can engage β2-adrenergic receptors on both melanocytes and immune cells, thereby promoting oxidative stress and potentiating cytotoxic immune responses. Plasma NPY levels correlate with NE during stress[12], and these

mediators together can exacerbate melanocyte damage in vitiligo lesions. Moreover, NPY also modulates the hypothalamic-pituitary-adrenal axis to maintain emotional homeostasis[37].

Recent mechanistic work has begun to specify how cutaneous nerves can amplify melanocyte-directed autoimmunity. Spatial transcriptomic analyses of vitiligo lesions have demonstrated the dermal co-localization of calcitonin gene-related peptide (CGRP)-expressing nerve fibers with $CD8^+$ T cells and cDC1s, supporting a model in which CGRP enhances autoreactive T-cell responses within depigmented skin[38]. It may also be relevant to SV, however, SV-specific validation remains limited and warrants targeted investigation. Together, these observations underscore a coordinated neuroimmune network linking cutaneous perfusion, sympathetic nerve dysfunction, and regionally confined melanocyte loss.

Thus, a neuroimmune axis model emerges in which a mosaic neurochemical environment contributes to SV: the mutant melanocyte region might have an inherently hyperactive sympathetic tone, which under stress releases bursts of melanocyte-toxic neurotransmitters. This can precipitate or intensify the immune destruction of melanocytes in that segment.

Melanocyte-keratinocyte-neurovascular niche collapse

Unlike NSV, SV is frequently accompanied by leukotrichia[39,40]. Once the immune attack in SV has run its course, the affected segment is typically depigmented not only at the epidermal level but also in the hair follicles. The presence of leukotrichia in SV indicates that the melanocyte stem cell niche in the hair follicle has been obliterated or rendered non-functional in that area. In healthy skin or even NSV, hair follicle bulge regions harbor melanocyte stem cells that can regenerate pigment cells[41,42].

In NSV, follicular melanocyte stem cells are thought to contribute to repigmentation following therapy, suggesting relative preservation of this reservoir in many cases. In SV, the high frequency of leukotrichia may indicate deeper involvement of follicular melanocyte lineage cells, although direct evidence of immune-mediated stem cell targeting remains limited.

The result is a collapse of the regenerative niche: there are no reserve melanocytes left in the affected skin to repopulate the area, which explains why SV patches tend to persist without repigmentation. Moreover, the local keratinocytes and other cells in the SV segment may be affected by the inflammatory milieu and mosaic mutation. Keratinocytes in SV patches have been found to express high levels of CXCL16, hinting that they too respond abnormally to stress or may even carry the mosaic mutation if it occurred early enough to include multiple cell lineages[32]. The vascular changes in SV could influence the microenvironment by increasing local inflammatory cell trafficking or oxygen free radical generation. Over time, chronic catecholamine exposure from sympathetic hyperactivity might also cause microvascular damage. A comprehensive proposed pathomechanism encompassing the mechanisms discussed above in SV is illustrated in **Fig. 3**.

All these microenvironmental factors—absence of stem cells, altered keratinocyte signaling, and changed neural/vascular support—create a scenario where the melanocyte "soil" is inhospitable for regrowth. Even if some melanocytes remained or were reintroduced, the niche may fail to support their survival. This helps explain why conventional treatments have limited success in SV. Importantly, because SV's pathogenesis is multifactorial, it underscores that SV is pathophysiologically distinct from the straightforward autoimmune attack seen in NSV. However, in some cases, damage to melanocytes harboring somatic mosaicism may result in the exposure of antigens from otherwise normal melanocytes to the immune system. Once a systemic autoimmune response is initiated, this process may drive disease progression toward a NSV phenotype.

CLINICAL CHARACTERISTICS OF SEGMENTAL VITILIGO AND THEIR IMPLICATIONS FOR THERAPEUTIC STRATEGIES

Clinical patterns of SV

SV has historically been categorized using multiple distribution-based schemes, and accumulating mapping studies indicate that SV follows reproducible territorial patterns[6,7]. Importantly, large cohort studies integrating facial and truncal morphologies suggest that disease behavior and recurrence are also territory-linked, as relapses tend to occur within the same predefined distributional unit, supporting the utility of pattern-based subtyping for prognosis, follow-up, and mechanistic interpretation within a developmental mosaicism framework[8]. One of the most notable clinical differences between SV and NSV is the rate and pattern of recurrence after stabilization. Studies report low long-term recurrence rates in SV—on the order of 12%–20% in follow-ups—which is markedly lower than NSV. Additionally, when SV does recur or extend, it usually remains limited to the original affected body regions[7,8].

Therapeutic approaches based on pathogenic mechanisms and clinical characteristics

A guiding principle in SV is the urgency of early intervention. There is evidence that treating SV in early stage yields better outcomes than treating long-established lesions[43-45]. In clinical practice, a practical treatment algorithm for SV may involve the segmental diagnosis, followed by treatment with potent topical

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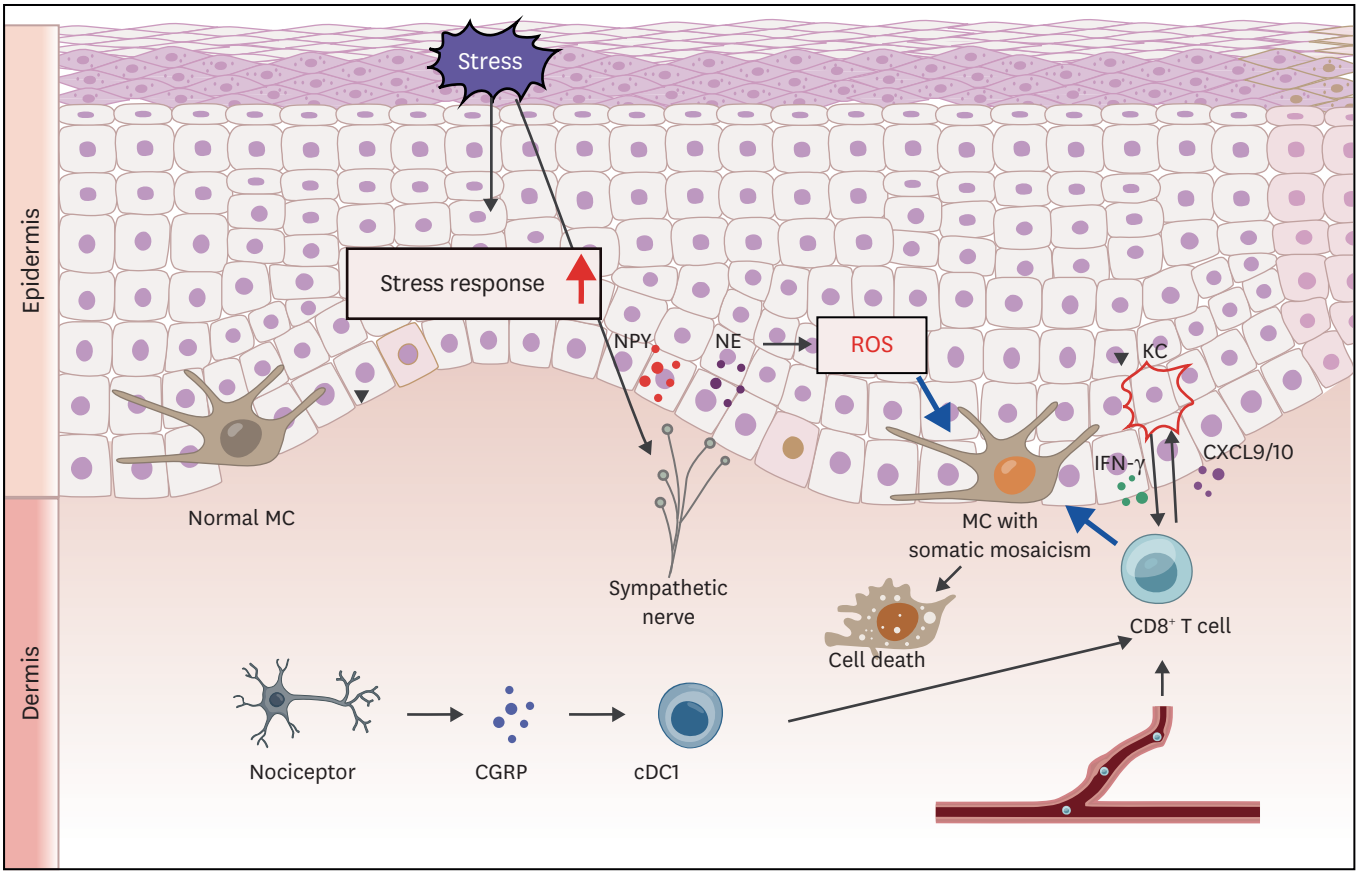

Fig. 3. Proposed pathogenesis of segmental vitiligo.
Schematic illustration of a proposed model for segmental vitiligo. Postzygotic somatic mutations give rise to a mosaic population of MCs with intrinsic susceptibility to cellular stress. These mosaic MCs exhibit increased oxidative stress, characterized by the accumulation of ROS, mitochondrial dysfunction with release of mitochondrial DNA, and induction of stress signals such as inducible heat shock protein 70. Local neurogenic factors released from sympathetic and sensory nerves, including N), NPY, and CGRP, further amplify oxidative stress and MC dysfunction. Stress-associated signals and damage-associated molecular patterns promote local immune activation through KC-derived chemokines and antigen-presenting cells, leading to the recruitment and activation of $CD8^+$ T cells. The combined effects of intrinsic vulnerability, neurogenic stress, and localized immune responses ultimately result in apoptotic loss of MCs within segmental vitiligo lesions.
ROS: reactive oxygen species, NE: norepinephrine, NPY: neuropeptide Y, CGRP: calcitonin gene-related peptide, MC: melanocyte, KC: keratinocyte, IFN: interferon, CXCL: C-X-C motif chemokine ligand, cDC: conventional dendritic cell.

steroids and/or phototherapy for up to 3–6 months in early-stage disease. If meaningful repigmentation is not achieved and the disease remains inactive, surgical grafting may then be considered to restore pigmentation. Indeed, SV is widely considered the ideal candidate for surgical repigmentation techniques. Although SV often reaches natural stabilization within 1–2 years, surgical intervention may be considered once no clinical signs of disease activity have been observed for at least 6 months, as supported by recent clinical outcome data[46]. Clinical evidence indicates that autologous surgical treatments for SV yield favorable and sustained long-term prognoses[45-49]. Reported success rates for surgical repigmentation in SV are high - many studies document that the majority of patients achieve >70%–80% repigmentation of the treated patch, especially on the face and trunk.[36,49] The key is that the disease is inactive, so the transplanted melanocytes are not attacked by the immune system and can survive.

Given that SV is thought to result from somatic mosaicism, leading to the local elimination of affected melanocytes through immune and neurovascular mechanisms, early loss of the melanocyte stem cell niche and minimal systemic autoimmune involvement with a low recurrence rate, SV is a suitable subtype for surgical treatment and early surgical intervention may be more efficient and effective than prolonged medical or targeted phototherapy in refractory cases.

CONCLUSION AND FUTURE DIRECTIONS

Future research in SV should prioritize defining its causal biology and translating this into improved patient care. Key directions include identifying the putative somatic mosaic mutations

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through deep multi-omics profiling of lesional versus nonlesional skin (including single-cell approaches), and developing experimental systems—such as iPSC-derived melanocyte platforms and animal lineage-tracing models—to test how mosaic melanocyte clones interact with local immunity and neurovascular cues. Clinically, comparative studies of early-onset versus adult-onset SV are needed to clarify whether age at presentation reflects differences in mutational timing, triggering factors, or immune context. Therapeutically, regenerative strategies that extend beyond conventional grafting—such as melanocyte stem cell–based approaches, bioengineered scaffolds, and eventually gene-corrected autologous melanocytes—represent promising avenues for durable repigmentation. Finally, artificial intelligence–assisted mapping of segmental patterns and the establishment of long-term, multicentre registries will be essential to refine phenotypic classification, quantify progression and recurrence, and validate prognostic biomarkers and treatment outcomes over time.

In conclusion, SV research is moving toward a more precise dissection of its causes and more effective, individualized therapies. SV serves as a model for mosaic autoimmunity, and insights gained here may apply to other mosaic inflammatory conditions. The ultimate vision is a world where we can identify an SV lesion's root cause at the molecular level, intervene before depigmentation becomes permanent, or even prevent SV from manifesting in those at risk. Integrating embryologic, genetic, and immunologic perspectives will be key to achieving this. With a clearer understanding and continued innovation in treatments, the distinct puzzle of SV is gradually being solved, piece by mosaic piece.

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CONFLICTS OF INTEREST

The authors have nothing to disclose.

DATA SHARING STATEMENT

Data available on request due to privacy/ethical restrictions.

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