

Defect-driven incoherent skin localization

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The process of dephasing during wave evolution has traditionally been viewed as an obstacle to localization, leading to diffusion even in strongly disordered Hermitian lattices. In contrast, here we demonstrate how the interplay of dephasing with non-Hermitian defects can be harnessed to engineer wave localization. Specifically, we identify a dynamical localization phenomenon characterized by wave function accumulation at the lattice's boundary due solely to dephasing, despite globally reciprocal couplings. Furthermore, we study the incoherent skin effect arising from coupling asymmetry and investigate the interplay between these antagonistic localization mechanisms. By reframing dephasing from a hindrance into a tool, this study overturns established paradigms of wave localization and paves the way for unconventional approaches to control localization phenomena in non-Hermitian physics.

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Introduction. Non-Hermitian physics [1] has garnered significant attention as a robust framework for describing open systems. Following the first observation of parity-time (PT) symmetry in optics [2–11], photonic lattices became the leading platform for engineering non-Hermitian Hamiltonians via tailored gain and loss. Beyond complex potentials, non-Hermiticity can also arise from asymmetric couplings, as in the Hatano-Nelson (HN) model [12,13]. The properties of the HN lattice exhibit pronounced sensitivity to boundary conditions; under open boundary conditions (OBCs), the system possesses a real spectrum and exhibits the non-Hermitian skin effect (NHSE) [14–18]; i.e., its eigenstates are exponentially localized at one lattice's boundary. In such lattices, wave propagation is unidirectional toward one edge, a phenomenon often termed the *dynamical* skin effect [19–27].

Recently, a plethora of studies have explored the NHSE beyond the HN model [28–41], including many-body [42–45], higher-dimensional and higher-order [46–50], and Floquet-driven [51–53] skin effects, with emphasis on their topological properties [18,54,55]. Experimentally, skin effects have been realized in optics using synthetic-dimension mesh lattices [56], laser arrays [57], and ring resonators [58], which effectively simulate asymmetric couplings. Beyond optics, NHSEs have also been demonstrated in acoustical [48,59], mechanical [60], and cold-atom platforms [61].

In a seemingly unrelated context, dephasing, i.e., the randomization of a wave function's phase, effectively modeling interactions between a system and its environment, is known to induce diffusion even in strongly disordered

conservative systems, thereby suppressing Anderson localization [62–72]. Remarkably, recent studies have shown that in complex-disordered non-Hermitian systems, dephasing can, rather than causing delocalization, enhance eigenmode localization [73–75].

In this Letter, we demonstrate that dynamical skin effects can be induced in non-Hermitian lattices with lossy defects via fast dephasing. We show that, despite reciprocal couplings, dephasing leads to asymmetric propagation through directional jumps between distant lattice regions, ultimately resulting in wave accumulation at one boundary. We further investigate the incoherent NHSE induced by coupling asymmetry in the HN model [76] and analyze the interplay between these two distinct skin-effect mechanisms. Our results provide a direct route for experimentally observing dynamical dephasing-induced skin effects and exploring their antagonism using readily accessible photonic mesh-lattice platforms.

Model and effects of dephasing. Our study begins with a one-dimensional symmetric lattice of N evanescently coupled waveguides, indexed by $n \in \{1, 2, \dots, N\}$, and described by the Hamiltonian:

$$H = \sum_{n=1}^N \epsilon_n |n\rangle \langle n| + \sum_{n=1}^{N-1} (|n+1\rangle \langle n| + |n\rangle \langle n+1|), \quad (1)$$

where we impose OBCs, $|0\rangle = |N+1\rangle \equiv 0$, and ϵ_n denotes the on-site potential at site n . Within the coupled-mode theory framework, the coherent evolution of $|\psi\rangle \equiv \sum_{n=1}^N \psi_n |n\rangle$ is governed by the equation $H|\psi\rangle = -i \frac{d|\psi\rangle}{dz}$, where ψ_n represents the complex amplitude of the electric field's envelope at site n . We will refer to the latter quantity as the wave function, for simplicity.

To incorporate dephasing into the system's dynamics, we modify the previous equation by adding a perturbation term $\Phi(z)$,

$$H|\psi\rangle + \Phi(z)|\psi\rangle = -i \frac{d|\psi\rangle}{dz}, \quad (2)$$

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which randomizes the wave function's phase at each lattice site at propagation intervals $z_a = al$ ($a \in \mathbb{N}$), where l is the dephasing period. Specifically, $\Phi(z) = \sum_a \Phi^{(a)} \delta(z - al)$ where $\Phi^{(a)} = \sum_n \theta_n^{(a)} |n\rangle\langle n|$ is a diagonal matrix of uncorrelated random phases $\theta_n^{(a)} \in [0, 2\pi]$. Under incoherent conditions, the evolution of the probability density $P_n(z) \equiv |\psi_n(z)|^2$, averaged over multiple realizations of the uncorrelated phases, is described by

$$P_n(z = al) = \sum_{m=1}^N (\mathcal{S}^a)_{nm} P_n(z = 0), \quad (3)$$

where $\mathcal{S}$, which is the incoherent propagator, has elements given by $\mathcal{S}_{nm}(l) = |U_{nm}|^2$, with $U \equiv e^{iHl}$. Since H is symmetric, $\mathcal{S}$ is Hermitian (see the Supplemental Material [77]), and its eigenvalue equation reads $\mathcal{S}|v_j\rangle = \beta_j|v_j\rangle$, with eigenvalues ordered as $\beta_1 \geq \beta_2 \geq \dots \geq \beta_N$.

From Eq. (3), we have

$$|P(z = al)\rangle = \sum_{j=1}^N d_{j,0} \beta_j^a |v_j\rangle, \quad (4)$$

where $|P(z)\rangle \equiv \sum_{n=1}^N P_n(z)|n\rangle$ and $d_{j,0} \equiv \langle v_j|P(0)\rangle$.

Thus, the projections $d_j(z) \equiv \langle v_j|P(z)\rangle$ evolve as $|d_j(z)| = |d_{j,0}| \beta_j^{z/l}$. A sudden *jump* occurs during the dynamics when the projection $|d_k|$ of an eigenstate $|v_k\rangle$ surpasses the projection $|d_r|$ of the previously dominant eigenstate $|v_r\rangle$, under the condition $\beta_k > \beta_r$ [75,78–80]. Consequently, after a critical distance z_{cr} , the eigenmode $|v_1\rangle$ associated with the largest eigenvalue β_1 dominates, yielding $P_f \equiv P(z > z_{\text{cr}}) \propto |v_1\rangle$. Although z_{cr} depends on the initial excitation, the asymptotic distribution P_f is uniquely determined by the Hamiltonian H and dephasing period l , irrespective of the initial condition.

For fast dephasing, i.e., $l \ll 1$, we can approximate $\mathcal{S}_{kj} \equiv U_{kj} U_{kj}^*$ up to $\mathcal{O}(l^2)$ as

$$\mathcal{S}_{kj} \approx \begin{cases} 1 + i(H_{kk} - H_{kk}^*)l \\ + (|H_{kk}|^2 - \frac{1}{2}[(H^2)_{kk} + (H^2)_{kk}^*])l^2, & k = j, \\ |H_{kj}|^2 l^2, & k = j \pm 1. \end{cases} \quad (5)$$

From these relations, it follows (see the Supplemental Material [77]) that for a uniform lattice ($\epsilon_n = 0$ for all n), $\mathcal{S}$ is tridiagonal with diagonal terms $\mathcal{S}_{kk} = 1 - 2l^2 + (\delta_{k,1} + \delta_{k,N})l^2$ and couplings $\mathcal{S}_{k,k\pm 1} = l^2$. Its eigenspectrum is given by

$$\beta_j = 1 - 2l^2 + 2l^2 \cos\left[\frac{(j-1)\pi}{N}\right],$$

$$|v_j\rangle \propto \sum_{n=1}^N \cos\left[\frac{(j-1)\pi}{N} \left(n - \frac{1}{2}\right)\right] |n\rangle \quad (6)$$

with $j = 1, \dots, N$.

Therefore, the largest eigenvalue is $\beta_1 = 1$ with eigenstate $|v_1\rangle \propto (1, 1, \dots, 1)^T$, while all other eigenvalues satisfy $\beta_j \in (1 - 4l^2, 1]$. As follows from Eq. (5), introducing real on-site deviations from uniformity (defects or disorder) leaves $\mathcal{S}$ unchanged up to $\mathcal{O}(l^2)$ [77]. Anderson localization is therefore fragile under dephasing, even strongly disordered lattices yield uniform asymptotic probability distribution $P_f \propto (1, 1, \dots, 1)^T$.

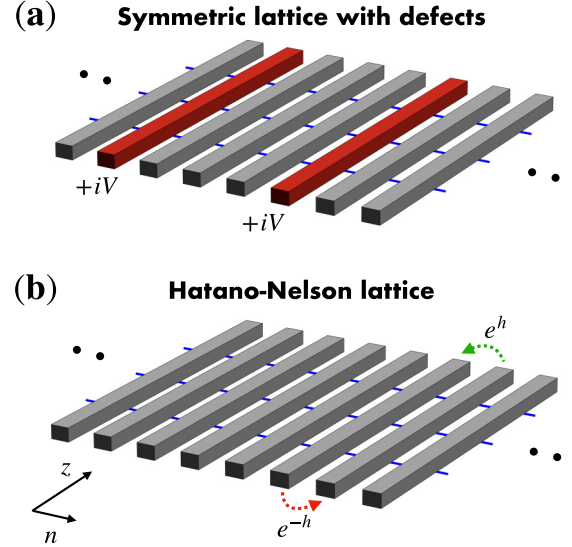


FIG. 1. Schematic representation of the waveguide lattice models examined in this work. (a) Lattice with symmetric couplings between neighboring sites, including a few on-site lossy defects. (b) Hatano-Nelson lattice with asymmetric couplings between neighboring sites. In both configurations, blue lines along the propagation direction z indicate the periodic action of *dephasing*.

This dynamical behavior is significantly altered when *imaginary* deviations from uniformity are introduced. Generally, in this work, we will consider a lattice having a finite number $r \geq 1$ of imaginary defects at sites q_j [as illustrated in Fig. 1(a)], i.e., $\epsilon_n = iV \sum_{j=1}^r \delta_{n,q_j}$ thereby being non-Hermitian ($H \neq H^\dagger$).

Single non-Hermitian defect. For a single imaginary defect at site q , the relative difference between the defect's diagonal element and the other (bulk) diagonal elements, normalized by the coupling terms, is

$$\Delta \equiv \frac{|\mathcal{S}_{qq} - \mathcal{S}_{kk}|}{l^2} = \left| 2V^2 - \frac{2V}{l} \right|, \quad (7)$$

which takes huge values for $l \ll 1$. Consequently, the defect eigenvalue, up to $\mathcal{O}(l^2)$, is given by

$$\beta_{\text{def}} \approx \mathcal{S}_{qq} \approx 1 - 2Vl + (2V^2 - 2)l^2, \quad (8)$$

and satisfies $\beta_{\text{def}} > 1$ for gain ($V < 0$) and $\beta_{\text{def}} < 1$ for loss ($V > 0$), while it corresponds to an almost single-site localized eigenvector, $|v_{\text{def}}\rangle \approx |q\rangle$.

For a gain defect ($V < 0$), the defect-induced eigenmode dominates the dynamics ($\beta_1 = \beta_{\text{def}}$), leading to a strongly localized asymptotic distribution $P_f \propto |q\rangle$ after a critical distance z_{cr} , which depends on the initial condition $\psi_n(z = 0)$ [77].

In contrast, the presence of a lossy defect ($V > 0$) leads to richer dynamics, effectively dividing the lattice into two segments: sites 1 to $q-1$ and $q+1$ to N , while the defect acts like a *hard wall*. As derived analytically in the Supplemental Material [77], the long-term probability distribution $P_f \propto |v_1\rangle$ predominantly occupies the largest segment $L \equiv \max(q-1, N-q)$, and vanishes elsewhere, irrespective of the initial condition $\psi_n(0)$. Within this region, it is shown

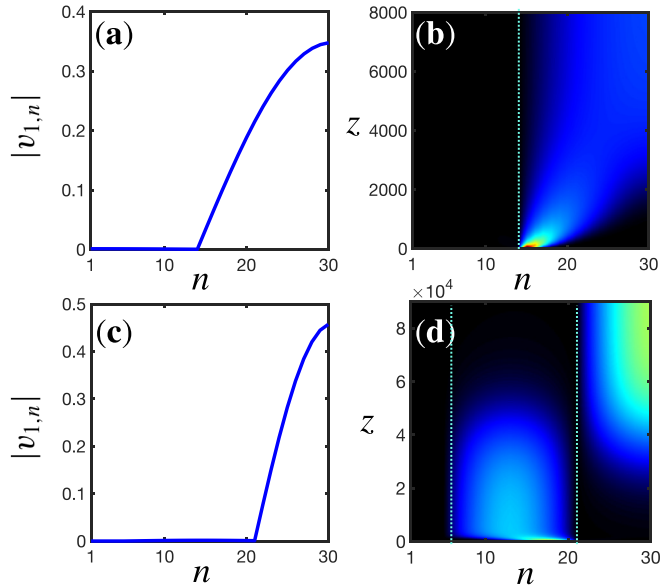


FIG. 2. Incoherent dynamics in a lattice of $N = 30$ sites with dephasing period $l = 0.01$. Top row: a single lossy defect of strength $V = 0.5$ at site $q = 14$, i.e., $\epsilon_n = iV \delta_{n,14}$. Bottom row: two identical lossy defects of strength $V = 0.5$ at sites $q_1 = 5$ and $q_2 = 21$, i.e., $\epsilon_n = iV(\delta_{n,5} + \delta_{n,21})$. (a), (c) Amplitude $|v_{1,n}|$ of the asymptotically dominant eigenmode of $\mathcal{S}$. (b), (d) Evolution of the averaged probability density $P_n(z)$ (normalized at each z) for initial conditions $\psi_n(0) = \delta_{n,15}$ in (b) and $\psi_n(0) = \delta_{n,20}$ in (d). Defect sites are indicated by blue dashed lines in (b) and (d).

in the Supplemental Material [77] that the elements $v_{1,n} \equiv \langle n | v_1 \rangle$ are given by

$$v_{1,n} \propto \sin\left(\frac{\pi}{2L+1}|n-q|\right) \quad (9)$$

in excellent agreement with the numerical calculations, for large enough V .

A pertinent example is shown in Fig. 2, for a lattice of $N = 30$ sites with an imaginary defect, $\epsilon_n = 0.5i\delta_{n,14}$, and $l = 0.01$. The lossy defect induces a localized eigenmode with eigenvalue $\beta_{\text{def}} = \beta_{30} = 0.9898$, distinctly outside the unperturbed band (ranging between 0.9996 and 1). As predicted analytically, the eigenmodes $|v_j\rangle$ exhibit spatial asymmetry, while the eigenmode $|v_1\rangle$ corresponding to the largest eigenvalue aligns perfectly with Eq. (9) [Fig. 2(a)] [77]. Consequently, despite symmetric couplings, the averaged probability density exhibits asymmetric propagation toward $|v_1\rangle$, as illustrated in Fig. 2(b) for the single-channel initial excitation $\psi_n(0) = \delta_{n,15}$. This dephasing-induced asymmetry in the dynamics forms the basis for engineering dynamical incoherent skin effects in non-Hermitian systems.

Two or more defects and dynamical skin effects. We now consider a lattice with two identical lossy defects at positions q_1, q_2 , i.e., $\epsilon_n = iV(\delta_{n,q_1} + \delta_{n,q_2})$ with $V > 0$. As before, the defect-induced eigenvalues follow Eq. (8). The eigenstate $|v_1\rangle$ associated with the largest eigenvalue β_1 depends strongly on the defect positions. Physically, the lattice partitions into three segments: the interdefect region of length $M = q_2 - q_1 - 1$ and the left/right edge segments of length $E_L = q_1 -$

1, $E_R = N - q_2$. As shown analytically under the hard-wall approximation [77], asymptotic localization occurs at the largest edge if $M < 2\max\{E_L, E_R\}$ (forming an *edge-well* state), and within the interdefect region if $M > 2\max\{E_L, E_R\}$ (forming a *bulk-well* state). Quantitatively, the eigenmode's nonzero region is given by

$$v_{1,n} \propto \begin{cases} \sin\left[\frac{\pi(n-q_2)}{2(N-q_2)+1}\right], & M < 2E_R \text{ with } E_R = \max\{E_L, E_R\}, \\ \sin\left[\frac{\pi(q_1-n)}{2(q_1-1)+1}\right], & M < 2E_L \text{ with } E_L = \max\{E_L, E_R\}, \\ \sin\left[\frac{\pi(n-q_1)}{q_2-q_1}\right], & M > 2\max\{E_L, E_R\}. \end{cases} \quad (10)$$

A representative example of this behavior is shown in the bottom row of Fig. 2, which depicts the evolution of the probability density $P(z)$ for an initial excitation $\psi_n(0) = \delta_{n,20}$ on a lattice of $N = 30$ sites with two identical lossy defects ($V = 0.5$) and a dephasing period $l = 0.01$. For defects placed at $q_1 = 5$ and $q_2 = 21$, we have $M = 15$, $E_L = 4$, and $E_R = 9$, satisfying $M < 2E_R$; thus, the eigenmode $|v_1\rangle$ localizes at the right edge [Fig. 2(c)], and the asymptotic probability distribution P_f is confined near the right boundary [Fig. 2(d)]. Similar examples for left-edge localization are provided in Ref. [77]. Figure 2(d) shows a jump in the probability distribution, marking the crossover from an initially dominant eigenmode to the asymptotically dominant state $|v_1\rangle$. Notably, when the defect's separation is sufficiently large, satisfying $M > 2E_L$, the wave function localizes between the two defects, forming a bulk-well state with defects effectively acting as impenetrable walls [77].

Generalizing the two-defect case, we now consider an N -site lattice with r lossy defects of strength V , placed at sites $q_1, \dots, q_r$. We define the edge-well lengths as $E_L = q_1 - 1$ and $E_R = N - q_r$, and the interdefect bulk-well lengths as $M_k = q_{k+1} - q_k - 1$, $k = 1, \dots, r-1$. Denoting $M_{\text{max}} \equiv \max M_k = q_{j+1} - q_j - 1$, the eigenstate $|v_1\rangle$ is given by

$$v_{1,n} \propto \begin{cases} \sin\left[\frac{\pi(n-q_r)}{2(N-q_r)+1}\right], & M_{\text{max}} < 2E_R \text{ with } E_R = \max\{E_L, E_R\}, \\ \sin\left[\frac{\pi(q_1-n)}{2(q_1-1)+1}\right], & M_{\text{max}} < 2E_L \text{ with } E_L = \max\{E_L, E_R\}, \\ \sin\left[\frac{\pi(n-q_j)}{q_{j+1}-q_j}\right], & M_{\text{max}} > 2\max\{E_L, E_R\}, \end{cases} \quad (11)$$

in the respective edge or bulk segment where the mode is nonzero. This expression illustrates clearly how the relative positions of multiple defects determine whether the asymptotic dominant mode manifests as an edge- or bulk-well state.

A pertinent engineered configuration is presented in the top row of Fig. 3 for an $N = 30$ -site lattice with four identical lossy defects ($V = 0.5$) located at sites $q_i = \{3, 10, 18, 26\}$ and dephasing period $l = 0.01$. Here, since $M_{\text{max}} < 2E_R$, the eigenmode $|v_1\rangle$ forms a tightly confined right edge state [Fig. 3(a)]. In Fig. 3(b), we show the evolution of the average probability density $P(z)$ for an initial excitation $\psi_n(0) = \delta_{n,1}$. Notably, the probability density shifts toward the right edge through a sequence of directional jumps, demonstrating a pronounced dynamical incoherent skin effect. This effect in the asymptotic probability density P_f arises irrespective of

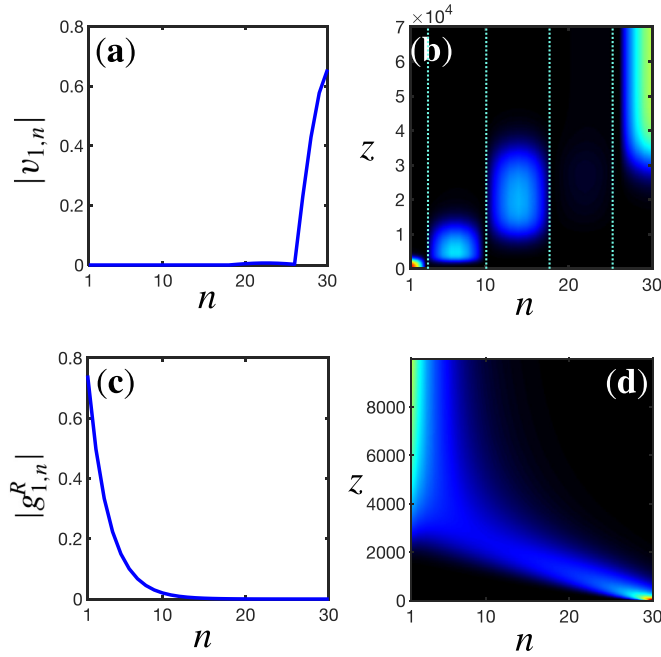


FIG. 3. Dynamical skin effects under incoherent dynamics in a lattice of $N = 30$ sites with dephasing period $l = 0.01$. Top row: symmetric couplings with four lossy defects of strength $V = 0.5$ at sites $q_i = \{3, 10, 18, 26\}$. Bottom row: Hatano–Nelson lattice with asymmetric couplings ($h = 0.2$) and no defects. (a), (c) Amplitude of the asymptotically dominant eigenmode: $|v_{1,n}|$ in (a) and $|g_{1,n}^R|$ in (c). (b), (d) Evolution of the averaged probability density $P_n(z)$ (normalized at each z) for initial conditions $\psi_n(0) = \delta_{n,1}$ in (b) and $\psi_n(0) = \delta_{n,30}$ in (d). In (b), defect sites are marked by blue dashed lines.

the initial excitation and results from the synergistic action of dephasing and the non-Hermitian lossy defects. Without dephasing, the asymptotic dynamics are dominated by the Lifshitz tails of the spectrum [81], which correspond to extended states, yielding diffraction across the lattice (see Ref. [77]). Thus, this defect-engineered skin effect emerges exclusively under incoherent dynamics. Notably, since the coexistence of real disorder leaves $\mathcal{S}$ unchanged up to $\mathcal{O}(l^2)$, the reported incoherent skin effect is robust to any level of disorder. The antagonism of this phenomenon with the conventional non-Hermitian skin effect in HN lattices under dephasing is studied next.

Dephasing in Hatano–Nelson lattices. Here, we consider a HN lattice with OBCs, under dephasing, schematically shown in Fig. 1(b). Its Hamiltonian $\tilde{H}$ reads

$$\tilde{H} = \sum_{n=1}^N \epsilon_n |n\rangle \langle n| + \sum_{n=1}^{N-1} (e^{-h} |n+1\rangle \langle n| + e^h |n\rangle \langle n+1|), \quad (12)$$

where $h > 0$ is introducing non-Hermiticity via hopping asymmetry. Since $\tilde{H}$ is nonsymmetric, the incoherent propagator Σ , with elements $\Sigma_{kj} = |(e^{i\tilde{H}l})_{kj}|^2$, is non-Hermitian. Its right- and left-eigenvalue problems read as $\Sigma |g_j^R\rangle = \lambda_j |g_j^R\rangle$ and $\Sigma^\dagger |g_j^L\rangle = \lambda_j^* |g_j^L\rangle$, respectively, where the biorthogonality

condition $\langle g_k^L | g_j^R \rangle = \delta_{kj}$ holds. From Eq. (3), it follows that

$$|P(z = al)\rangle = \sum_{j=1}^N c_{j,0} \lambda_j^a |g_j^R\rangle, \quad (13)$$

where $c_{j,0} \equiv \langle g_j^L | P(z = 0) \rangle$.

The HN Hamiltonian $\tilde{H}$ relates to Hamiltonian H [Eq. (1)] through the similarity transformation $\tilde{H} = \Omega^{-1} H \Omega$, with $\Omega = \sum_{n=1}^N e^{hn} |n\rangle \langle n|$. It follows that the incoherent propagator Σ can be written as $\Sigma_{kj} = |(e^{i\tilde{H}l})_{kj}|^2 = e^{2h(j-k)} \mathcal{S}_{kj}$. Hence, Σ and $\mathcal{S}$ are similar, $\Sigma = \Omega^{-2} \mathcal{S} \Omega^2$, and thus share the same eigenvalue spectrum. In the absence of on-site deviations from uniformity (i.e., $\epsilon_n = 0$ for all n), the eigenvalues λ_j of Σ coincide with the eigenvalues β_j of $\mathcal{S}$ given in Eq. (6), while the right- and left-eigenvectors of Σ , relate to the eigenvectors $|v_j\rangle$ as

$$|g_j^R\rangle = \Omega^{-2} |v_j\rangle \propto \sum_{n=1}^N e^{-2hn} \cos\left[\frac{(j-1)\pi}{N} \left(n - \frac{1}{2}\right)\right] |n\rangle, \\ |g_j^L\rangle = \Omega^2 |v_j\rangle \propto \sum_{n=1}^N e^{2hn} \cos\left[\frac{(j-1)\pi}{N} \left(n - \frac{1}{2}\right)\right] |n\rangle. \quad (14)$$

Since $\mathcal{S}$ has $\beta_1 = 1$ and also all other eigenvalues satisfy $|\beta_j| \leq 1$, the same holds for λ_j . It follows from Eq. (13) that projections onto $\{|g_m^R\rangle\}_{m \neq 1}$ vanish as $z \rightarrow \infty$. In this limit, any initial excitation evolves into an asymmetric distribution, $P_f = |g_1^R\rangle = \Omega^{-2} |v_1\rangle \propto (e^{-2h}, e^{-4h}, \dots, e^{-2hN})^T$, which manifests the non-Hermitian skin effect in the incoherent regime.

In Fig. 3(c), we depict the right eigenmode $|g_1^R\rangle$ of Σ for a lattice with $N = 30$, $l = 0.01$, and $h = 0.2$, in agreement with the expression of Eq. (14). Figure 3(d) depicts the dynamics of the average probability density $P(z)$ for the same lattice with initial excitation $\psi_n(0) = \delta_{n,30}$. The system evolves into the asymmetric edge-localized distribution $P_f \propto (e^{-2h}, e^{-4h}, \dots, e^{-2hN})^T$.

Antagonism of non-Hermitian skin effects. We now consider lattices with both defects and coupling asymmetry, leading to competition between the two types of non-Hermitian skin effects identified previously in the incoherent regime.

In Fig. 4(a), we plot the asymptotic probability density $P_f = |g_1^R\rangle$ for a lattice with $N = 30$ sites under dephasing ($l = 0.01$) with four identical defects at $q_i = \{3, 10, 18, 26\}$, corresponding to Figs. 3(a) and 3(b). In the symmetric case ($h = 0$), edge localization arises solely due to defects. With introduced coupling asymmetry $h = 0.3$, P_f shifts away from pure edge localization, forming a *jagged-shaped* pattern, broadly distributed with nodes at the defect positions. However, this asymmetry remains insufficient to shift localization entirely to the opposite edge. Further increasing asymmetry to $h = 0.8$ predominantly localizes the wave function on the left side, while the defect at site $q_1 = 3$ forces the existence of a node.

To quantify the interplay between the defect-induced and coupling-induced skin effects, we calculate the averaged asymptotic mean position $\langle n \rangle_f$ and position uncertainty Δn_f [82] of the wave function as functions of the coupling-asymmetry parameter h and the defect strength V [Figs. 4(b)

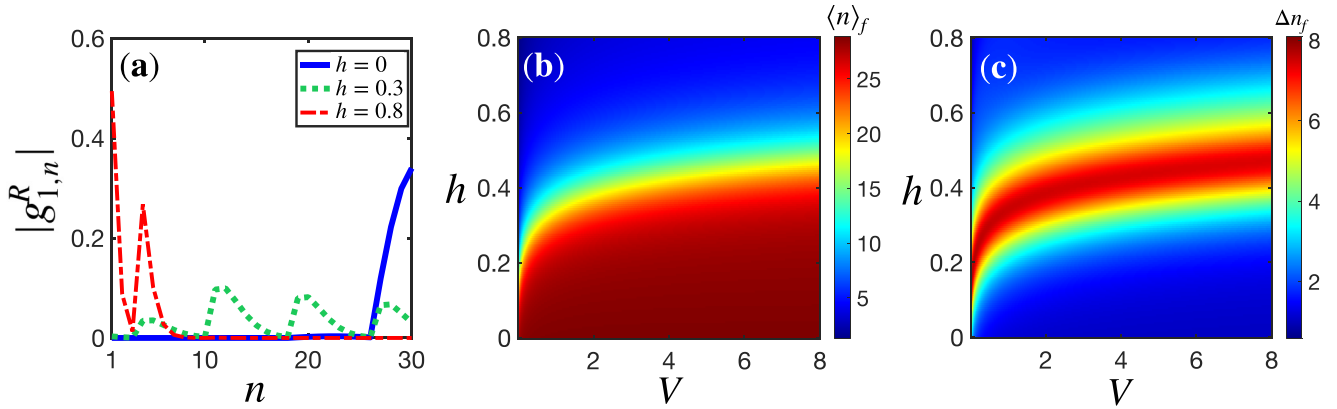


FIG. 4. Interplay between defect-induced and coupling-asymmetry-induced incoherent skin effects in a lattice of $N = 30$ sites under dephasing period $l = 0.01$, with four lossy defects placed at $q_i = \{3, 10, 18, 26\}$. (a) Amplitude $|g_{1,n}^R|$ of the asymptotically dominant eigenmode for $V = 0.5$ and $h = 0$ (blue), $h = 0.3$ (green), and $h = 0.8$ (red). (b) Asymptotic mean position $\langle n \rangle_f$. (c) Asymptotic position uncertainty Δn_f . Panels (b) and (c) are shown as functions of the coupling asymmetry h and the defect strength V .

and 4(c)], revealing the transition between the two skin effects. As shown in Fig. 4(b), for a fixed V there exists a range of h values where the defect-induced incoherent skin effect remains dominant, with $\langle n \rangle_f \sim N$. Beyond a certain threshold of h , the mean position shifts and the long-term probability density becomes more extended, since Δn_f is increasing. A further increase in h ultimately leads to the manifestation of the asymmetric-coupling induced skin effect, with $\langle n \rangle_f \rightarrow 1$ and decrease of Δn_f . Moreover, as evident in Figs. 4(a) and 4(b), the critical value of h needed for this transition, rises as the strength V of defects is increased. Although our quantitative analysis is based on the configuration of Fig. 4(a), these conclusions hold broadly; defect-driven dynamical skin effects can be precisely engineered in incoherent lattices, with preferential localization persisting robustly until antagonistic coupling asymmetry becomes sufficiently strong to overturn it.

Conclusions. In this work, we have identified a route to realizing a dynamical incoherent non-Hermitian skin effect

without relying on asymmetric couplings. Strategically engineered configurations of lossy defects, combined with fast dephasing, give rise to asymmetric propagation via directional jumps toward the lattice edge. We also examined the incoherent dynamics of a Hatano-Nelson lattice and investigated the interplay between these two distinct skin-effect mechanisms, revealing the emergence of jagged-shaped long-term probability distribution. Our results open avenues for the experimental observation of dynamical skin effects and for the theoretical exploration of dephasing as a tool for wave manipulation.

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Data availability. The data that support the findings of this article are not publicly available. The data are available from the authors upon reasonable request.

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