METAPLEX NETWORKS: INFLUENCE OF THE EXO-ENDO STRUCTURE OF COMPLEX SYSTEMS ON DIFFUSION

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Abstract. Complex networks represent the global behavior of complex systems in terms of interacting subcomponents. This article introduces metaplex networks, which include the internal structure, dynamics and function of these subcomponents, and analyzes their interplay with the network structure for the global dynamics of the system. We illustrate the use of this framework for diffusion and superdiffusion in metaplexes whose nodes are domains in $\mathbb{R}^n$. Long-range hopping leads to superdiffusive behaviour across the whole metaplex, and it survives independently of the internal structure. The global diffusion dynamics, however, strongly reflects the geometry of the nodes, the nature of the coupling, as well as the internal diffusion processes. We provide analytical and numerical results to shed light on this interaction of internal and external dynamics.

Key words. Complex networks, metaplex network, k-path Laplacian, anomalous diffusion, metapopulations

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1. Introduction. A complex system is characterized by the existence of many components which are interdependent on each other [20, 37, 42]. Each of these components is at the same time characterized by certain structure, dynamics and function [10], which influences the global behaviour of the system. The interconnection between these components represents the exo-skeleton of the complex systems, and it is well characterized by the use of complex networks. Nowadays, the use of networks to represent a large variety of these complex systems is very ubiquitous [20, 37, 42]. In such networks, which mathematically are equivalent to graphs $G = (V, E)$, the set of nodes $V$ represents the entities of the complex system and the set of edges $E$ captures the interactions between such entities. For instance, in a molecular graph [58] the nodes represent atoms and the edges account for the covalent bonds between pairs of atoms. At a larger scale, a macromolecule like a protein, can be represented by considering that the nodes represent not atoms but entire molecular building blocks, like amino acids [3]. Then, the edges represent the interactions (covalent and non-covalent) between such molecular motifs. Bigger inter-molecular systems can then be considered if the nodes are used to represent entire macromolecules–like genes, proteins or metabolites–and the edges represent the interactions, or even chemical transformation, among these macromolecules [11]. Abandoning the molecular world it is also possible to represent cellular systems in which the nodes account for specific cells, e.g., neurons, and the edges describe the physical connection between such cells, e.g. synaptic connections between neurons [12]. Such representations can go further in the size scale and represent complete species by nodes, like in food webs [49],

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or even human populations and cities like in epidemiologic metapopulation analysis [13, 38, 44] or in urban spatial systems [5, 33], respectively.

The success of network-theoretic approaches for studying complex systems relies on the fact that it goes beyond the study of individual entities to focus on the higher level of the interactions between such entities. The complexity of representing the connectivity of complex systems is manifested by the necessity of using a variety of mathematical objects for this purpose. Apart from the use of graphs (simple, directed and weighted) [20, 37, 42], other objects like hypergraphs [23, 36, 55], simplicial complexes [16, 24, 32] and multiplex networks [6, 9, 35] are frequently used. However, it is undeniable that a large proportion of the connectivity patterns of these interactions networks depend on the internal organization (endo-structure) of the entities represented by these nodes. For instance, the connectivity of atoms, i.e., their valence, depends on their internal electronic structure [15], and the interaction between proteins is mainly determined by the electrostatic, geometric and hydrophobic properties of individual molecules [34]. At larger scales we can find that the shape of a cell [14] or of a city [7] may determine the number of connections it can have with others and the way in which such interactions take place.

More importantly, the dynamical processes typically considered to take place among the nodes and edges of complex networks [10] can be dramatically influenced by the internal structure of the entities (nodes) of these systems. We can think about the diffusion of a chemical substance between the neurons in a neural network [45]. At the network-level such diffusion is studied by considering that the time consumed by the diffusive particle at each neuron is only proportional to the number of connections it has. In other words, we obviate the internal processing of such a chemical inside the neurons, which may influence the global diffusive process [51]. When analyzing a metapopulation model of epidemic spreading we typically reduce the internal structure of every patch to a well-mixed population [13, 38, 44]. Obviously, different neighborhoods in a city will have different structural organizations of their populations and will influence in different ways the global epidemic dynamics. Moreover, let us think about traveling between different cities using the road network that connect them. Obviously, it will take more time to traverse some cities than others due to their different internal patterns of streets and traffic. From a different perspective [25], networks with internal structure in the nodes can be relevant to represent diffusion processes in complex geometries, in the presence of obstacles and re-entrant corners. Similar to the metapopulation model, we subdivide the geometry into patches, each corresponding to the node of a graph. We thereby represent the diffusion process in terms of simpler processes inside each patch, in combination with the network diffusion between the patches.

In Figure 1 we illustrate schematically some examples of complex systems in which we Zoomed in the internal (endo) structure of the nodes capturing schematically their exo-endo structure. They include a molecular graph, a protein-protein interaction network, a neuronal network, a food web, a metapopulation system of population patches, and a road network between cities. The first, and up to now the only, attempt to combine the exo-endo structure of a system in a mathematical framework was published by Pogliani in 2003 [52]. It consists of a naive model in which the connectivity of a molecule is captured by a molecular graph, while the internal structure of the atoms is represented by “complete graphs” (see [52] for details). However, we are interested here in more general mathematical frameworks which can also allow to consider a large variety of physical, chemical, biological and social processes taking place on these networks in which the internal structure of the nodes may play a fun-
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They include diffusive processes, synchronization, epidemic spreading, quantum and chemical transport, reaction-diffusion, rumor spreading, etc. Then, the consideration of the endo-structure of the nodes in networks could be of vital importance in a variety of systems and processes taking place on them, ranging different size scales, topologies, nature of entities, and directionality of edges.

In this work we focus in formulating a new mathematical framework for representing and studying “metaplex networks”, which consider at the same time the exo-endo structure of complex systems. Using a metaplex formulation we consider diffusive dynamical processes taking place inside the nodes, represented as a continuous space, and hopping between the nodes in the discrete space of a network.

![Schematic illustration of networks at different size-scales with internal structure of nodes zoomed in.](image)

**Fig. 1.** Schematic illustration of networks at different size-scales with internal structure of nodes zoomed in. (a) Molecular graph in which every node represents an atom with internal structure and edges represent covalent bonds; (b) Protein-protein interaction network in which every node represents a protein and edges represent the non-covalent interaction between two proteins; (c) Neuronal network in which every node represents a neuron and links represent synaptic interaction between neurons; (d) Food web in which every node represents a species and links represent trophic interactions (who eats who) between species; (e) Metapopulation system in which nodes represent patches (neighbourhoods or entire cities) and links represent commuting patterns between patches; (f) Road system where node represent cities and links the roads connecting them.

In this article we introduce metaplex networks as a framework to discuss the exo-endo structure of complex systems.

To demonstrate the potential of our framework, we explore linear diffusion and superdiffusion in metaplex networks. We specifically consider networks of planar domains connected by localised sources and sinks, with diffusive dynamics inside each node and an external network dynamics described by a \( k \)-path Laplacian \([22, 19]\). Dynamical results show that internal superdiffusion accelerates the global diffusion.
in the network, depending on the geometry of the nodes. While superdiffusion in the
nodes cannot speed up regular diffusion in the network to superdiffusion, superdiffu-
sion in the network survives irrespective of the internal dynamics. The geometry of
the nodes plays a crucial role: In networks of small nodes, superdiffusion typically
slows down the dynamics, but it leads to considerable speed up in large nodes.

We explain these findings through a local description of the distribution of parti-
cles inside each node and propose an efficient delay description of the global dynamics.

In a combination of analysis and computations we describe the spectral gap, and
therefore equilibration times, depending on the geometry of the domains and their
coupling. Analytical formulas are obtained when the internal dynamics dominates,
and also the opposite limit of strong network interactions is discussed.

The results shed light on the rich and substantially different nature of the dy-
namics of metaplex networks and the interplay of their exo and endo structure. The
framework introduced in this article provides a language to include the structure,
dynamics and function of individual components into network theory.

Outline of this article: Section 2 provides a formal definition and examples of meta-
plex networks. Diffusion processes in metaplexes are the content of Section 3. They
are a particular example of a dynamical system on a metaplex, as defined in Sec-
tion 4. The generator of the diffusion is the relevant analogue of the graph Laplacian
in this context, and Section 4 also studies the long-time behavior of a diffusion in
terms of the spectrum of its generator. Section 5 obtains analytic results for the
time-dependent problem, before Section 6 explores the interaction of the internal and
external dynamics numerically. The conclusions are summarized in Section 7.

2. Definitions and motivation. One of the important characteristics of a com-
plex systems is given by the connectivity among its entities. Once such connectivity
is defined we can apply the appropriate mathematical methods to extract information
about the complex system. Depending on the level of coarse-graining used to detail
the entities we can have different types of mathematical representations of their con-
nectivity. The simplest of these approaches is to consider a set of binary relations
between the entities of the system in the form of a graph \( G = (V, E) \), where the set
of nodes represents the individuals and the set of edges represent their pairwise in-
teractions. Extra information about the nature of the interactions can be added in the
form of a weighted graph \( G = (V, E, F, \varrho) \) where \( \varrho : F \to E \) is a surjective mapping
assigning weights to edges [4, 43]. Departing from binary relation we can enter into
the world of hypergraphs $H = (V, E)$ where the set of hyperedges $E$ accounts for any $k$-ary relation between the agents in the system [23, 55, 59]. Such hypergraphs can also be directed and weighted [28, 36]. When the set of individual entities of the system is closed under the subset operation, the connectivity of the system is better represented by simplicial complexes $K = (V, \Sigma)$ where $\Sigma$ is a set of simplices, such that every face from $K$ is also in $K$, and the intersection of any two simplices is a face of both simplices [16, 17, 24, 32, 39]. Finally, there is the case where the set of nodes is split into several layers, each one with its own connectivity. In this case, the complex system is better represented by a multiplex [6, 9, 35, 53], mathematically defined as the triple $M = (G, \mathcal{A}, \mathcal{S})$, where $G = \{G_1, \cdots, G_h\}$ is a set of simple graphs, $\mathcal{A}$ is a set of node identities with an equivalence relation, and $\mathcal{S}$ is a set of connections to a node in a layer to its equivalent in a different layer (see [21] for details).

Even with this arsenal of mathematical representations — graphs, hypergraphs, simplicial complexes and multiplexes — there are important characteristics of complex systems which are not covered by them. As we have mentioned in the Introduction one of these characteristic features is the fact that two kind of dynamics can be taken place at the same time in the system. One, is a dynamical process taking place inside the nodes, which may be controlled by continuous equations and that can be dependent on the internal structure of the nodes. The other is the discrete dynamics taking place between the nodes of the system, which is mainly controlled by the connectivity of the system. To account for these complex scenarios we propose here the following representation.

**Definition 2.1.** A metaplex network is a 4-tuple $\Upsilon = (V, E, I, \omega)$, where $(V, E)$ is a graph, $\omega = \{\Omega_j\}_{j=1}^k$ is a set of locally compact metric spaces $\Omega_j$ with Borel measures $\mu_j$, and $I : V \to \omega$.

In relevant examples $\omega$ is either a set of open domains $\Omega_j \subset \mathbb{R}^n$, endowed with the Lebesgue measure, or a set of graphs $(V_j, E_j)$ with the counting measures.

For instance, let us consider a metaplex in which $(V, E)$ is the linear graph, i.e. a path graph, on 5 nodes and in which the shape of each node is given by the unit disk $B(0, 1) \subset \mathbb{R}^2$, so that $\omega = \{B(0, 1)\}$ and $I$ is constant. Then, we obtain the metaplex illustrated in Figure 2. In addition, by Definition 4.1 we introduce the notion of a dynamical system on a metaplex. A key example here is the local diffusion processes inside each node (Section 3), coupled by localized sinks and sources according to the edges $E$. The resulting density distribution of the diffusing particles is illustrated at the nodes of the metaplex in Figure 2.

When $\omega = \{G_0\}$ is a given graph, we recover multiplex networks [30, 54] as a special case, in the case of a uniform diffusion dynamics between the nodes, see Subsection 3.2 and Subsection 6.4. However, the natural occurring examples and typical dynamics for a metaplex network are the opposite of the homogeneous couplings studied for multiplexes.

### 3. Diffusion in metaplexes.
Metaplex networks give us a context to study the coupled dynamics between the network $(V, E)$ and a dynamical system in each node $\Omega_j$. Motivated by the above-mentioned applications, this article considers a network of bounded Euclidean domains $\Omega_j \subset \mathbb{R}^n$ and the interaction of anomalous, linear diffusion processes inside and between the nodes. For normal diffusion, corresponding to standard Brownian motion, the mean squared displacement $\langle x^2 \rangle$ of a particle is proportional to time. Anomalous diffusion is characterized by a power-law shape of the fundamental solution, i.e. the spatial particle distribution for a Dirac initial
condition, with power $1 < s < 2$ and by a mean squared displacement, $\langle x^2 \rangle \sim t^{2/s}$ of particles which for long times grows faster than for Brownian motion. The exponent $s = 1$ corresponds to ballistic transport, while $s = 2$ is the case of normal diffusion.

The prototypical diffusion process in a network of point nodes is a random walk, generated by the graph Laplacian. Superdiffusive processes arise from long range hopping beyond nearest neighbors, where the probability of jumping algebraically decays with the increase of the distance between nodes. Simple examples of superdiffusion processes are obtained from the $k$-path Laplacian $\Delta_k$: For $f \in C(V)$,

$$\Delta_k f(v) = \sum_{w \in V, \text{dist}(v, w) = k} (f(v) - f(w)).$$

In the following we briefly discuss two transformed $k$-path Laplacian that are later considered in Section 6.

For $s > 1$, the Mellin-transformed path Laplacian is given by

$$(3.1) \Delta_M f = \sum_{k=1}^{\infty} k^{-s} \Delta_k f.$$

In the one-dimensional linear network $\mathbb{Z}$, superdiffusion emerges for $s \in (1, 3)$ [22]. For $s > 3$ normal diffusion is present in the network for long times.

Similarly, normal diffusion persists (see experiments in Section 6) for the Laplace-transformed $k$-path Laplacian

$$(3.2) \Delta_e f = \sum_{k=1}^{\infty} e^{-ks} \Delta_k f,$$

where the dynamics in the network is dominated by exponentially short ranged hopping.

In the continuum, e.g., when $\Omega \subset \mathbb{R}^n$, linear superdiffusion is similarly associated to a random walk involving long runs [26], or a Lévy process. The fractional Laplacian on $\mathbb{R}^n$ is defined in terms of the Fourier transform as $(-\Delta)^s u = F^{-1}(|\xi|^{2s} F u)$, $s \in (0, 1]$. It coincides with the ordinary Laplacian for $s = 1$. In a domain $\Omega$, the appropriate definition is in terms of the bilinear form

$$a(u, v) = \frac{c_{n,s}}{2} \iint_{(\Omega \times \mathbb{R}^n) \cup (\mathbb{R}^n \times \Omega)} \frac{(u(x) - u(y))(v(x) - v(y))}{|x - y|^{n+2s}} dy \, dx$$

on $H^s(\Omega)$, where $c_{n,s} = \frac{2^{1-s} \Gamma(s) \Gamma(s + n/2)}{\pi \Gamma(1 + s)}$. For $s = 1$, it is interpreted as a Laplacian with Neumann boundary conditions [18].

### 3.1. Sinks and sources

As depicted in Figure 2, the coupling of endo (internal node) and exo (hopping between nodes) diffusion in a metaplex may involve nontrivial geometries, depending on the physical origin. In the simplest case, sinks and sources in the interior of the nodes correspond to a coupled system of diffusion equations

$$(3.3) \partial_t u_j(t, x) = \text{div} J_j(u_j(t, x)) - \sum_{i \in \{v_j, v_i\} \in E} \alpha_{ij}(x) u_j(t, x)$$

$$+ \sum_{i \in \{v_i, v_j\} \in E} \alpha_{ji}(\psi_{ji}^{-1}(x)) \det(\nabla \psi_{ji}^{-1}) u_i(t, \psi_{ji}^{-1}(x)).$$
Here $u_j$ is the density of particles in the node $v_j \in V$, and $\text{div} J_j$ is the generator of the diffusion process in $\Omega$, for example $\text{div} J_j = -(-\Delta)^s$. The edges $(v_i, v_j) \in E$ are realized by a map $\psi_{ji} : \Omega_j \to \Omega_i$ which specifies the jumps between domains, and the coefficients $\alpha_{ji}(x)$ are local transition probabilities: Particles jump from $\psi_{ji}(x)$ to $x$ and from $x$ to $\psi_{ji}(x)$ with amplitude $\alpha_{ji}(x)$. It is easy to see that the total number of particles $\sum_j \int_{\Omega_j} u_j \, dx$ does not depend on $t$.

Physically, the system (3.3) arises for nodes which correspond to spatially distant domains.

A physical situation serving as example here is the diffusion of electrical and chemical signals through the neurons in the brain. While the internal diffusion inside the neuron and its transmission between nearest neighbours neurons are well-known processes, the long-range jumps can represent higher-order interactions taking place through glial cells such as astrocytes, which are found to play a fundamental role in the modulation of the neuronal electrical activity [50, 2, 47].

3.2. Diffusion with internal states. In the case $\Omega = \Omega_j$ for every $j$ and $\psi_{ji} = \text{Id}$, the density $u$ in Subsection 3.1 may be interpreted as a vector valued function $u = (u_1, \ldots, u_N) : [0, \infty) \times \Omega \to \mathbb{R}^N$. In this case, the network encodes the dynamics of the “internal state” of the particle described by a vector in $\mathbb{R}^N$. In biology, such processes are of interest to describe the diffusion of complex organisms. If $\alpha_{ij}$ is independent of $x$ and $\psi_{ji}$ is the identity, then we recover a multiplex network as a special case of the metaplex dynamics.

3.3. Interface problems. In complex systems such as a road system or transport of chemicals between cells, the coupling between the nodes occurs through the boundary. Every edge $(v_i, v_j) \in E$ is physically realized by open entrances and exits $\Gamma_{ij} \subset \partial \Omega_i$ and $\Gamma_{ji} \subset \partial \Omega_j$, together with a homeomorphism $\phi_{ij} : \Gamma_{ij} \to \Gamma_{ji}$ identifying points between them. We define $\Gamma_{ji} = \partial \Omega_i \setminus \bigcup_j \Gamma_{ij}$. If there is an edge between $\Omega_i$ and $\Omega_j$, particles leave $\Omega_i$ through $\Gamma_{ij}$ and arrive at $\Gamma_{ji}$ in $\Omega_j$.

The resulting system of diffusion equations is coupled through the boundary conditions, with a Kirchhoff’s law: For $x \in \bigcup_i \Gamma_{ji}$,

$$u_j(t, x) = \sum_{i : x \in \Gamma_{ji}} \alpha_{ji} \det(\nabla_{\partial \Omega} \phi_{ji}) u_i(t, \phi_{ji}(x)),$$

$$J(u_j(t, x)) \cdot \nu_j(x) = - \sum_{i : x \in \Gamma_{ji}} \alpha_{ji} \det(\nabla_{\partial \Omega} \phi_{ji}) J(u_i(t, \phi_{ji}(x))) \cdot \nu_i(\phi_{ji}(x)),$$

where $\nu_j$ is the exterior unit normal vector. To preserve the number of particles, we require that the transition probabilities $\sum_i \alpha_{ji} = 1$ for every $j$.

4. Operators and spectra for metaplexes. A fundamental tool for the analysis of a network are the adjacency matrix and related graph Laplacians. These matrices do not only describe the transition probabilities of a random walk, and therefore dynamical properties of the complex system like the rate of convergence to equilibrium. Their eigenvalues and eigenvectors also encode topological and geometric information such as connectivity and measures of centrality.

The corresponding tool to describe the dynamics of a metaplex network is an operator matrix. The diffusion processes in Section 3 take the abstract form

$$\frac{du}{dt} = Du,$$
where $\mathcal{D}$ is an $N \times N$ block operator matrix of unbounded operators on the Hilbert space $\bigoplus_{j=1}^{N} \mathcal{L}^2(\Omega_j)$, formally

$$
\mathcal{D} = \begin{pmatrix}
\text{div}J_1 & \alpha_{12}T_{12} & \cdots & \alpha_{1N}T_{1N} \\
\alpha_{21}T_{21} & \text{div}J_2 & \cdots & \alpha_{2N}T_{2N} \\
& \ddots & \ddots & \ddots \\
& & \alpha_{N1}T_{N1} & \cdots & \alpha_{NN}T_{NN}
\end{pmatrix}.
$$

Here the $T_{ij}$ are transition operators between $\Omega_i$ and $\Omega_j$, as given by the sources and sinks or boundary conditions, and $\alpha_{ij}$ relates to the transition probabilities.

For a general metaplex network we define:

**Definition 4.1.** A dynamical system on a metaplex network $\Upsilon = (V,E,T,\omega = \{\Omega_i\})$ is a tuple $(\mathcal{H}, \mathcal{T})$. Here $\mathcal{H} = \{H_{V}\}_{v \in V}$ is a family of operators on $\mathcal{L}^2(\Omega_{V}(v),\mu_{V}(v))$ such that the initial value problem $\partial_t u_t = H_v(u_t)$, $u_t|_{t=0} = u_0$, is well-posed, and $\mathcal{T} = \{T_{vw}\}_{(v,w) \in E}$ is a family of bounded operators $T_{vw} : \mathcal{L}^2(\Omega_{V}(v),\mu_{V}(v)) \to \mathcal{L}^2(\Omega_{V}(w),\mu_{V}(w))$.

Under mild regularity assumptions on the measure $\mu_j$, given a general metaplex $\Upsilon$ the metric spaces $\Omega_j$ admit $\mu_j$-symmetric diffusion processes on $\mathcal{L}^2(\Omega_j)$ [56]. Coupling them according to $E$, one obtains a dynamical system on $\Upsilon$ analogous to the diffusion generated by $\mathcal{D}$.

The long-time behavior of the linear diffusion equation (4.1) is determined by the spectral properties of $\mathcal{D}$ [57]. In line with Definition 4.1 we write $\mathcal{D} = H + T$, with

$$
H = \begin{pmatrix}
\text{div}J_1 & 0 & 0 & \cdots & 0 \\
0 & \text{div}J_2 & 0 & \cdots & 0 \\
& \ddots & \ddots & \ddots & \ddots \\
0 & 0 & \cdots & \cdots & \text{div}J_N
\end{pmatrix},
$$

and

$$
T = \begin{pmatrix}
-\sum_{j} \alpha_{j1}T_{11} & \alpha_{12}T_{12} & \cdots & \alpha_{1N}T_{1N} \\
\alpha_{21}T_{21} & -\sum_{j} \alpha_{j2}T_{22} & \cdots & \alpha_{2N}T_{2N} \\
& \ddots & \ddots & \ddots \\
& & \alpha_{N1}T_{N1} & \cdots & \alpha_{NN}T_{NN}
\end{pmatrix}.
$$

While the spectrum of $H$ is determined by the internal structure of the nodes, the spectrum of $T$ combines the details of the location and strength of sinks, resp. sources, with the external network structure.

### 4.1. Variation of eigenvalues with fractional exponent and spatial scale.

Note that $H = \bigoplus_{j=1}^{N} H_j = \bigoplus_{j=1}^{N} \text{div}J_j$ is a block diagonal operator matrix acting on $\mathcal{L}^2(\Omega_1) \times \cdots \times \mathcal{L}^2(\Omega_N)$. A basis of eigenfunctions of $H$ is constructed from bases $\{u_{j,k}\}_k$ of eigenfunctions of $\text{div}J_j$ in $\Omega_j$: It is given by $\{u_{j,k}e_j\}_{j,k}$. Here $e_j$ denotes the $j$-th standard unit vector in $\mathbb{R}^N$.

If $H_j = -(-\Delta)^{s_{nod}}$ is the fractional Laplacian in $\Omega_j$ with Neumann boundary conditions, the eigenvalues of $H_j$ are homogeneous functions of the spatial scale. More
precisely, the bilinear form of $H_j$ is homogeneous under scaling $\Omega_j \to \Lambda \Omega_j$, $\Lambda > 0$. From their characterization in terms of a Rayleigh quotient, the eigenvalues in $\Lambda \Omega_j$ are given by $\{\Lambda^{-2s_{\text{nod}}(\mu_j,k)}\}_{k=1}^\infty$, if $\{\mu_{j,k}\}_{k=1}^\infty$ are the eigenvalues of $H_j$ in $\Omega$. Only the lowest eigenvalue $\mu_{j,1} = 0$ is fixed under rescaling.

We see that for small domains, $\Lambda \to 0$, the spectral gap $\mu_{j,2} - \mu_{j,1}$ in the node $\Omega_j$ increases with $s_{\text{nod}}$, and therefore Brownian motion in the nodes gives the fastest convergence to equilibrium. For large domains, $\Lambda \to \infty$, the spectral gap $\mu_{j,2} - \mu_{j,1}$ in the nodes $\Omega_j$ decreases with $s$, and therefore the long jumps of the fractional diffusion lead to faster equilibration than Brownian motion inside each node.

4.2. Multiplex-like metaplex networks. The analogue of a multiplex network in our setting corresponds to $\Omega = \Omega_j$ for all $j$ and $T_{ij} = \text{Id}$ [30, 54]. In this case $T$ recovers the dynamics in the network with structureless nodes. Let $\nu \in \mathbb{R}^N \subset \mathbb{R}^N \otimes L^2(\Omega)$ be a constant eigenfunction of $T$, $Tv = \lambda \nu$, and assume that constant functions are in the kernel of $H$. Then $\nu$ is in the kernel of $H$, and therefore it is also an eigenfunction of $H + T$: $(H + T)v = \lambda \nu$. Therefore the spectrum of the network Laplacian with point nodes is contained in the spectrum of the full operator $H + T$ for the network with internal structure. A refined analysis is given below.

4.3. Weakly and strongly interacting limits. The spectral properties of $\mathcal{D}_\varepsilon = H + \varepsilon T$ are most easily understood in the limit $\varepsilon \to 0$ of weak network interactions between the nodes, respectively the strong network interactions for $\varepsilon \to \infty$. In the former case, a particle is trapped for a long time inside the domain in which it was initially placed. The time scale of the slow global equilibration is determined by $T$. In the latter case, the transient dynamics is governed by the dynamics of the network and geometry of the sinks and sources, i.e. $T$, but the dynamics $H$ inside the nodes determines the long-time approach to equilibrium.

We first consider $\Omega = \Omega_j$ for all $j$ and $T_{ij} = \text{Id}$. In this case $T = \tau \otimes \text{Id}$ and the eigenspaces of $T$ are infinite dimensional. If $v_{k,\text{netw}} \in \mathbb{R}^N$ is a normalized eigenvector of the network matrix $\tau$, $\tau v_{k,\text{netw}} = \lambda_k v_{k,\text{netw}}$, then $\lambda_k^\text{netw} \otimes L^2(\Omega)$ is an infinite dimensional space of eigenfunctions for $T$ to the same eigenvalue $\lambda_k$.

4.3.1. Weak network interactions. For $\varepsilon > 0$ small, we first consider $\Omega = \Omega_j$ for all $j$ and $T_{ij} = \text{Id}$. In this case the eigenvalues of $\mathcal{D}_\varepsilon$ are asymptotically given by

$$\mu_{j,1} - \varepsilon \alpha_{jj},$$

provided $\mu_{j,1}$ is a simple eigenvalue. Multiple eigenvalues arise, in particular, when several of the $H_j$ coincide. In this case the eigenvalues are determined by the restriction $T_r$ of $T$ to the subnetwork of those nodes where the internal diffusion has the eigenvalue $\mu_{j,1}$. If the eigenvalues of $T_r$ are $\{\lambda_k^r\}$, then the eigenvalues of $H + \varepsilon T$ are asymptotically given by $\mu_{j,1} + \varepsilon \lambda_k^r$. For the stationary state the eigenvalue $\mu_{j,1} = 0$, $j \in \{1, \ldots, N\}$, splits into exact eigenvalues $\varepsilon \lambda_k$ of $\varepsilon T$.

For general interactions $T$ the stationary states corresponding to $\mu_{j,1} = 0$, $j \in \{1, \ldots, N\}$, again split into eigenvalues $\varepsilon \lambda_k + o(\varepsilon)$, where $\lambda_k$ are the eigenvalues of the matrix with entries $\tau_{ij} = (1 - 2\delta_{ij}) \int_{\Omega_j} \alpha_{ij} T_{ij} \, dx$. Higher eigenvalues depend on the location of the sources and sinks. The spectral gap of the network is $\varepsilon \lambda_2$, and thus independent of the diffusion process $H$.

Local equilibration within the nodes, however, happens on faster time scales, with little effect of the coupling. For small $\varepsilon$, the gap between the higher bands $\mu_{j,k} + \varepsilon \lambda_k^r$ to the equilibrium is determined by the diffusion $H$, up to terms of order $\varepsilon$. 
4.3.2. Strong network interactions. A second analytically accessible regime considers \( D_{\varepsilon}^{-1} = \varepsilon^{-1}(\varepsilon H + T) \) for small \( \varepsilon > 0 \). As \( H \) is nonnegative, the eigenvalues of \( \varepsilon H + T \) are increasing functions of \( \varepsilon \).

We first consider \( \Omega = \Omega_i \) for all \( i \) and \( T_{ij} = \text{Id} \). In the notation from above, degenerate perturbation theory implies that for small \( \varepsilon \) the eigenvalues of \( \varepsilon H + T \) are asymptotically given by

\[
\lambda_{k,l}^{1,...,l_j}(\varepsilon) = \lambda_k + \varepsilon \sum_{j=1}^{N} (v_{k,j}^{\text{netw}})^2 \mu_{j,l_j}.
\]

This generalizes formula (12) of [30] for multiplex networks for \( k = 1, \lambda_1 = 0 \). We conclude that there is a branch of eigenvalues of \( D_{\varepsilon}^{-1} \) which stays finite as \( \varepsilon \to 0 \) and determines the spectral gap of \( D_{\varepsilon}^{-1} \). The branch is given by \( \sum_{j=1}^{N} (v_{k,j}^{\text{netw}})^2 \mu_{j,l_j} \), hence greater than \( \min_j \{ \mu_{j,2} \} \). It is determined by the internal diffusion, averaged according to the network interactions. Diffusion within the nodes of the network will therefore be faster than in the slowest node of the network. This generalizes formula (15) of [30].

For general \( T_{ij} \) the spectrum of the network interaction operator \( T \) will be continuous, and the perturbation theoretical arguments require more care.

4.4. Numerical results. We illustrate the spectral gap of \( D_{\varepsilon} \) for a linear network of 11 unit disks \( \Omega = \Omega_i = B(0, 1) \subset \mathbb{R}^2 \). The lowest 10 nonzero eigenvalues are considered as a function of \( \varepsilon \), the coupling operator \( T \) and the diffusion \( H \). We choose the generator \( H_j \) of the diffusion process inside \( \Omega_j \) as a fractional Laplacian \( -(-\Delta)^{s_{\text{nod}}} \) with Lévy exponent \( s_{\text{nod}} \) and approximate it by finite elements on quasi-uniform spatial meshes. See Figure 6 below for a plot of the mesh and [27] for the approximation of the fractional Laplacian.

The network coupling \( T_{ij} \) is taken to be a \( k \)-path Laplacian (3.2) with hopping between \( \Omega_i \) and \( \Omega_j \) proportional to an exponential \( 2^{-s_{\text{net}}|i-j|} \).

As \( H \) is unbounded the eigenvalues of its discretization extend over several orders of magnitude, while the spectral gap is tiny. To resolve it, the discretization of the mesh \( \mathcal{h} \) needs to be small compared to the strength of the coupling, and standard Matlab routines do not identify the bounded branches of eigenvalues obtained in Subsection 4.3.2 for large \( \varepsilon \). Discretization errors significantly increase as \( s \to 0 \) because the smoothness of the solutions in \( \Omega \) decreases.

Figure 3 indicates the complications inherent in approximating the spectrum of a large system of differential operator. The lowest 10 nonzero eigenvalues of \( D_{\varepsilon} \) are depicted for the exponential coupling as a function of the coupling strength \( \varepsilon \in [2^{-10}, 2^{10}] \) for meshes of 347 and 1325 nodes, \( s_{\text{net}} = 0.8 \), \( s_{\text{nod}} = 0.4 \). We consider a prototypical metaplex coupling as depicted in Figure 2 for \( \alpha_{i,j} = 10 \). While results agree for large coupling strengths, the spectral gap is significantly smaller for the finer mesh at small coupling. Nevertheless, the qualitative behavior from Subsection 4.3 is recovered: the gap increases linearly for small \( \varepsilon \), and for large \( \varepsilon \) it converges to the lowest eigenvalue of \( H \).

Figure 4 connects the theory of Subsection 4.3 with the numerical results from Section 6 and evidences the effect of the geometry of the nodes on the dynamics of the network. In the case of the small nodes (Figure 4 left) when the coupling is weak, the dynamics of the network is dominated by the diffusion process inside the
nodes and we have almost no hopping between domains. As we increase the coupling strength, for $s_{nod} = 0.8$ the spectral gap is bigger that for $s_{nod} = 0.2$ in agreement with Subsection 4.1. As was shown in [54] for the case of multiplex networks, the first non-zero eigenvalue of $D_\varepsilon$ is related to the equilibration time in the whole networks, i.e., $t = \lambda_2^{-1}$. From Figure 4 (left) the equilibration time for the case $s_{nod} = 0.2$ is much longer than for the normal diffusion case inside the nodes. This is in agreement with Figure 9a, where the superdiffusion inside the nodes slows down the equilibration in the network.

In the case of big nodes (Figure 4 right) that the spectral gap decreases as we increase the parameter $s_{nod}$. This means that, opposite to the small node case, a metaplex with superdiffusion inside the nodes ($s_{nod} = 0.2$) reaches the equilibrium faster than for internal Brownian motion. This was also observed in Figure 9b.

5. Analysis of diffusion in metaplexes. In this section we indicate how PDE techniques allow an analysis of the time-dependent diffusion introduced in Subsection 3.1 in a metaplex network of bounded domains $\omega = \{\Omega_j \subset \mathbb{R}^n\}_{j=1}^k$, as illustrated in Figure 2. We compare the interaction of diffusion, respectively superdiffusion, in the nodes with either short or long range diffusion in the external network. While for general complex networks the connectivity and large scale geometry of the network will crucially influence the dynamics, we here focus on simple linear networks $V = \mathbb{Z}$
and short times.

More precisely, we assume that the diffusion process in each node $\Omega_j$ is governed by either a fractional Laplacian $H_j = (-\Delta)^{s_{\text{nod}}}$ or a self-adjoint elliptic differential operator of second order like the Laplacian, $H_j u = \text{div}(a\nabla u)$, $a \in C^\infty(\Omega_j)$, with Neumann boundary conditions. The network coupling $T_{ij}$ consists of disjoint sinks and sources, and satisfies the estimate (5.1), respectively (5.2), for short times. For long times, it satisfies a (fractional) Fokker-Planck equation [8]. It satisfies a (fractional) Fokker-Planck equation and satisfies the estimate (5.1), respectively (5.2), for short times. For long times, $\tau_{\text{source, sink}}$ decays exponentially fast in time.

The situation may be considered as a toy example to describe neural activity in the brain, where the coupling between different domains is often disjoint, so that the network decomposes into disjoint nodes in the absence of internal diffusion. Inside each node $\Omega_j$ the solution $e^{tH_j} u_{j,0}$ to the heat equation for $H_j$ with initial condition $u(t=0) = u_{j,0}$ is an integral operator $(e^{tH_j} u_0)(t, x) = \int_{\Omega_j} K_{H_j}(t, x, y) u_0(y) \, dy$. The integral kernel $K_{H_j}$ describes the evolution of a Dirac point mass in $y$ at time $t = 0$. For ordinary diffusion away from the boundary $\partial\Omega_j$ it satisfies the Gaussian estimate

$$|K_{H_j}(t, x, y)| \leq C t^{-n/2} e^{-C \frac{|x-y|^2}{t}}$$

for some $C > 0$, analogous to the explicit solution formula for the heat equation on $\mathbb{R}^n$ [48]. Similar bounds are known for ordinary diffusion on metric measure spaces $\Omega_j$ [31]. Fractional diffusion, on the other hand, exhibits slow algebraic decay in the form of a Poisson estimate

$$|K_{H_j}(t, x, y)| \leq C t \left( |x-y| + t^{1/(2s_{\text{nod}})} \right)^{-n-2s_{\text{nod}}}$$

away from $\partial\Omega_j$ [29]. In a convex domain the estimate is sharp for short times, and it allows us to estimate the diffusion between nodes across the network.

Consider the linear network in Figure 2, with an initially uniform density localized in $\Omega_1$. From Equation (3.3) we calculate the change of the total density in node $j$:

$$\partial_t \left| \int_{\Omega_j} u_j(t, x) \, dx \right| = -\delta_{j1} |\Omega_j| \sum_k \int_{\Omega_j} \alpha_{ij}(x) dx + \frac{1}{|\Omega_j|} \int_{\Omega_j} \alpha_{j1}(\psi_{j1}^{-1}(x)) \text{det}(\nabla \psi_{j1}^{-1}) \, dx .$$

So, initially particles hop from node 1 to node $j$ according to the transition probabilities $\alpha_{ij}$ of the network, from $x$ to $\psi_{j1}(x)$. From Duhamel’s formula for the solution of the inhomogeneous diffusion equation, they then evolve inside $\Omega_j$ according to $K_{H_j}$ before jumping back to node 1, or further to a different node $k$. Both processes only enter into the next, quadratic term of the Taylor expansion in $t$. These formal arguments are made rigorous in terms of an asymptotic expansion for $t \to 0$ of the heat kernel $K_D$ for the network of interacting domains, see e.g. [41].

Compared to nodes without internal structure, hopping to node $k$ is reduced by the rate $K_{H_j}(\tau, \psi_{j1}(x), y)$ of diffusing from $\psi_{j1}(x)$ to a point $y$ in the region of the sink to node $k$ within time $\tau$. The internal diffusion thus always slows down the network dynamics if sources and sinks are disjoint.

The exit time $\tau^j_{\text{source, sink}}$ taken to get from source to sink is well studied both for normal and fractional diffusion [8]. It satisfies a (fractional) Fokker-Planck equation and satisfies the estimate (5.1), respectively (5.2), for short times. For long times, $\tau^j_{\text{source, sink}}$ decays exponentially fast in time.
For short times the dependence on the internal diffusion $H_j$, the geometry of $\Omega_j$ and the location of sinks and sources are well described by (5.1) and Equation (5.2). For sources and sinks which are far apart, in the case of normal diffusion Equation (5.1) shows a faster than exponential suppression of the transition rate due to the internal structure. Fractional diffusion suppresses the transition rate algebraically in the distance between sink and source, according to Equation (5.2). The smaller $s_{nod}$, the faster the equilibration and the smaller the suppression in $\Omega_j$.

Similarly if $T_{ij}$ is nonzero only for $|i - j| \leq 1$, hopping to next $j$-th nearest neighbors is suppressed $j$ times by the internal diffusion. Starting from an initial density localized in node 1, the total density in node $j$ will be exponentially small, $\int_{\Omega_j} u_j(t, x) \, dx \leq C e^{-C|j-1|}$ for some $C = C(t) > 0$ depending on the endo structure.

A more careful analysis shows that superdiffusion cannot arise from short-range hopping in the network:

**Theorem 5.1.** Let $V = \mathbb{Z}$ and $T_{ij}$ given by the $k$-path Laplacian (3.2). Then there exists $C > 0$ such that $\int_{\Omega_j} u_j(t, x) \, dx \leq C e^{-C|j-1|}$ for $j \in \mathbb{Z}$. In particular, superdiffusion is not possible for the considered internal diffusions $\{H_k\}_k$.

This is confirmed by the numerical experiments in Section 6. In the case of the Mellin-transformed $k$-Laplacian (3.1), the numerical results indicate that superdiffusion in the network may persist even for nodes with distant sinks and sources. While in general the range of Mellin exponents $s_{nod}$ which allow superdiffusion may shrink, for certain experiments in the linear network $V = \mathbb{Z}$ we numerically recover superdiffusion for $s_{nod} \in (1, 3)$, as in absence of internal structure [22].

6. **Numerical analysis of diffusion in metaplexes.** This section further illustrates the influence of the internal structure of the nodes on the global network dynamics, using numerical experiments. Compared to the uniform coupling in a multiplex network, discussed below in Subsection 6.4, the localized coupling $T_{ij}$ in a given area (Figure 5), shows the rich dependence on the internal structure in a metaplex network, while still being analytically accessible in certain limits.

6.1. **Set up of numerical experiments.** We consider a linear network of 51 identical circular domains $\Omega = \Omega_i \subset \mathbb{R}^2$. Starting from a uniform distribution in node 1, we study the evolution of the density in the nodes depending on the network and internal diffusion, the size of the nodes and the strength and nature of the coupling between the nodes. Equivalently, one may interpret the experiments as the expected value for the dynamics starting from a localized density in a (uniformly distributed) random point in $\Omega$.

Two different sizes of nodes are considered, $\Omega_a = B(0, 1)$, $\Omega_b = B(0, 100)$. The diffusion equation (3.3) inside each node with Levy exponent $s_{nod}$ is approximated by finite elements in space on a quasi-uniform spatial mesh with 347 degrees of freedom. See Figure 6 for a plot of the mesh and [27] for the approximation of the fractional Laplacian. For the time discretization we use a backward Euler method in time with a sufficiently small, fixed time step $dt = 0.01$.

Unless stated otherwise, the network coupling between nodes $(i, j)$ is taken to be of short range according to (3.2), proportional to $2^{-s_{net}|i-j|}$ for different exponents $s_{net}$.

6.2. **Experiment 1: Central coupling region.** For different values of the network exponent $s_{net} = 0.4, 0.8$, we compare close to normal diffusion (Lévy exponent $s_{nod} = 0.8$) to superdiffusion ($s_{nod} = 0.2$) inside a network of small nodes $\Omega_a$. We
consider a coupling through a central region, as depicted in Figure 5a. The coupling strength is fixed to be $\alpha = 10$.

Figure 7 shows the time at which the density in node 1 reaches the equilibrium given by $\left| \int_{\Omega_1} u(x, t) dx - \frac{1}{N} \int_{\Omega} u_0(x) dx \right|$, where $u(x, t)$ is the density in the node at a given time $t$ and $u_0(x)$ is the initial density in the network. The figure illustrates the strong effect of the dynamics inside the nodes on the diffusion process, even though the networks dynamics dominates. We see that superdiffusion inside each node slows down the diffusion in the network: Due to the nature of the Lévy process, particles quickly diffuse far away from the sink. As they take time to return and hop to the next node, diffusion between nodes in the whole network is slowed down. In Subsection 6.3 we discuss how the size of the nodes affects this behavior.

The spatial localization decreases the total strength of the coupling, resulting in a slower equilibration of the densities in the node compared to a uniform coupling in Figure 15a below. Changing the strength of a sufficiently strong localized coupling does not affect this behavior much, as the equilibration time saturates: independent of the coupling strength, particles which are located away from the sink cannot hop to a neighboring node.

In Figure 8 we show the evolution in time of the total density $\int_{\Omega_i} u(x, t) dx$ in each node $\Omega_i$. While superdiffusion inside the nodes has only limited effect on the equilibration of the densities in the network, it speeds up hopping to distant nodes.
6.3. Experiment 2: Distant coupling regions. To study the effect of the node geometry on the global dynamical process, we consider a prototypical metaplex coupling as depicted in Figure 2: For the odd nodes the coupling region is located as in Figure 5b, while for even nodes the coupling region is on the opposite side. The coupling areas in both domains are equal, therefore, in the case of $\Omega_b$, the areas are more localized and distant. Approximately normal diffusion (Lévy exponent $s_{nod} =
0.8) and superdiffusion ($s_{nod} = 0.2$) are considered both in small nodes $\Omega_s$ and in large nodes $\Omega_b$.

For the network dynamics, we consider the short range coupling from Experiment 1, which is proportional to $2^{-s_{net}|i-j|}$ with $s_{net} = 0.4, 0.8$, and the coupling strength is fixed to be $\alpha = 10$. In Subsection 6.3.1 we compare the resulting dynamics to a long-range coupling proportional to $|i-j|^{-s_{net}}$ with the Mellin-transformed $k$-path Laplacian (3.1).

Fig. 9. Density equilibration in node 1 for disjoint sinks and sources (a) $\Omega_s$ (b) $\Omega_b$.

Fig. 10. Density distribution for small nodes $\Omega_s$, $s_{net} = 0.8$ (top panel), resp. 0.4 (bottom panel).
Figure 9a shows the deviation of the density in node 1 from equilibrium, corresponding to Figure 7 in Experiment 1. Similar to Experiment 1, superdiffusion inside the small nodes $\Omega_s$ slows down equilibration. However, the evolution in time of the total density $\int_{\Omega_i} u(x,t) dx$ in nodes $\Omega_i$ is depicted in the top panel of Figure 10, at times $t = 10, 100, 1000$. One observes that a smaller Lévy exponent $s_{nod} = 0.2$ allows particles to reach distant nodes more efficiently than approximately normal diffusion $s_{nod} = 0.8$. This confirms the interpretation in Section 4. The bottom panel of Figure 10 exhibits similar dynamics as in Experiment 1, where $s_{nod} = 0.8$ equilibrates the network’s density faster on long time scales.

We conclude that particles undergoing approximately normal diffusion ($s_{nod} = 0.8$) are slower, but more precise. Superdiffusing particles ($s_{nod} = 0.2$) explore the network faster, but take more time to equilibrate the density across the whole network. Similar observations have been made in [40], where the targeting efficiency of E. coli bacteria was studied in simulations. They observed that bacteria with higher motility, following a superdiffusion process, find targets faster, but are also at risk of moving rapidly away from the target due to the nature of their Lévy walk. Individuals with lower motility were slower but more precise.

In the current example, the distance between the sinks and sources also leads to a delay between when the particles reach a node at the source and find the sink to hop to another node. Coupled with the network dynamics, the delay leads to small density oscillations between neighboring nodes. For clearer illustration in Figures 10 to 13 we only consider the odd nodes of the network.

Fig. 11. Density distribution for big nodes $\Omega_b$, $s_{net} = 0.8$ (top panel), resp. 0.4 (bottom panel).

The following figures compare these conclusions to those obtained for a network of big nodes $\Omega_b$. 
In Figure 9b we observe that superdiffusion inside big nodes speeds up the equilibration of the densities considerably: For $s_{\text{nod}} = 0.2$ particles require a much smaller time to reach their distant target, the sink, than for $s_{\text{nod}} = 0.8$.

In Figure 11 we plot the density as a function of the node, in a log-log plot at times $t = 1000, 5000, 10000$. Note that because of the large distance between sources and sinks for nodes $\Omega_b$, the time scale to approach equilibrium increases significantly. We observe that superdiffusion inside the nodes accelerates the equilibration over the whole network in all cases, unlike for the small nodes $\Omega_s$ (Figure 10). In the bottom panel of Figure 11, for $s_{\text{net}} = 0.4$, we observe the accelerated diffusion particularly clearly, with a density distribution which is far from a Gaussian parabola in the log-log plot.

6.3.1. Network superdiffusion. From the discussion of Experiments 1 and 2 so far, we conclude that for the short-ranged network diffusion (3.2), proportional to $2^{-s_{\text{net}}|i-j|}$, superdiffusion in the nodes can accelerate the equilibration in the network, but it cannot lead to superdiffusion in the network. This is in line with the discussion in Section 5. We now consider a long-ranged network coupling proportional to $|i-j|^{-s_{\text{net}}}$, given by the Mellin-transformed $k$-path Laplacian (3.1) for $s_{\text{net}} = 1.5, 2, 2.5$ and 4. For an ordinary network of point nodes, superdiffusion was demonstrated in [22] for $s_{\text{net}} \in (1,3)$.

As in Figure 11, Figure 12 plots the density at times $t = 10, 100, 1000, 10000$ as a function of the node of the network in a log-log plot. The linear decay of the density and the peaked behaviour at node 1 indicate superdiffusion irrespective of the internal dynamics. Because of the strong network diffusion, for the big nodes $\Omega_b$ the internal proves irrelevant to the network dynamics.

In Figure 13 we exhibit the absence of superdiffusion for large Mellin exponent $s_{\text{net}} = 4$ and $s_{\text{nod}} = 0.8$: The density distribution recovers a Gaussian shape, characteristic of normal diffusion, as is clearly visible for longer times. Note that for the illustration of the Gaussian shape we have symmetrically reflected the network with respect to the $y$-axis.

Finally Figure 14 shows the equilibration of the density in node 1 with time. It confirms the interplay of the nodal diffusion process and the size of the node, as observed for the short-ranged network diffusion above in Figure 9.

6.4. Experiment 3: Uniform coupling and multiplex networks. When the coupling $T_{ij} = \text{Id}$ is uniform within all nodes, or equivalently every node is both source and sink of constant coupling strength, the metaplex networks studied in this article reduce to a multiplex network [54]. We consider $\alpha_{ij} = 1$ and $s_{\text{net}} = s_{\text{nod}} = s$ in the network from above. In fact, from the analysis of multiplex networks in Subsections 4.3.1 and 4.3.2 the external dynamics of the network will be independent of the node domains $\Omega_i$ and the internal diffusion process $s_{\text{nod}}$. It is determined by the coupling matrix $T$, not the diffusion within the nodes.

Figure 15a describes the density equilibration in node 1 for different values of $s$. In Figure 15b we show the equilibration of the density as a function of time for all the nodes of the network, when $s = 0.4$. We observe an overshooting of the density in nodes $\geq 2$ as particles cascade from node 1 to node 2 and further with a delay and pile up. The dynamics for different values of $s = 0.2, 0.8$ is shown in Figures 16a and 16b on the appropriate time scales. For $s = 0.8$ the characteristic Gaussian shape of an approximately normal diffusion emerges, while the superdiffusive peak is observed for $s = 0.2$. 

7. **Summary.** Complex networks are a much-used tool to represent and understand the global behavior of complex systems in terms of interacting subcomponents. The *metaplex networks* introduced in this article allow to include the internal structure, dynamics and function of these subcomponents into network theory.

In this article we provide the basic notions and motivation for the study of metaplexes. We show their relevance already for linear diffusion and superdiffusion processes in complex systems and illustrate the key role that the interplay between their endo and exo structures may take.

Our numerical results show that superdiffusion due to long-range hopping in a network, as in [22], survives irrespective of the internal structure of the nodes. The
Fig. 14. Density equilibration in node 1 for the Mellin-transformed k-path Laplacian for $\Omega_s$ (a) and $\Omega_b$ (b).

Fig. 15. (a) Density equilibration in node 1 for the multiplex coupling for different values of $s$. (b) Density equilibration for all nodes when $s = 0.4$.

Fig. 16. Density evolution for $s = 0.8$ (a) and $s = 0.2$ (b).
parameter range in which superdiffusion is observed, \( s_{\text{net}} \in (1, 3) \), is the same as for ordinary networks of point nodes without internal structure.

The effect of the internal dynamics in the whole network strongly depends on the geometry of the nodes and the nature of the coupling. When sinks and sources overlap, internal superdiffusion slows down the network dynamics and normal diffusion is faster on small scales. When sinks and sources are in separate, distant locations, superdiffusion in the nodes allows particles to explore the entire network much faster than classical diffusion. While it accelerates diffusion, the internal superdiffusion cannot, by itself, induce superdiffusion in the whole network.

Our results can be understood from the local description we provide of the distribution of particles inside each node. The combination of analytical methods for the PDE description in the node, and network methods for their interconnection in the exo-skeleton gives a new perspective not only on classical complex network descriptions, but also for the study of PDEs describing physical systems which can be either split into continuous regions interconnected in a discrete way or involve a network of internal degrees of freedom. This illustrates how the study of metaplex networks will draw tools from a wide range of areas, such as the geometric analysis of coupled PDEs and interface problems, spectral theory of operator matrices, high-dimensional stochastic processes etc. Conversely, it suggests the relevance of network theory for old problems in these fields, such as the problem of finding effective descriptions of interacting many-particle systems with high-dimensional internal degrees of freedom. From a computational point of view, numerical experiments for large metaplex networks become a challenge, due to the new internal degrees of freedom in each node. For applications to real-world networks, future work should explore methods which represent this internal dynamics efficiently. Model order reduction or generalized finite element methods [46] are examples which have been used in related settings to achieve a reasonable accuracy already for small degrees of freedom. It is worth noticing that the internal structure of the nodes in the metaplex might for some applications be replaced by a random delay \( \tau^v \) in every node \( v \), see the discussion in Section 5. This has been already proposed for the analysis of traffic flow in urban street networks [1].

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