Active transport and cluster formation on 2D networks

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Abstract. We introduce a model for active transport on inhomogeneous networks embedded in a diffusive environment and investigate the formation of particle clusters. In the presence of a hard-core interaction, cluster sizes exhibit an algebraically decaying distribution in a large parameter regime, indicating the existence of clusters on all scales. The results are compared with a diffusion limited aggregation model and active transport on a regular network. For both models we observe aggregation of particles to clusters which are characterized by a finite size-scale if the relevant time-scales and particle densities are considered.

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1 Introduction

Active transport processes play an important role for the functionality of social and biological systems. Examples are road traffic or active intracellular transport of vesicles and organelles by motor proteins that perform directed movement along microtubules or actin filaments. In recent years stochastic systems of self-driven particles have been applied to model intracellular transport of motor proteins in a number of works [1,2,3,4,5,6,7]. While the microtubules usually arrange in an ordered pattern (e.g. a radial structure in most mammalian cells, though longitudinal in neuronal axons), actin filaments often form randomly structured undirected networks. Therefore the investigation of transport on networks arises to be an interesting object of research.

Active transport on an undirected but regular network has been investigated by Klumpp et al. [5]. They studied the dynamic properties of diffusive non-interacting particles on a lattice with an embedded regular square network consisting of active stripes where particles perform biased motion. They showed that though movement of particles remains globally diffusive on long time-scales, the diffusion constant is enhanced by the presence of the network.

In transport systems considering steric exclusion interaction between particles, aggregation is a common phenomenon, manifesting in the formation of jams. Jams can form in one dimensional systems with single tracks due to boundary conditions (boundary induced phase transitions [9]), induced by defects [10,11,12,13,14] or they emerge spontaneously due to stochastic slow down of vehicles in highway traffic [15,16]. In two dimensional regular street networks, mutual interference of vehicles at intersections lead to jamming [17].

Studies of transport on inhomogeneous topological networks (graphs with nodes and edges, no distances) revealed an interesting phenomenology. E.g. non-interacting particles performing a random walk exhibit an inhomogeneous density distribution on the nodes [18], while inclusion of an attractive zero range interaction even allows...
the particles to aggregate and form a condensate, corresponding to nodes containing a finite fraction of particles [19]. These results show that the structure of a transport network strongly influences transport properties. In order to model active transport on actin filament networks, it is therefore necessary to consider realistic network structures.

For many biological processes, concentration gradients are crucial. One example is the aggregation of proteins inside the cell or in the cell membrane. Clusters of aggregated proteins can be observed and characterized experimentally for example by fluorescence microscopy. In some cases these clusters are essential for cell functionality but they can also lead to dysfunctions or even apoptosis. In yeast cell membranes for example one observes the aggregation of Erd2p-receptors which can promote the internalisation of toxins [20]. The existence of a quasi two-dimensional irregular actin filament network beneath the membrane [21,22] suggests a jamming mechanism of vesicles prior exocytosis, resulting in clusters of receptors on the membrane surface. The limits of resolution in optical microscopy [23] do not allow the localization of single receptors. By contrast the size of larger particle aggregates can in principle be given with relatively high precision. Therefore it is useful to relate the cluster size distribution with microscopic transport mechanisms by means of theoretical modelling.

In this work we propose a model for active transport of extended hard-core particles (corresponding to vesicles) on a two-dimensional randomly disordered network embedded in a diffusive environment. The model is motivated by intracellular transport on submembranal networks, we therefore adapt the model parameters to this reference system. We check particle configurations in order to identify the formation of clusters and investigate cluster size distributions. The results are compared with a regular network in diffusive environment and a diffusive system without network where attractive particle-particle interactions promote cluster formation similar to a van der Waals gas. The main focus will be on robust properties of clusters that serve as criteria to discriminate between different microscopic dynamics.

2 Network models

In the following, we introduce stochastic models in order to study the influence of the network structure on dynamical properties. While the presence of a network in a diffusive environment without interaction between particles merely leads to the enhancement of the diffusion process [8], we are rather interested in collective patterns emerging due to interactions. Our simulations use stochastic dynamics in order to integrate the many-particle Master equation. At each time step particles are randomly chosen and updated (random sequential update) according to the rules given in table 1 applying periodic boundary conditions. Time steps are normalized so that on average each free particle performs one diffusive step per time step $\Delta t$.

2.1 Regular networks

As a the first example for active transport on networks a discrete lattice gas model similar to the model investigated in [8] is considered. $N \times N$ sites are arranged in a square lattice of edge length $Na$ where $a$ is the lattice spacing. Each site can either be empty or occupied by at most one particle. We distinguish the particle states attached (A) and detached (D). The system contains stripes of active sites that constitute a regular square transport network where attached particles perform a directed motion. Detached particles always move diffusively. The orientation of stripes was chosen randomly with equal probability. Attached Particles on active stripes can unbind becoming detached, while detached particles can bind to stripes becoming attached. Steps that would result in double occupation of a site are prohibited if not state differently.

Compared to the dynamics of non-interacting self-driven particles qualitatively new features arise due to the steric particle-particle interactions at intersections of the network. Here we introduce an additional parameter, the blocking
Fig. 1. Illustration of the dynamics in a regular network. Dark grey discs are particles diffusing freely with rate $\omega_D$. Black discs represent attached particles that can only step in the preferred direction of the active stripe they occupy (bold arrows) with rate $p$. On filament sites (light grey), particles can interchange between attached and detached state with rates $\omega_a$ and $\omega_d$, respectively. Crossed arrows denote steps that are inhibited due to the exclusion principle.

**Probability:** If at least two particles are at sites adjacent to an intersection site, each particle may only access the intersection site with the probability $1-b$ (cf. figure 1). Particles on intersection sites retain their moving direction.

The explicit rules for the particle dynamics are displayed in table 1 and illustrated in figure 1. We have chosen the default parameter values analogous to [8] $\omega_D = 1$, $p = 0.5$, $\omega_d = 0.02$ and system size $N = 200$. In [8] the attachment rate is equal to one, which corresponds to an effective attachment rate $\omega_a = 0.25$ if a particle is on an adjacent non-active site. To be consistent with the subsequent continuous space model, we choose $b = 1$.

### 2.2 Inhomogeneous networks

Generalising to continuous space and allowing for arbitrary directions and lengths of active stripes we present a continuous model with randomly generated linear filaments where hard-core particles can perform directed paths along these filaments.

#### 2.2.1 General properties of the model

The main components of our model are filaments and particles interacting via a spherical hard-core potential represented by a disc of radius $r_p$. This hard core potential is implemented by cancelling any steps that would result in an overlap of discs. Filaments are represented by linear sequences of nodes with a distance of $d_n$ between nodes. They are directed with a (-)-end at which they can shrink and a (+)-end at which new nodes can be generated to elongate the filament. Particles can attach to nodes that are within a distance less than $d_n$ and perform steps to adjacent nodes in the (+)-direction of the filament.

#### 2.2.2 Dynamics of filaments and particles

The filament network is generated by stochastic dynamics, updating network configurations by the processes commented in table 2 and illustrated in fig. 3. Since the model is motivated by the dynamics of actin filaments some additional parameters originating from the biological processes in cells are included.

The quantity $\rho_{ARP}$ introduced in table 2 represents the density of free ARP2/3-complexes that serve as nucleation and branching seeds for filaments, while the actin density $\rho_{act}$ corresponds to the density of free nodes (actin monomers) constituting the filaments. Their initial values are $\rho_{0,ARP}$ and $\rho_{0,act}$ which corresponds to the case if all monomers are dissociated. The densities decrease with the growing filament network as shown in fig. 4. After 5000 steps a stationary actin density is reached. We assume that also the structure of the network is stationary and in qualitative agreement with a real actin network. We therefore stop network dynamics at this point.

After construction of the network particles obeying the exclusion principle are fed into the system at random positions. As mentioned above, the particle positions are updated following a random sequential update scheme, whereby the...
| Process      | Particle state(s) | Description                                                                 | Probability |
|--------------|-------------------|------------------------------------------------------------------------------|-------------|
| Diffusion    | D                 | Detached particles move to sites randomly chosen from the four neighbors      | $\omega_D = 1$ |
| Forward Step | A                 | Attached particles move to the next site in forward direction of filament    | $p = 0.5$   |
| Attachment   | D→A               | Detached particles on filament sites becomes bound                           | $\omega_a = 0.25$ |
| Detachment   | A→D               | Attached particles become detached                                          | $\omega_d = 0.02$ |
| Blocking     | D                 | Forward movement of particles adjacent to intersection sites is inhibited if other particles occupy sites adjacent to intersection | $b = 1$ |

Table 1. Brief prescription of the dynamic processes in the square lattice model. Column 2 displays the particle states “attached” (A) and “detached” (D). The right column displays the probability that the respective process occurs within one time step. Numerical values given in the right column are default values which are used if not stated else and are chosen to fit the ones in [8].

| Process    | Description                                                                 | Probability |
|------------|------------------------------------------------------------------------------|-------------|
| Nucleation | Initialization of filaments with arbitrary direction at an arbitrary point in the system. The (-)-end receives a cap inhibiting shrinking. | $\omega_n \rho_{act} \rho_{ARP}$ |
| Branching  | New filaments are initialized at an existing one (not necessarily the (+)-end; angle between parent filament and branch=70° [24]. | $\omega_b \rho_{act} \rho_{ARP}$ |
| Growth     | New nodes are generated at the (+)-ends of filaments.                        | $\omega_g \rho_{act}$ |
| Shrinking  | Nodes are removed at the (-)-end of filaments if the end is not capped.     | $\omega_s$ |
| Uncapping  | Caps are removed.                                                            | $\omega_u$ |

Table 2. Dynamics of the filament network.

Although we do not consider a particular biological system, we choose parameters to fit the typical order of magnitude in real vesicular transport. If not stated differently, we will use default parameters displayed in Table 4 for our simulations. The referenced works used experimental and modeling techniques to obtain the data given in the third column. For particle dynamics, we choose the parameters to be consistent with the discrete model introduced in the last section relying on the model in [8].

3 Results
3.1 Characterisation of Clusters

As already mentioned in the introduction we aim to relate the microscopic particle dynamics to the size distribution of their aggregates. In this section we discuss the definition of clusters for the different model systems.

Clusters are groups of particles that are connected by overlapping neighborhoods. We therefore introduce the $\lambda-$neighborhood of a particle representing a disc of radius $\lambda$ around the
Process | Particle state(s) | Description | Probability
--- | --- | --- | ---
Diffusion | D | Detached particles move in a random direction. Step widths are uniformly distributed between 0 and $2l_D$. | $\omega_D = 1$
Step | A | Attached particles move to adjacent node in (+)-direction. | $p$
Attachment | D→A | Particles bind to nodes if their distance is less than $d_b$, becoming 'attached'. | $\omega_a$
Detachment | A→D | Particles detach. | $\omega_d$

Table 3. Particle dynamics. A='attached'; D='detached'

| Parameter name | Reference | Reference Value | model parameters |
|--- | --- | --- | --- |
| Filament dynamics: | | | |
| nucleation rate $\omega_n$ | 25 | $8.7 \times 10^{-5} \mu M^{-2} s^{-1}$ | $1.0 \times 10^{-5} \text{lu}^{-6} \text{tu}^{-1}$ |
| growth rate $\omega_g$ | 24 | $8.7 \mu M^{-1} s^{-1}$ | $0.25 \text{lu}^3 \text{tu}^{-1}$ |
| shrink rate $\omega_s$ | 26 | $4.2 s^{-1}$ | $0.075 \text{tu}^{-1}$ |
| branch rate $\omega_b$ | 25 | $5.4 \times 10^{-4} \mu M^{-3} s^{-1}$ | $0.0001 \text{lu}^9 \text{tu}^{-1}$ |
| uncap rate $\omega_u$ | 25 | $0.0018 s^{-1}$ | $0.0001 \text{tu}^{-1}$ |
| actin density $\rho_{act}$ | 27 | free actin: $0.1 - 1 \mu M$ | $2 \text{tu}^{-3}$ |
| ARP2/3 density $\rho_{ARP}$ | 28 | $0.1 \mu M$ | $0.1 \text{lu}^{-3}$ |
| Particle dynamics: | | | |
| particle radius $r_p$ | 29 | 42.5nm (average) | $0.5 \text{lu}$ |
| binding distance $d_b$ | 8 | 1 site (50nm) | $0.5 \text{lu}$ |
| node distance $d_n$ | 21 | 36nm | $0.36 \text{lu}$ |
| attachment $\omega_a$ | 8 | 1/4 of diffusive steps | $0.25 \text{tu}^{-1}$ |
| detachment $\omega_d$ | 8 | $0.8 s^{-1}$ | $0.02 \text{tu}^{-1}$ |
| diffusive step $l_D$ | 8 | 1 per time step | $0.5 \text{lu}$ |
| step rate $p$ | 8 | $20s^{-1} \Rightarrow v = 1 \mu m/s$ | $0.75 \text{tu}^{-1}$ |
| particle density $\rho$ | [30] | 10-60 vesicles in bud ($\sim 0.75 \mu m$ radius) | $0.04 \text{lu}^{-3}$ |

Table 4. Default parameters of the model which are biologically motivated by transport of vesicles by myosin on actin filaments. The referenced values are either based on experimental data or existing models for intracellular transport [8] and filament dynamics [28]. Model parameters are chosen to be in the order of magnitude of referenced values, fitted to time and space scale of the simulations. Length scale: $1 \text{lu} = 100 \text{nm} = 2r_p \Rightarrow 1 \mu M = 0.6 \text{lu}^{-3}$. Time scale: $1 \text{tu} = \Delta t = 0.025s$. By default we consider square systems of system length $L = 200 \text{lu}$.

As shown in figure 4 the amount of overall actin is about ten-fold larger than free actin. Thus we chose this factor to extrapolate the experimental data to $\rho_{act}$.

A cluster is defined as a set of particles included in a connected area of λ–neighborhoods (cf. fig. 5). If continuous space variables are used, there exists no natural scale which identifies two particles as neighbors. We therefore have to specify the value of $\lambda$. In order to extract relevant results, we fix $\lambda$ so that qualitative results are robust on deviations. If not stated differently we choose $\lambda = 2r_p$, which turns out to meet this condition (cf. fig 19).

In lattice models, static particle clusters are usually considered as as connected sets of adjacent particles. However this definition is not appropriate in this context since clusters move by propagation of vacancies. Therefore we consider particles separated by a single vacancy as belonging to the same cluster.
Fig. 2. Illustration of the particle dynamics in an inhomogeneous network. Dark grey discs are free particles, black discs represent particles attached to filaments stepping to adjacent nodes (distance $d_n$) with rate $p$. Particles can attach to filaments with rate $\omega_a$ if they are within the binding distance $d_b$ and detach with rate $\omega_d$. Overlapping is inhibited due to exclusion.

Fig. 3. Illustration of the filament dynamics. Filaments are implemented as sequences of nodes (small dots, corresponding to actin monomers) separated by a distance $d_n$ (short bars). Filament segments are polarized, with a (+)-end where nodes are created to elongate, and a (-)-end where nodes dissociate causing shrinking.

Fig. 4. Density of free actin $\rho_{\text{act}}$ in dependence on time. After 5000 time steps a stationary state is reached.

Our main interest is in ensemble and time averages of *cluster size distributions (CD)* and their asymptotic behaviour. CDs display the relative frequency of cluster sizes emerging in the system. If not stated differently we averaged over 50000 time steps within individual runs, evaluating cluster distributions in distances of 500 time steps, taking an ensemble of 100 samples.

Clustering also occurs for random particle configurations. In fig. 6 and 7 we displayed cluster size distributions of random configurations in discrete and continuous space for different particle densities. Here the density $\rho$ is the particle number per area unit which corresponds to $(2r_p)^2$ in continuous space and one site in the lattice model. If densities are not too large, the formation of large clusters is impeded resulting in an exponentially decaying cluster size distribution. For high densities one observes a small peak at the right end. At these densities clusters spanning the whole system emerge. In order to rule out these kinds of random clustering we will only consider densities below the regime of spanning clusters at relevant scales $\lambda$. In this work we are interested in cluster formation mechanisms beyond random clustering.

In the following, we will focus on particle configurations and cluster-size distributions in several transport models.
3.2 Aggregation without network

A possible scenario for clustering of receptors on the cell surface is aggregation due to an attractive interaction between proteins diffusing in the cell membrane. This process can be formulated as an equilibrium model consisting of diffusing hard-core particles (radius \( r_p \approx 10\text{nm} \); within the size-scale of membrane proteins) interacting via an attractive potential. We apply the particle dynamics discussed in sec. 2.2 but do not consider filaments. In addition we introduce a particle-particle interaction between the particles realized by a square well potential of the form

\[
V(x - x') = \begin{cases} 
-V_0 & \text{for } |x - x'| \leq d_V \\
0 & \text{for } |x - x'| > d_V 
\end{cases}
\]  

(1)

where \( x, x' \) are particle positions. This potential can be implemented using a Metropolis acceptance probability \( p = \min\{e^{-\beta(V(x_{n+1}) - V(x_n))} , 1\} \) for a step from \( x_n \) to \( x_{n+1} \) (\( n \) denotes the time index). In the following we use dimensionless quantities and put \( \beta = 1 \). The default parameters are \( V_0 = 2, d_V = 3.5r_p \) and particle density \( \rho = 0.04 \). Assuming a diffusion constant for membrane proteins \( D \approx 0.0025\mu m^2/s \) we choose a time step corresponding to \( \Delta t = 0.02 \text{ seconds} \) so that one diffusive step of length \( r_p = 10\text{nm} \) is performed per time step \( \Delta t \).

In fig. 8 typical particle configurations at several runtimes are displayed, while in fig. 9 ensemble averages of cluster size distributions are shown. Initial clustering already occurs on a rather small time-scale. Regarding the particle config-
urations we see that the number of clusters decreases with increasing runtime while the average size of remaining clusters increases. This is due to diffusion and merging of existing clusters after long times. Movement of large clusters is strongly suppressed, so that merging occurs quite slowly. The coarsening process can also be observed in the cluster size distribution. We observe a characteristic scale for larger clusters, manifesting in the emergence of a maximum, indicating a characteristic scale for cluster sizes. The dominant clusters always are within the same size-scale which increases with time.

Since the cell membrane changes its structure continuously, patterns arising at time-scales corresponding to a finite fraction of a cell cycle cannot be assumed to be in a stationary state. Computing time averages we therefore focus on intermediate times and fix the averaging interval starting at 20000 time steps (corresponding to \( \sim 7 \) minutes in real time) after random initialisation of particles and ending at 30000 time steps. The time interval lies in the transient regime for default parameters. Within this interval we computed cluster size distributions (time and ensemble averages, 200 samples) for different parameter regimes and displayed them in figure 10. One observes that for weak interaction no significant clustering occurs manifesting in an exponentially decaying CD, while for strong interaction \( V_0 \), including the default parameters, a maximum emerges hallmarking the formation of clusters.

### 3.3 Directed transport on regular networks

In this section we examine features of particle configurations and cluster distributions in the model introduced in section 2.1, i.e. a regular network of active stripes. As in the last section we start time averaging after \( t_s = 20000 \) time steps. We carefully checked that a stationary state has been reached at this point. (cf. fig. 12). As time averaging interval we choose 50000 time steps. In fig. 11 particle configurations for moderate and high densities are displayed. For particle density \( \rho = 0.04 \) one observes small L-shaped clusters centering at intersections. For higher densities it appears that clusters that are becoming

![Fig. 8. Configurations of particles (black discs = particle neighborhoods with radius \( \lambda = 2r_p \)), exhibiting a mutual attractive interaction. Snapshots at different times for \( V_0 = 2, dV = 3.5, \rho = 0.04, L = 200, 1 \) timestep \( = 0.025 \) sec. One observes that already at small times clusters form and for long run times the number of clusters decreases, while the average size of cluster increases.](image-url)
larger and merge with each other to form large mesh-shaped clusters (cf. fig. 11(b)). However, in this case clusters are hardly distinguishable and not well separated which results in sensitive dependence on the coarse graining scale (cf. fig. 14). In fig. 13 we plotted the cluster size distributions averaged over time and 100 individual runs. Examining the cluster size distributions in fig. 13 one observes an exponential decay for densities which are biologically relevant (cf. see also the configuration in figure 11(a)). Therefore only small clusters emerge while large clusters are exponentially suppressed.

For large densities ($\gtrsim 0.1$) the decay of the cluster size distribution becomes algebraic, indicating that clusters on all size-scales exist. These large clusters correspond to the ones generated by merged small clusters as displayed in fig. 11(b).

### 3.4 Inhomogeneous networks

The filament growth dynamics described in section 2.2 generate a network where single filaments have random length and direction. In order to keep dynamics simple but retaining the crucial features of disordered networks, we here neglect branching and the dynamics of ARP2/3 to obtain a network of uncorrelated filament orientations. The other processes are required to obtain a disordered stationary network configuration.

![Fig. 9.](image)

Fig. 9. Cluster distributions in dependence on the runtime in a system without network but attractive square well interaction potential. Parameters are $V_0 = 3$, $dV = 3.5$, $\rho = 0.04$, $L = 200 r_p$, average over 200 runs. A maximum establishes, that moves slowly towards larger scales.

![Fig. 10.](image)

Fig. 10. Plots of cluster distributions in the attractive particles model at intermediate times ($t = 20000 - 30000$ time steps) in dependence on the potential depth $V_0$ (a), the potential width $dV$ (b) and particle density $\rho$ (c). One observes the transition from an exponential decay (non clustering phase) to the formation of a maximum, corresponding to clusters at this size-scale (condensation). Default parameters are given in the text.
3.4.1 Dynamics of interacting particles

We obtained particle configurations and cluster size distributions applying steric interactions, which are shown in figures 15-19. The time evolution of the cluster size distribution (fig. 17) shows that a stationary state is reached after 10000 time steps. Starting time averaging after 20000 time steps (averaging interval=50000 time steps) therefore captures the steady state dynamics.

The configuration for $\rho = 0.04$ (fig. 15) shows that well separated compact clusters exhibiting different sizes emerge (see also scaling in fig. 19), while at $\rho = 0.008$ no clusters are observed. In a large parameter regime including the biological relevant default parameters (table 1), the asymptotic decay of the CD is algebraic in contrast to the predominant exponential behaviour on a regular network. For a given combination of network structure and particle dynamics the properties of the CD are determined by the particle density, while varying other parameters does not lead to qualitative changes except in extreme regimes. In fig. 20 cluster size distributions of a regular and inhomogeneous network are compared. One observes that clustering is significantly enhanced in the inhomogeneous network.

Due to the finite number of particles there is a cut-off at the upper end (e.g. in fig. 19). Fig. 18(a) shows that for increasing system size $L$ the cut off regime tends to larger values, indicating that this indeed is a finite size effect and asymptotically algebraic behaviour prevails in the thermodynamic limit. Though the exponent $\gamma$ of the algebraic decay varies for different particle densities, the algebraic form is a robust feature. This indicates that in the thermodynamic limit clusters on all size-scales exist. In contrast to reg-

\[ \text{Fig. 12. Cluster size distributions in a regular network for different runtimes (given in time steps). For a given runtime, we chose the last 100 steps to perform the measurement, taking 100 samples. The CD does not change after 1000 time steps, indicating that the system is in a stationary state.} \]
Fig. 13. Cluster distributions in a lattice gas model with exclusion interaction and a regular square network of active stripes in dependence on particle density $\rho$, logarithmic plot (a), double logarithmic plot (b) blocking rate $b$ (c), and mesh size $l$ (d). One observes that cluster size distributions decay exponentially for moderate densities resulting in a finite size-scale of clusters. For very large densities decay is algebraic indicating the emergence of clusters on all size-scales (see also fig. 11). Default parameters: see table 2.

Fig. 14. Cluster size distributions in the regular network obtained by using different definitions of the coarse graining scales. $d$ is the distance allowed between two particles to connect a cluster. One observes that while variations are small for moderate density, there is a significant influence on the scale for larger densities, indicating that clusters are not well separated.

Fig. 15. Particle configurations (black discs = neighborhoods with $\lambda = 2r_p$) for default parameters ($\rho = 0.04$) and system size $L = 200r_p$. One observes big and small clusters. This general picture is predominant for a large parameter regime and moderate densities.

Fig. 16. The long range behaviour of the CD vanishes if crucial properties for cluster formation are absent, which can be seen in the particle configurations in fig. 16. E.g. for small densities of the filaments (controlled by the actin density parameter $\rho_{act}$), filaments are only dilutely distributed and no network with intersections can constitute networks, scale free clustering occurs even for moderate densities ($\rho \approx 0.012$) exhibiting a pattern of well separated clusters.
3.4 With branching

In real actin networks, branching of filaments takes place quite frequently, resulting in a dendritic network structure. If one is interested in the dynamics of vesicle transport on submembranal actin networks, one has to consider this process as well. We checked CDs in a system with finite branching rate (here: branching probability $\omega_b = 0.0035$) including the dependence of growth dynamics on the ARP2/3-density $\rho_{\text{ARP}}$ (cf. section 2.2). In this system, filament orientations are highly correlated.

In fig. 21 we displayed cluster size distributions for different particle densities $\rho$. Like in the more basic case of uncorrelated filaments, $\omega_b = 0$, one observes an algebraic decay as well. This indicates that the scale free behaviour of the clusters is a robust feature of inhomogeneous transport networks and active particles exhibiting mutual steric interaction.

3.4.3 No exclusion

In order to distinguish the influence of exclusion and network structure, we switch off the exclu-
Fig. 18. Cluster size distributions in a diffusive system with an inhomogeneous active transport network. Default parameters are the ones given in table 4, while single parameters are varied: (a) system size $L$, (b) particle density $\rho$, (c) detachment rate $\omega_d$, (d) actin density ($\sim$ network density) $\rho_{\text{act}}$. In a vast regime of parameter space the cluster distribution decays algebraically indicating the absence of an intrinsic size-scale. Only for small densities or extreme parameters clusters do not emerge. Default parameters: see table 4.

Fig. 19. Cluster size distributions in the inhomogeneous network for different coarse graining scales. $\lambda$ is the radius of the neighborhood as defined in section 3.1. As well in the regime where large clusters emerge and in the non-clustering regime, the dependence on the coarse graining scale is weak indicating well separated clusters.

Fig. 20. Comparison of regular and inhomogeneous network displaying cluster size distributions for particle density $\rho = 0.04$. While the CD decays exponentially in the regular network, its slope is algebraic in the inhomogeneous one, demonstrating significant enhancement of clustering by the inhomogeneous network structure.

Fig. 21. Cluster size distribution for branch rate $\omega_b > 0$. Like in the system with non-correlated filaments, the CD exhibits algebraic decay for large clusters.
sion interaction. In figure 22 cluster size distributions of the inhomogeneous system with and without exclusion are compared for default parameters. One observes that switching off exclusion suppresses formation of large clusters significantly.

In order to quantify the inhomogeneity of the particle distribution, we determined the average particle density in the proximity of intersection points. Figure 23 displays the relative frequency of intersection points with local particles density within density intervals of length 0.005. We see that while the density of particles is almost the same on all intersections in the regular networks, there is a wide distribution of dilute as well as crowded intersection points in the inhomogeneous network, marking a highly inhomogeneous distribution of particles.

### 3.5 Clustering mechanisms

In this section we want to conjecture mechanisms involved in the formation of clusters in active transport systems.

The most common clustering mechanism is probably nucleation of droplets in oversaturated van-der-Waals gases. If temperature or density is low enough, the attractive interactions of gas molecules favours aggregation of particles in clusters representing fluid phase droplets. A similar system was shown in section 3.2 to exhibit clusters on characteristic size-scales.

Fig. 24 illustrates a mechanism that is similar to clustering (jamming) at intersections in city street networks [17]. If two particles are on sites adjacent to an intersection site, they can block each other, forming an initial two-particle cluster. They can only leave the cluster by bypassing (which is inhibited for block rate $b = 1$) or detachment followed by hopping on an adjacent non-filament site. The rate of these events corresponds to the flow out of the cluster $J_{\text{out}}$. Particles attached to one of these filaments can run into this cluster from behind, contributing to the flow into the cluster $J_{\text{in}}$. If inflow is larger than outflow, a queue emerges similar to high density regimes in TASEP-like driven lattice gases [9]. As long as inflow is larger than outflow, clusters grow, decreasing the number of free particles, thus $J_{\text{in}}$ decreases. In contrast, if inflow is less than outflow, clusters shrink, releasing particles that contribute to an increasing free-particle density, which consequently increases $J_{\text{in}}$. Finally, each cluster will reach a stationary state with $J_{\text{in}} = J_{\text{out}}$, providing a scale for the cluster sizes which is spatially invariant.

In inhomogeneous networks intersection points also serve as nucleation seeds for clusters. However, the density of intersection points may vary. If multiple filaments cross in a small area, clusters that extend over other intersection points constitute additional obstacles for particles moving on other filaments. Hence clusters will extend on these filaments (see fig. 25), where they might cover additional intersection points.
4 Discussion

We examined transport of hard core particles on regular and inhomogeneous networks embedded in a diffusive environment. The models consist of regions where particles perform diffusive motion and of one dimensional stripes (filaments) to which particles can attach and perform directed motion. In most parameter regimes clusters are observed which are assumed to emerge due to blockage of particles at intersecting points of filaments. As a reference for clustering without transport networks we examined cluster features of a model with attractive interaction of particles.

A detailed analysis of cluster size distributions shows that the network structure as well as the particle-particle interaction change qualitatively the form of the CD. On regular networks one typically observes exponentially decaying CDs at low densities, i.e. one can attribute a typical scale to the clusters. Algebraic CDs are only observed at large densities when single clusters merge to form large mesh-shaped cluster complexes.

In contrast disordered networks, where filament lengths and orientations are randomly distributed, exhibit algebraic cluster size distributions in a wide range of the parameter space, indicating that clusters on all size-scales exist. It is important to notice that the algebraic CDs are observed one order of magnitude below the respective densities in reference systems. In the disordered network, clusters are well separated exhibiting a compact structure.

Switching off the hard core potential results in a strong suppression of large clusters, while the distribution of particles in the disordered network still remains inhomogeneous. Hence, inhomogeneity of the network structure as well as exclusion interaction strongly enhance the formation of macroscopic clusters.

The analysis of cluster size distributions in the system of diffusive particles with attractive interaction shows clustering on a finite size-scale and a maximum in the cluster size distribution. While nucleation of clusters occurs on very small time-scales, the average size of clusters grows slowly. It is assumed that the stationary state is attained at very long times exhibiting only few or even a single large cluster including most particles of the system.

In summary, we have found that the microscopic particle dynamics as well as the network structure have significant influence on the qualitative form of the cluster distribution. From our
point of view this observation is of great importance for the analysis of biological systems since it is often not possible to identify the underlying microscopic mechanisms leading to experimentally observed aggregation. In these cases the analysis of the cluster distribution on larger scales may answer the question whether observed patterns are the result of active transport or of aggregation due to attractive interactions.

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