

# Analysis of the collaboration mode of molecular motors in the vesicle transport system of the Golgi apparatus

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## Abstract:

This article focuses on the collaborative mode of molecular motors in the Golgi vesicle transport system and conducts a systematic analysis. The article begins with an overview of the structure, function, and transport processes of the system, introducing the conventional mechanisms of various molecular motors within cells. Subsequently, it identifies the types of molecular motors involved in vesicle transport and their positioning at various stages, providing a deep analysis of co-directional and reverse collaborative modes and synergistic regulatory mechanisms. Furthermore, the article explores diseases caused by abnormal collaboration and regulatory strategies. Finally, it provides an outlook on new research techniques and drug development applications, offering key insights into understanding cellular physiology, pathology, and innovative therapies.

## Keywords:

Golgi  
Vesicle transport system  
Molecular motor  
Collaboration mode

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## 1. Introduction

In the field of cell biology, the Golgi complex, as an important hub for intracellular material transport and processing, has been a focus of scientific research. The Golgi vesicle transport system plays a crucial role in maintaining intracellular environmental stability, regulating cell signaling, and protein synthesis and modification. In this system, molecular motors serve as key factors driving vesicle transport, and studying their collaboration modes is significant for revealing the molecular mechanisms of intracellular material transport.

In recent years, with the continuous development of molecular biology, cell biology, and biophysics, researchers have gained a deeper understanding of the molecular motors in the Golgi vesicle transport system. However, many unknowns still exist regarding the collaboration modes of molecular motors during Golgi vesicle transport.

## 2. Overview of the Golgi Vesicle Transport System

### 2.1 Brief Introduction to the Structure and Function of the Golgi Complex

The Golgi complex is a polar organelle consisting of multiple stacked flat membrane saccules, which can be roughly divided into the cis-Golgi network (CGN), medial saccules, and trans-Golgi network (TGN). The CGN, located near the endoplasmic reticulum, is the entrance for vesicles into the Golgi complex and is responsible for receiving newly synthesized proteins and lipids from the endoplasmic reticulum. The medial saccules region is rich in glycosylation modification enzymes, undertaking the task of modifying and processing cargo such as complex glycosylation and sulfation. The TGN, located at the exit end of the Golgi complex, is responsible for sorting the modified substances, precisely packaging them into different vesicles, and transporting them to various targets within the cell or secreting them outside the cell. For example, in the synthesis pathway of secreted proteins, immature proteins transported from the endoplasmic reticulum undergo a series of modifications in the Golgi complex to acquire biological activity, and are then transported to the outside of the cell membrane to perform their functions.

### 2.2 The basic process and key nodes of vesicular transport

Vesicular transport is a way of exchanging substances between various organelles in the cell, the cell and the external environment, and this transport process is mainly regulated by coat protein, a supramolecular complex that aggregates on the cytoplasmic side surface of the membrane in the eukaryotic cell membrane system to promote the sorting of proteins, plasmids, etc. (i.e., cargoes) and the formation of transport vectors. Various types of envelope proteins have been described, including lattice proteins, COPI, COPII, retromer and retriever, etc. Envelope proteins continuously interact with the corresponding regulatory factors (or regulatory proteins) to precisely regulate the transport of cargoes while participating in the cargo transport process, and function at different locations in the membrane system<sup>[1]</sup>.

Once the vesicles are detached from the donor membrane, they start their transport journey, travelling

along the cytoskeletal tracks in different regions of the cell with the help of molecular motors. Upon arrival at the target membrane, the vesicle recognises the corresponding receptor on the target membrane with the help of Rab proteins to achieve a precise stopover, and then ultimately relies on the SNARE protein complex to facilitate the fusion of the vesicle with the target membrane and unload the cargo to the target site. In this regard, the precise screening of cargo during vesicle formation, navigation and orientation during transport, and specific recognition during fusion are the key links to ensure the accuracy of transport.

## 3. molecular motors in the Golgi vesicle transport system

### 3.1 Identified types of involved molecular motors

In the Golgi vesicle transport system, kinesins and dynamin are the main forces. Among the kinesins, kinesin-1 and kinesin-2 members frequently appear in the early stage of vesicle transport from the endoplasmic reticulum to the Golgi, as well as in the forward transport process within the Golgi; kinesins are mainly responsible for the control of reverse transport, ensuring that substances that need to be recycled and corrected can be returned to the endoplasmic reticulum or the near endoplasmic reticulum region of the Golgi to maintain the precision and cyclicity of the vesicle transport. 3.2 Molecular motors in vesicles

### 3.2 Positioning of molecular motors in various stages of vesicle transport

At the initial stage of vesicle formation, although molecular motors are not yet directly involved, some regulatory proteins have been aligned around the vesicle to prepare for the attachment of molecular motors<sup>[2]</sup>. During vesicle transport, kinesins are usually bound to the membrane surface of the vesicle near the front end of the transport direction, using the positive orientation of microtubules to drive the vesicle towards the opposite side of the Golgi or the cell membrane, while most of the kinetic proteins are localised on the side of the vesicle away from the direction of transport in preparation for balancing the excessive forward transport or initiating the

reverse transport when needed. As the vesicle approaches the target membrane, the activity of the molecular motor decreases, but it still helps to keep the vesicle stable near the target membrane through a weak force for the subsequent fusion process.

It is worth noting that there are subtle differences in the localisation of molecular motors in different cellular environments. In nerve cells, where the need for long-distance vesicle transport is prominent, the molecular motors are more firmly positioned to ensure the precise delivery of transmitters. With the help of fluorescent labelling, ultra-high resolution microscopy and other technologies, researchers have tracked their dynamics in real time, and found that changes in the temperature and pH of the environment can interfere with the positioning of the molecular motors, which also provides a key research direction for overcoming diseases caused by vesicle transport failures.

## **4. Analysis of molecular motor collaboration mode**

### **4.1 Isotropic Collaboration to Facilitate the Transport Process**

Kinesin, as a kind of molecular motor, can transport various goods along the microtubule track to supply cellular life activities. The same vesicle surface can combine kinesin-1 and kinesin-2 family members at the same time, which have the ability to hydrolyse ATP, and when these kinesins hydrolyse ATP synchronously, it will generate a strong synergy. This synergy not only improves the transport speed of the vesicles, but also allows the vesicles to maintain a more stable posture during transport, thus travelling along the microtubules more efficiently.

The cargo of proteins and lipids loaded in the vesicles can be delivered to the Golgi antipodal network on time with the collaborative transport of kinesins<sup>[3]</sup>. This process is crucial for the normal physiological function of the cell, as the Golgi antipodal network is an important site for further processing, sorting and packaging of proteins and other substances. Only when these goods arrive on time and accurately can the subsequent secretion process be guaranteed to proceed smoothly, as well as the normal development of physiological activities

such as intracellular membrane vesicle fusion, which in turn guarantees the high efficiency of the cellular secretion function and the maintenance of normal cellular metabolism and physiological state.

### **4.2 Reverse collaboration to maintain transport homeostasis**

Inside the cell, kinesins and dynamin work together to maintain the balance of vesicle transport through an exquisite mechanism of reverse collaboration. Kinesins are responsible for transporting vesicles rapidly from the endoplasmic reticulum to the Golgi apparatus and even to the cell edge, a process that requires great efficiency and accuracy<sup>[4]</sup>. However, during transport, incorrect distribution of cargo or excessive accumulation of material may occur, and this is where the role of kinesins becomes particularly important.

Kinesin is able to sense these abnormal signals and respond quickly. It exerts force in the opposite direction to that of the kinesin, pulling the vesicle back to its proper position in the endoplasmic reticulum or Golgi apparatus<sup>[5]</sup>. This process involves not only the precise adjustment of the vesicle position, but also the reconfiguration of the cargo components, which ensures the orderly transport of substances within the cell.

The importance of this reverse collaboration mechanism lies in its ability to correct errors in the transport process in a timely manner and prevent cellular dysfunction caused by substance mismatch. The intervention of dynamin is not only complementary to the role of kinesin, but also a necessary regulation, which enables the vesicular transport system to maintain high efficiency while coping with emergencies and maintaining the stability and adaptability of the system. Therefore, this collaborative mode is the key to maintaining the balance of intracellular substance transport and is essential for the normal functioning of the cell.

### **4.3 Synergistic regulatory mechanisms, signalling pathways and adaptor proteins**

In the microcosm of the cell, the co-regulatory mechanism of molecular motors is extremely delicate, in which signalling pathways and adaptor proteins play a key role. There is a complex system of signalling pathways in the cell, which builds up a sophisticated regulatory

network to manage the molecular motor collaboration in a comprehensive way [6]. When stimulated by extracellular signals such as growth factors and hormones, specific intracellular signalling pathways are activated, which start the process of modifying molecular motor proteins to precisely regulate their activities.

In the case of kinases, some of them phosphorylate kinesins upon activation of the signalling pathway. As a result of this chemical modification, the kinesin's ability to bind to microtubules is significantly enhanced, and its efficiency in hydrolysing ATP is dramatically increased, thus accelerating the rate of forward transport. At the same time, the corresponding kinesin-associated regulatory pathway is triggered in response to stress signals, allowing for enhanced reverse transport in response to specific cellular conditions [7]. Adaptor proteins, on the other hand, build key bridges between molecular motors and vesicles and cytoskeletal elements. It has the ability to accurately identify and summon suitable motors to the periphery of vesicles, and meticulously regulate the degree of binding and angle of action of the two, so as to ensure that the molecular motors are able to exert their force appropriately in each transport process, and to maintain orderly intracellular substance transport.

## 5. Abnormalities in Molecular Motor Collaboration Patterns and Disease

### 5.1 Diseases Caused by Abnormalities in Molecular Motor Collaboration Patterns

A variety of neurodegenerative diseases are associated with dysfunction of molecular motors. In Alzheimer's disease, abnormalities in the transport of beta-amyloid precursors are the result of an imbalance in the synergistic action of kinesin and dynamin. The precursor protein, which should be transported and processed precisely, is retained in the wrong site due to the malfunction of the motor coordination, accumulates excessively, cuts abnormally and generates amyloid plaques, which gradually erode neuronal function and trigger cognitive decline. In congenital muscular dystrophy, which is a hereditary disease, myosin gene mutation often originates from myosin gene mutation, resulting in structural and functional variations of myosin, and the chain of vesicle transport and energy transfer in muscle cells is broken,

and myofibres are not provided with sufficient nutrients and key proteins, which eventually leads to muscle weakness and atrophy.

### 5.2 Regulation strategy of molecular motor cooperation mode in disease state

Pharmacological intervention is a popular direction of exploration nowadays, aiming to find small molecule compounds that can fine-tune the activity and collaboration of molecular motors. For example, some drugs can target and inhibit over-activated signalling pathway node kinases to avoid abnormal hyperactivation of kinesins, or activate dormant retrograde regulatory pathways to restart the corrective function of kinesins to correct incorrect transport, so as to correct molecular motor coordination. Gene therapy is also aimed at the root of the disease, with the help of CRISPR-Cas9 and other gene editing technologies, to accurately repair mutated molecular motor genes, or introduce normal gene copies, to reshape the normal expression, assembly and collaboration of molecular motors from the source, but still facing off-targeting, immune response and other technological bottlenecks, and is still in the optimisation stage in the laboratory [8].

## 6. Research Prospects

### 6.1 New technology for molecular motor collaboration mode research

Ultra-high resolution microscopy technology continues to innovate, breaking through the traditional optical resolution limit, and is now able to track the dynamics of individual molecules of molecular motors at the nanoscale, capturing each step of their 'footprints' in microtubules and microfilaments, and analysing the details of the collaboration in real time [9]; cryo-electron microscopy technology is good at framing the high-resolution three-dimensional structure of the molecular motors and their complexes, and revealing the molecular motor collaboration mode [10]. Cryo-electron microscopy is good at framing the three-dimensional structure of molecular motors and their complexes in high resolution, revealing the site of action and conformational changes at the atomic level, and providing a static blueprint for the understanding of the collaboration mechanism;

proteomics technology can panoramically scan the network of proteins related to the collaboration of molecular motors, and dig out the potential regulatory factors; and gene editing technology is able to manipulate the genes as it pleases to create a customized cellular model, accurately simulate the abnormal scenarios of the motor collaboration, and speed up the interpretation of the pathogenic mechanism.

## 6.2 Prospects of molecular motor collaboration in drug discovery and development

There is unlimited potential for drug targets based on the molecular motor coordination mechanism. On the one hand, the development of small molecule drugs that directly regulate the activity of molecular motors is expected to overcome neurodegenerative diseases and other persistent diseases. Designing drugs that specifically inhibit the abnormal activity of lesion kinesin or enhance the reverse error correction ability of dynamin to reshape the transport balance. On the other hand, it is promising to construct a molecular motor-mediated intelligent drug delivery system, which encapsulates drugs into vesicles coupled with motors, and delivers them to specific regions

in the diseased cells by virtue of motor navigation, which not only enhances drug efficacy, but also reduces toxicity and side-effects of the drugs on normal tissues, and opens up a new chapter of precision therapy<sup>[10]</sup>.

## 7. Conclusion

The collaborative mode of molecular motors in the Golgi vesicle transport system is a key link in intracellular substance transport. The synergistic action of kinesin and dynamin ensures the efficiency and accuracy of vesicular transport and maintains the homeostasis of the intracellular environment. The regulation of signalling pathways and adaptor proteins further ensures the smooth transport process. However, abnormal molecular motor collaboration patterns can lead to the development of a variety of diseases, such as neurodegenerative and genetic diseases. In the future, as technology continues to advance, we will be able to more deeply analyse the molecular motor coordination mechanism and provide important clues for the development of novel drugs and attacking diseases.

### Disclosure statement

The author declares no conflict of interest.

## References

- [1] Yang F, 2021, Study on the Molecular Mechanism of TBC1D23-Mediated Endosome-Golgi Vesicle Transport, dissertation, Sichuan University. <https://doi.org/10.27342/d.cnki.gscdu.2021.004345>.
- [2] Tara, 2019, Theoretical Study on the Chemical Kinetics Characteristics of Molecular Motors, dissertation, Inner Mongolia University. <https://doi.org/10.27224/d.cnki.gnmdu.2019.000072>.
- [3] Wei M, 2019, The Effect of DRAM1 on Golgi Structure and Vesicle Transport, dissertation, Suzhou University. <https://doi.org/10.27351/d.cnki.gszhu.2019.001917>.
- [4] Zhang T, 2021, Study on the Mechanism of Kinesin's Lateral Movement, dissertation, Inner Mongolia University. <https://doi.org/10.27224/d.cnki.gnmdu.2021.001271>.
- [5] Wang P, 2019, Study on the Motion Characteristics and Mechanisms of Kinesin Molecular Motors at the Single Molecule Level, dissertation, Henan University. <https://doi.org/10.27114/d.cnki.ghnau.2019.000232>.
- [6] Li W, 2022, Study on the Mechanism of Small G Protein Rab43 Regulating Membrane Receptor Vesicle Transport Through Interaction with Golgin160 Protein, dissertation, Shenzhen University. <https://doi.org/10.27321/d.cnki.gszdu.2022.001813>.
- [7] Wang J, 2022, Molecular Dynamics Simulation of the Dissociation Process of Kinesin Along Microtubules, dissertation, Central South University of Forestry and Technology. <https://doi.org/10.27662/d.cnki.gznlc.2022.000511>.

- [8] Li Y, Liu N, Li S, et al., 2020, Classification of Molecular Motors and Their Application Progress in the Field of Medicine. Chinese Pharmaceutical Journal, 55(22): 1829-1835.
- [9] Cui Z, 2021, Study on the Mechanism of Mycobacterium Tuberculosis FIC Protein Interfering with Intracellular Endoplasmic Reticulum-Golgi Vesicle Transport. Heilongjiang Province, Harbin Veterinary Research Institute, Chinese Academy of Agricultural Sciences, 09-22.
- [10] Li C, 2014, Research on the Collaborative Transport Model of Multiple Homogeneous Molecular Motors. Hebei Province, Hebei University of Architecture and Engineering, 07-18.

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