

# **On The Dynamic Timescale Of Mind-Brain Interaction**

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## Introduction

Any physical system is by definition a quantum mechanical system. However, in order macroscopic quantum phenomena to be observed, the dynamical timescale of the system must be comparable to the coherence timescale.

Max Tegmark (1999) shows that if the above condition is not met i.e. the coherence time is much shorter than time of dynamics, the system can be well studied by classical physics.

## Introduction

Let's define the quantum system needed to explain consciousness: neuronal cytoskeleton, presynaptic and postsynaptic scaffold proteins, interneuronal intrasynaptic adhesive molecules ( $\beta$ -neurexin and neuroligin-1).

Mind is modeled via long-range quantum phenomena taking part in the cytoskeleton of brain cortical neurons and it is supposed that mind collapses the wave function.

## Introduction

Let's define the dynamical timescale for this quantum system: it is determined by the tubulin conformational transitions and scaffold protein enzymatic action.

The so determined timescale is the protein dynamical one and varies between 10 to 15 picoseconds. This result is supported by recent studies on myoglobin dynamics by Brunori *et al.* (1999) and Xie *et al.* (2000).

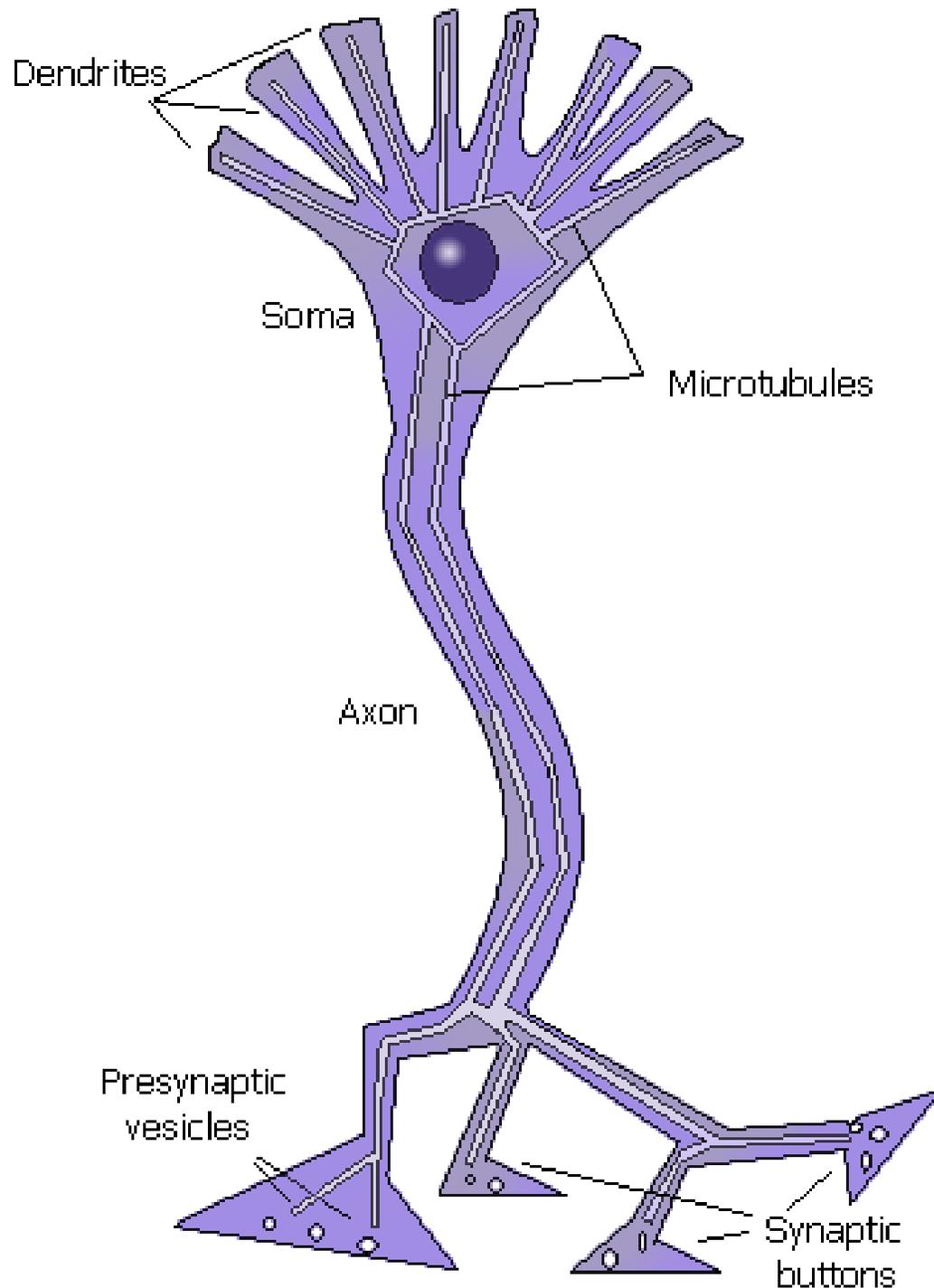
In order to observe macroscopic quantum phenomena in this system it follows that the needed coherence time is also from 10 to 15 picoseconds.

# Introduction

The model has the following advantages:

-> physically speaking it is more realistic to sustain coherence in wet, noisy and wet brain media for a duration of picoseconds than milliseconds.

-> the computational capacity of brain increases as the time for each computational cycle decreases i.e. we get a more powerful brain from a computational point of view.



# Constructing the general framework of mind-brain interaction

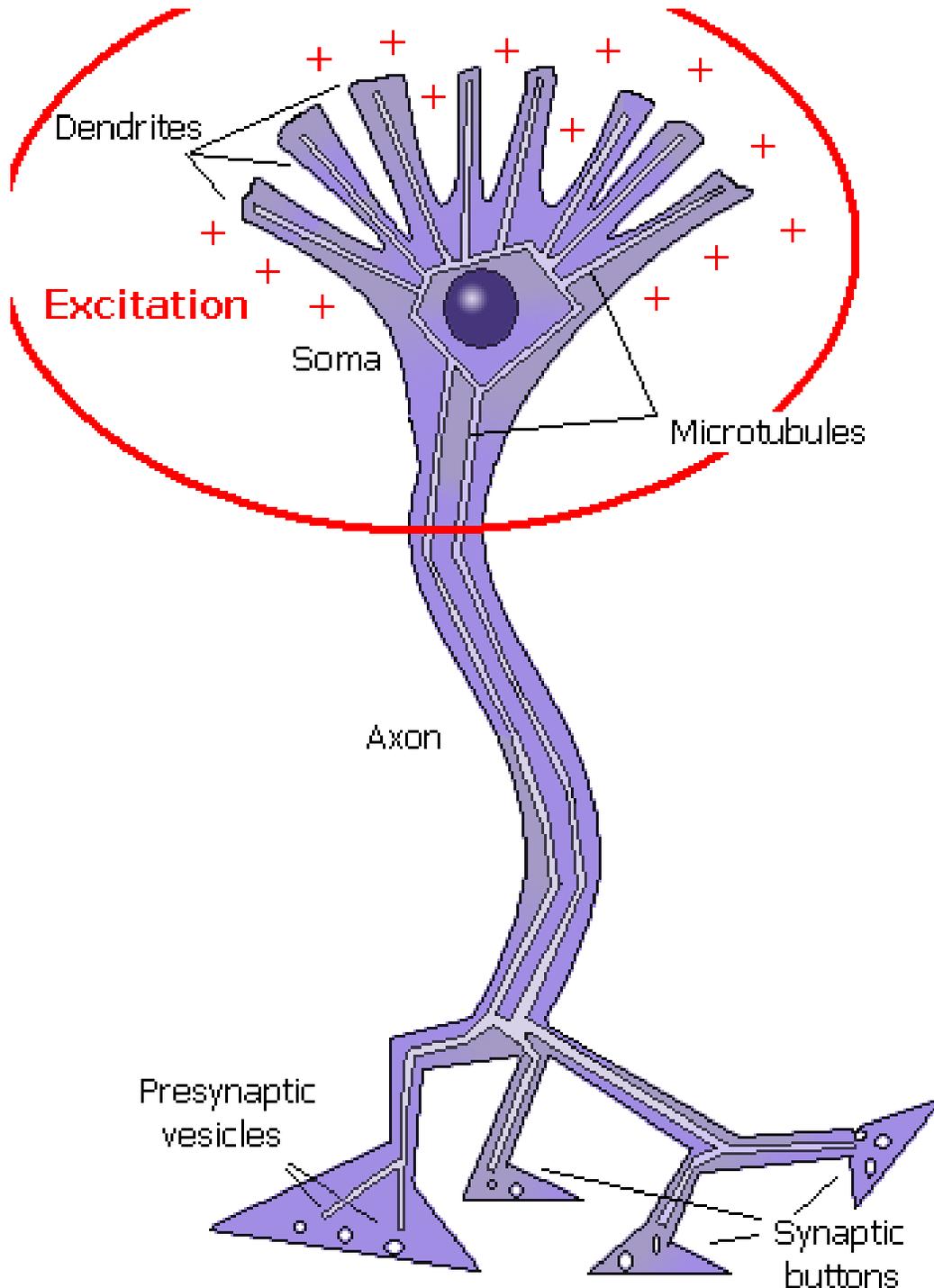
## Neuronal morphology

**Dendrites** – arborizations that input electric impulses

**Soma** – the main body of the neuronal cell

**Axon** – long fiber outputting information to other neurons

**Synaptic buttons** – active sites for interneuronal communication and neuromediator release

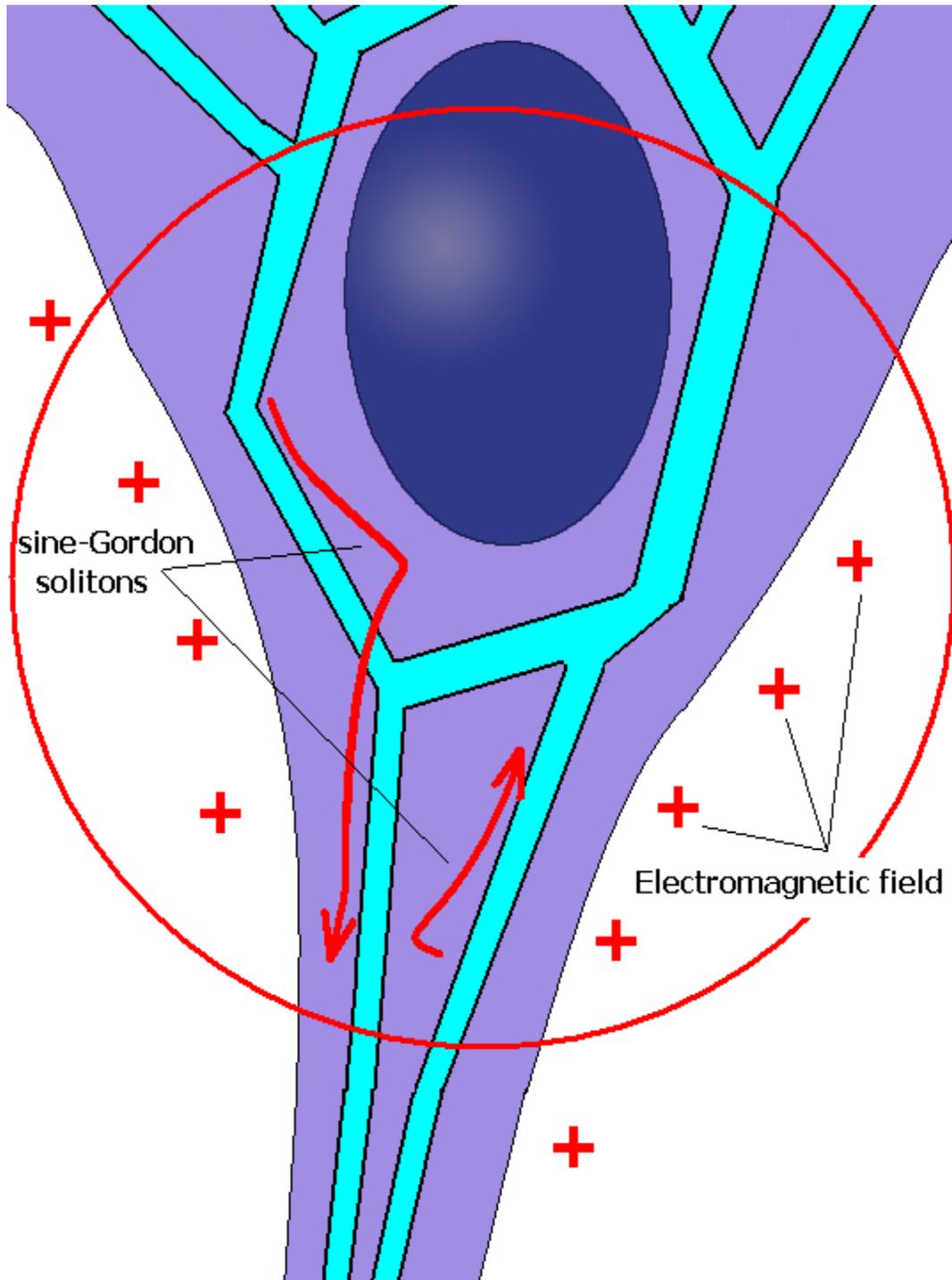


## Dendritic membrane excitation inputs biological information

The released neuromediator activates postsynaptic receptors (ion channels and G-protein coupled receptors) at the dendritic spines.

Activated ion channels then change the membrane potential influencing the local electromagnetic field.

The generated potential propagates electrotonically towards the soma and the axonal hillock.



## The local quantized electromagnetic field interacts with the neuronal microtubules

The microtubular cavities strongly interact with the electromagnetic field.

Abdalla *et al.* (2001) show that the quantized electromagnetic field interacts with the permanent dipole moment of the vicinal water in brain microtubules generating electromagnetic waves, that are sine-Gordon solitons traveling with velocity  $\sim 14$  m/s.

## What is a sine-Gordon soliton?

The localized excitations propagating in a system with constant velocity and colliding with each other without change in their shapes are called solitons. During the collision of solitons the solution cannot be represented as a linear combination of two soliton solutions but after the collision solitons recover their shapes and the only result of collision is a phase shift.

The solitons are solutions of the sine-Gordon equation:

$$\frac{\partial^2}{\partial t^2} \varphi(x, t) - \frac{\partial^2}{\partial x^2} \varphi(x, t) + \sin(\varphi(x, t)) = 0$$

## What is a sine-Gordon soliton?

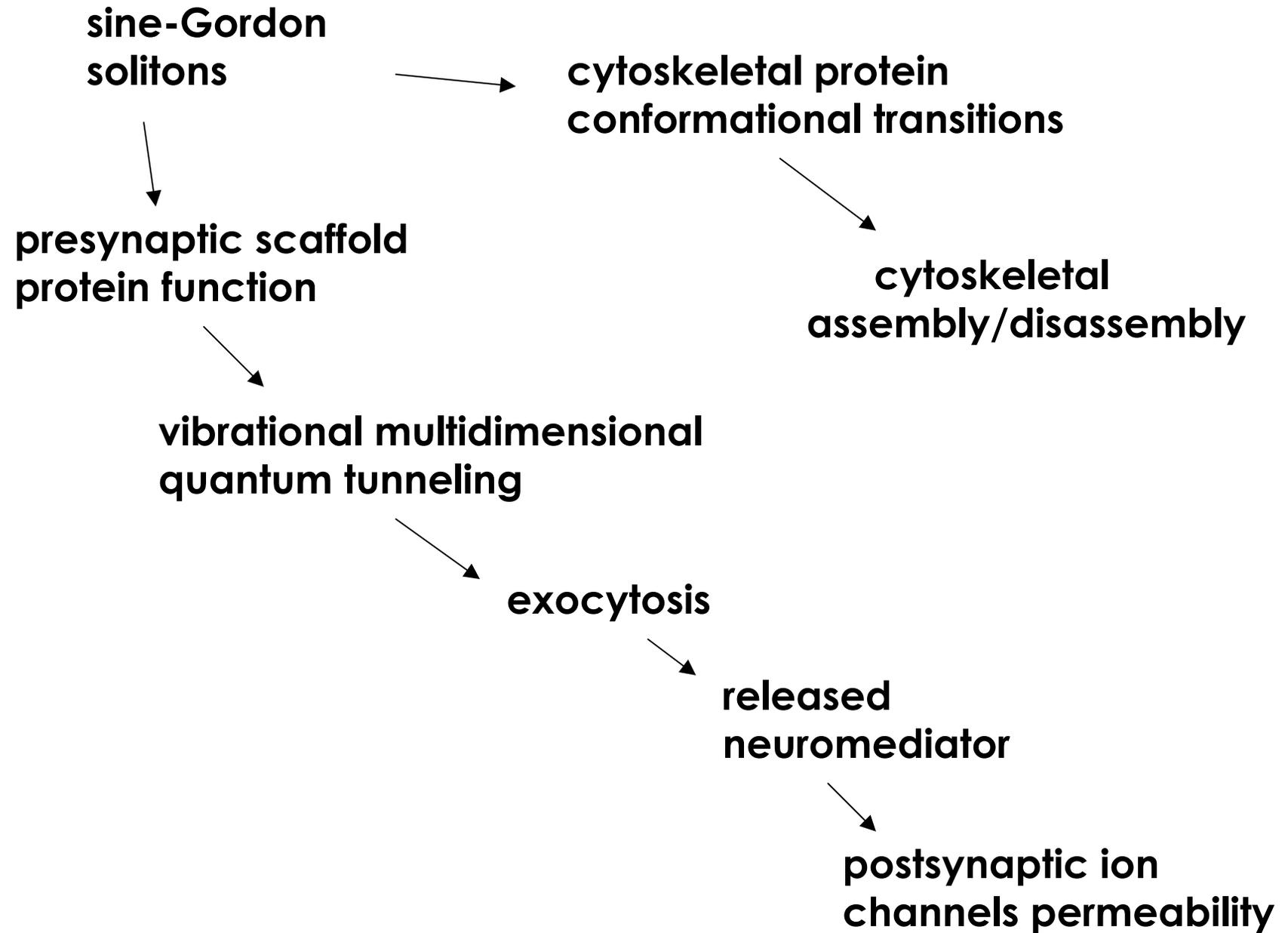
The concept of a soliton provides a new line in research in systems, which are wave-like in nature, e.g. systems involving water or light. The soliton is regarded as an entity, a quasi-particle which conserves its character and interacts with the surroundings and other solitons as a particle.

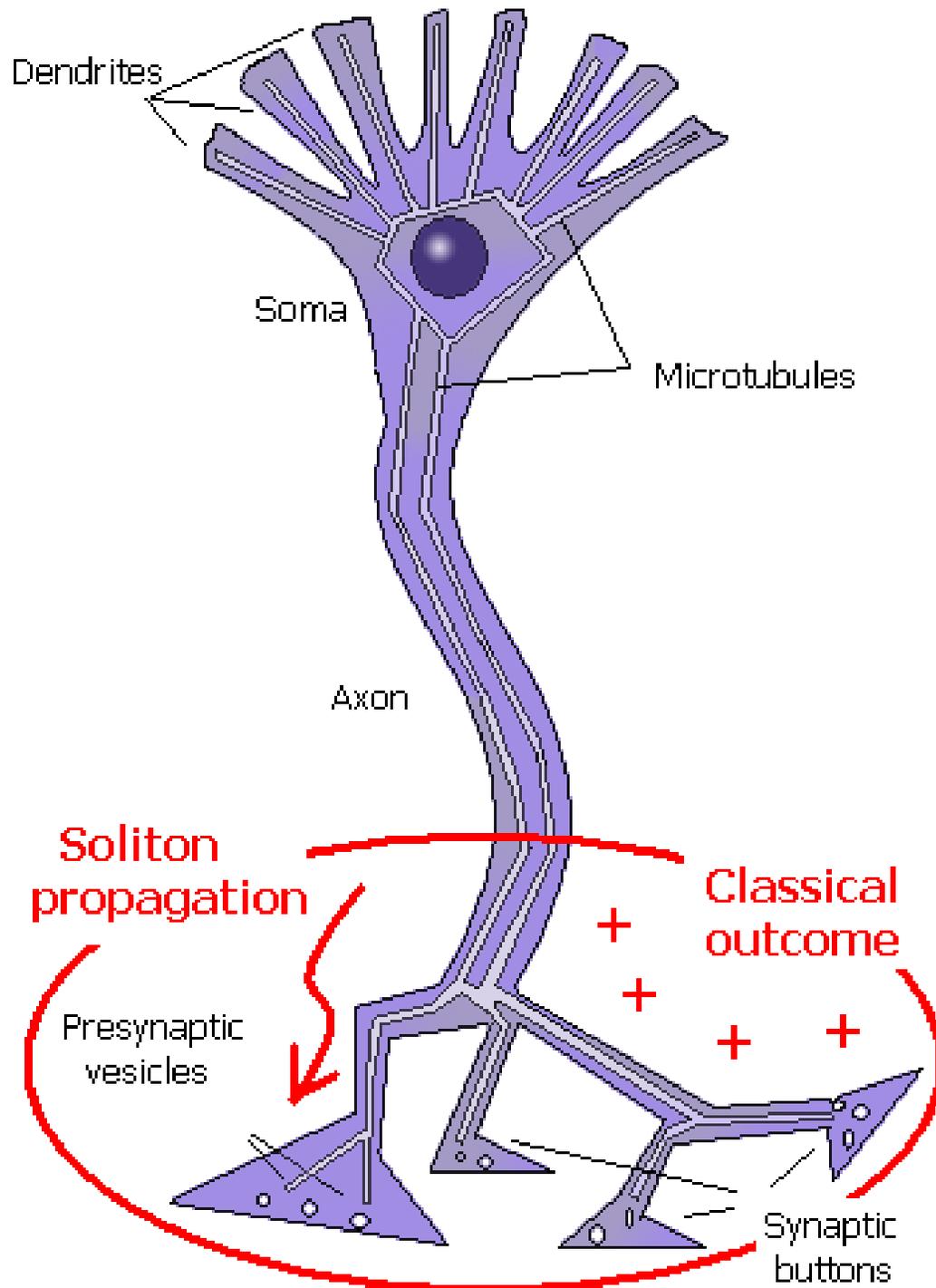
Some of the best known solutions of the sine-Gordon equation are the following solitons: **kinks**, **antikinks**, **breathers** etc.

# Solitons affect cytoskeletal and scaffold protein dynamics

The propagating fast sine-Gordon solitons affect both the cytoskeletal protein conformational transitions and the presynaptic scaffold protein function. The presynaptic scaffold proteins causally affect exocytosis via vibrational multidimensional quantum tunneling and the released neuromediator affects the postsynaptic ion channels.

Thus **mind** controls cytoskeletal and scaffold protein dynamics and indirectly postsynaptic membrane potential, while **brain** inputs information to mind directly using the electromagnetic field.





## Link between classical and quantum computing in brain cortical neurons

Solitons control presynaptic scaffold protein dynamics, thus regulating vesicle tethering, docking and fusion.

Membrane depolarization and  $\text{Ca}^{2+}$  entry is needed for exocytosis i.e. release of neuromediator molecules, but not every impulse leads to presynaptic vesicle fusion.

Beck & Eccles (1992) claimed that quantum tunneling is needed in order exocytosis to occur.

## Quantum tunneling in exocytosis

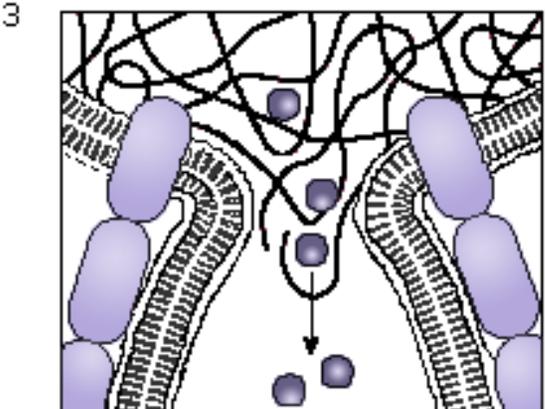
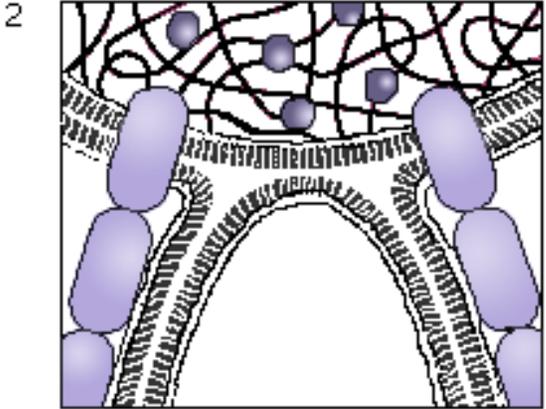
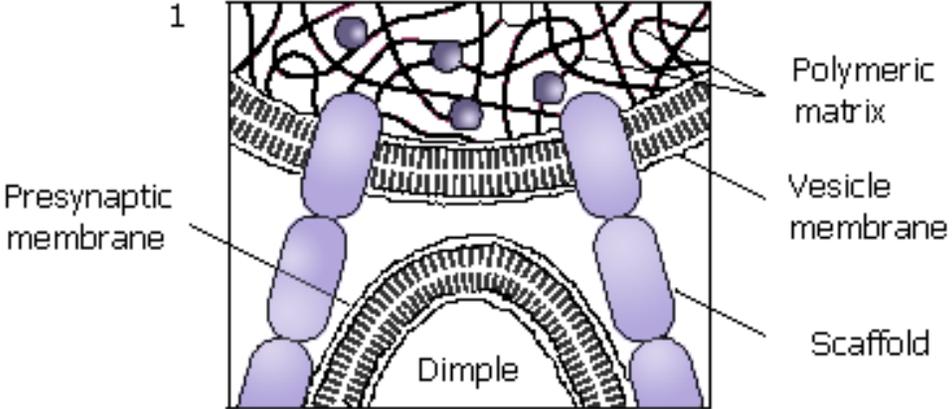
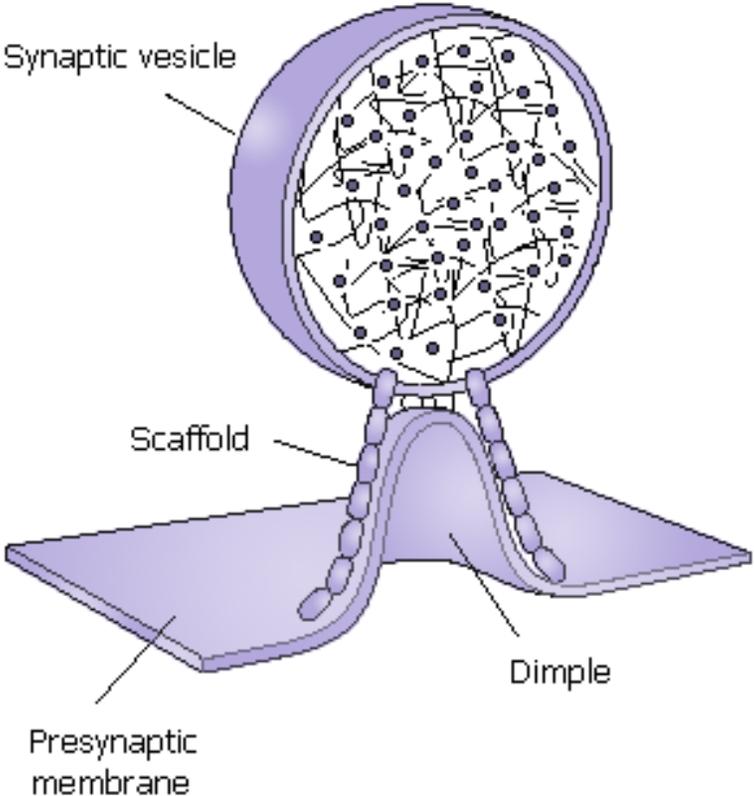
According to Beck (1996) the synaptic exocytosis of neurotransmitters is the key regulator in the neuronal network of the neocortex. This is achieved by filtering incoming nerve impulses according to the excitatory or inhibitory status of the synapses.

Findings by Jack *et al.* (1981) inevitably imply an activation barrier, which hinders vesicular docking, opening, and releasing of transmitter molecules at the presynaptic membrane upon excitation by an incoming nerve impulse.

Redman (1990) demonstrated in single hippocampal pyramidal cells that the process of exocytosis occurs only with probability generally much smaller than one upon each incoming impulse.

In the proposed by us model exocytosis is driven by the scaffold proteins via multidimensional tunneling. The cytoskeleton controls neuromediator release via sine-Gordon solitons.

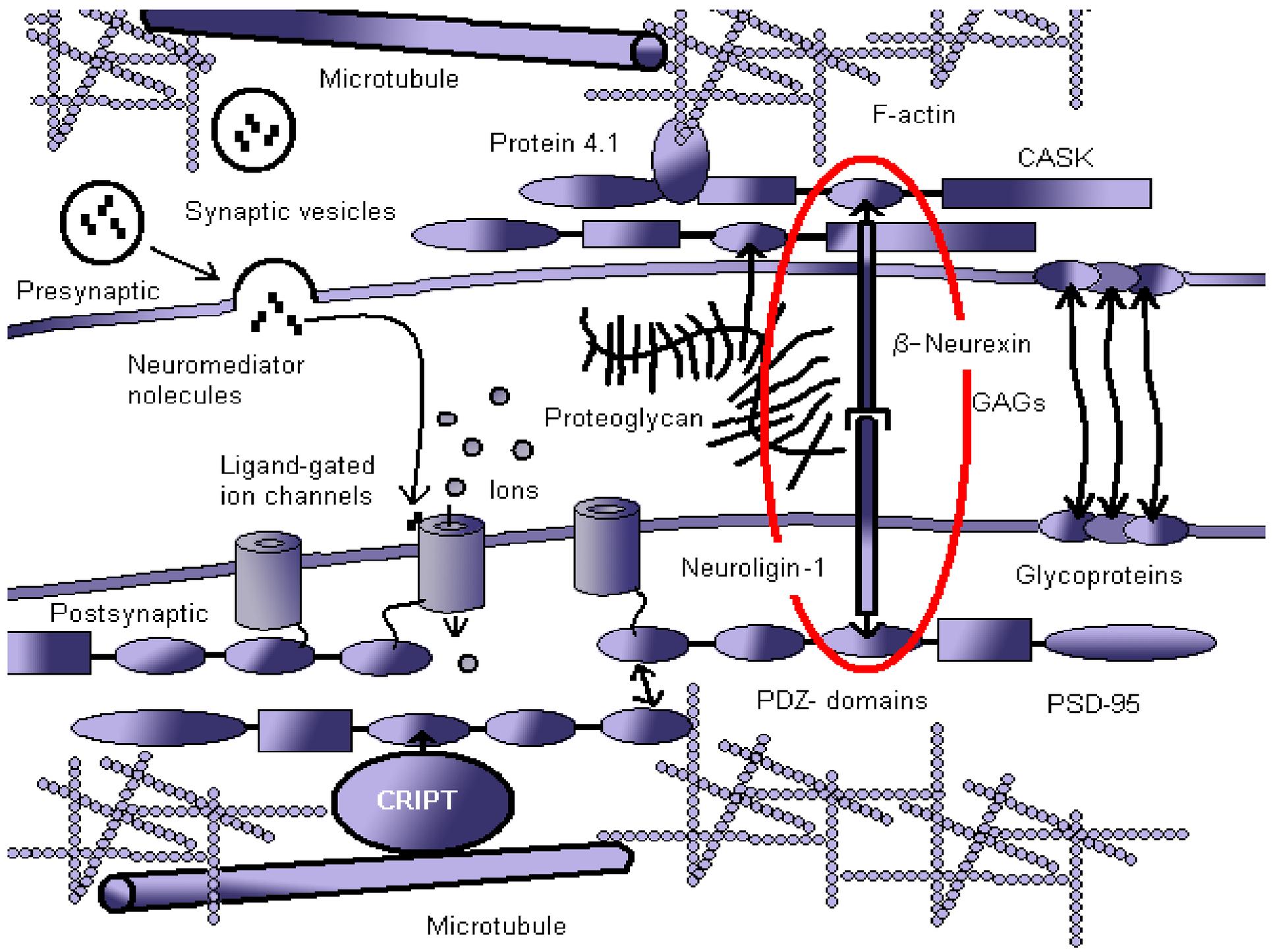
# Molecular mechanism of exocytosis



## How brain cortex sustains interneuronal coherence?

The problem of interneuronal quantum coherence is solved in a novel way using neuromolecular data. Central for the synapse adhesive molecules called  **$\beta$ -neurexin** and **neuroligin-1** that recruit the pre- and post- synaptic machinery are claimed to sustain long-range coherence for 10-15 picoseconds.

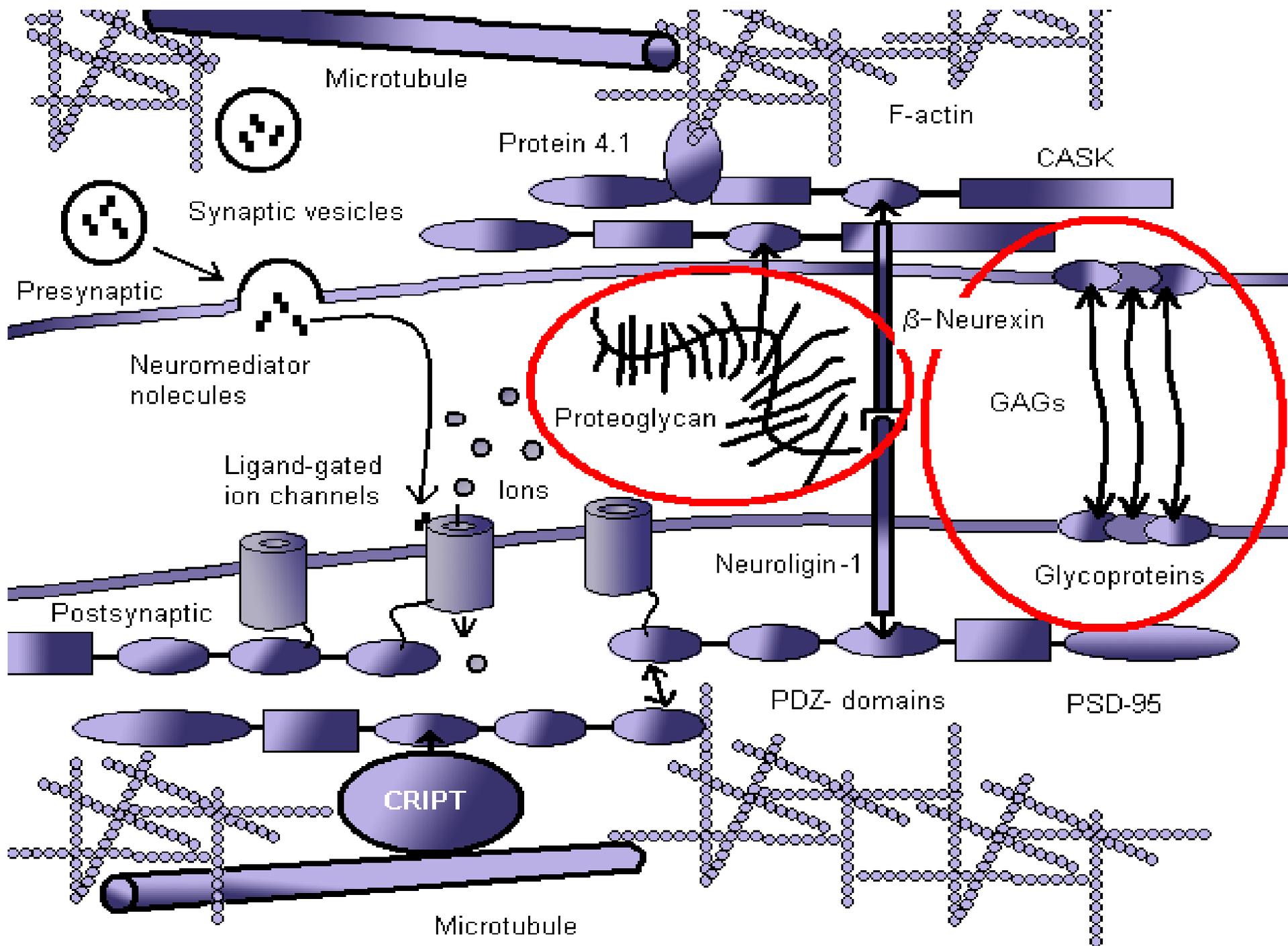
Both, neuroligin-1 and beta-neurexin are the cores of well-characterized intracellular protein-protein interaction cascades. These link  $\beta$ -neurexin to the presynaptic transmitter secretion apparatus and neuroligin-1 to components of the postsynaptic signal transduction machinery.



## How $\beta$ -neurexin-neuroigin-1 link is insulated?

The extracellular  $\beta$ -neurexin-neuroigin-1 adhesion could mediate interneuronal entanglement because it is surrounded by extracellular intrasynaptic matrix molecules and could be permanently shielded.

**Glycosaminoglycans (GAGs)**, which interconnect the two neural membranes, intrasynaptic **proteoglycans** molecules (like phosphacan or CAT-301) and **dystroglycan complexes (DGC)** project negative chemical groups ordering the water molecules and ions in the vicinity of the beta-neurexin-neuroigin-1 adhesion.

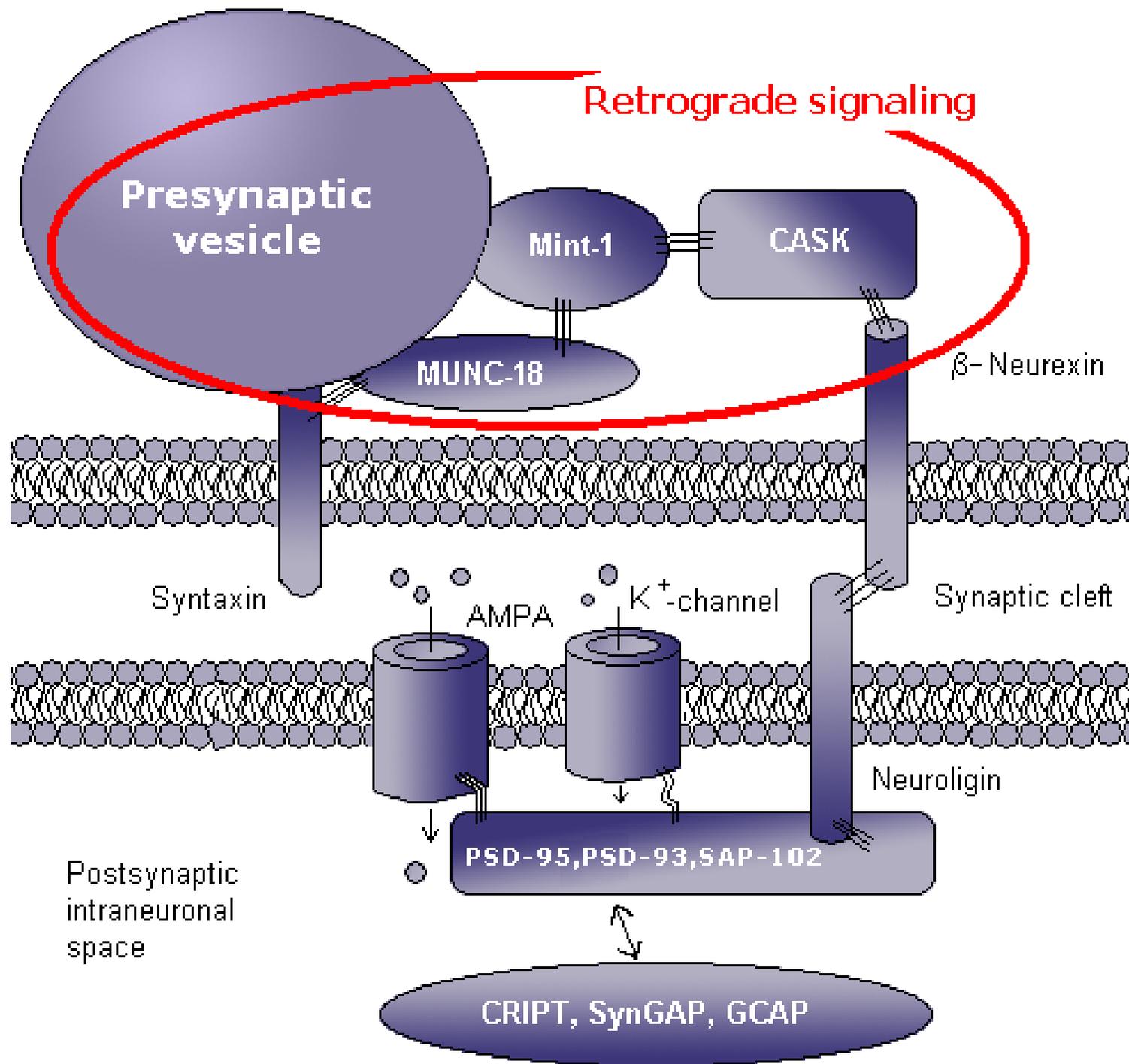


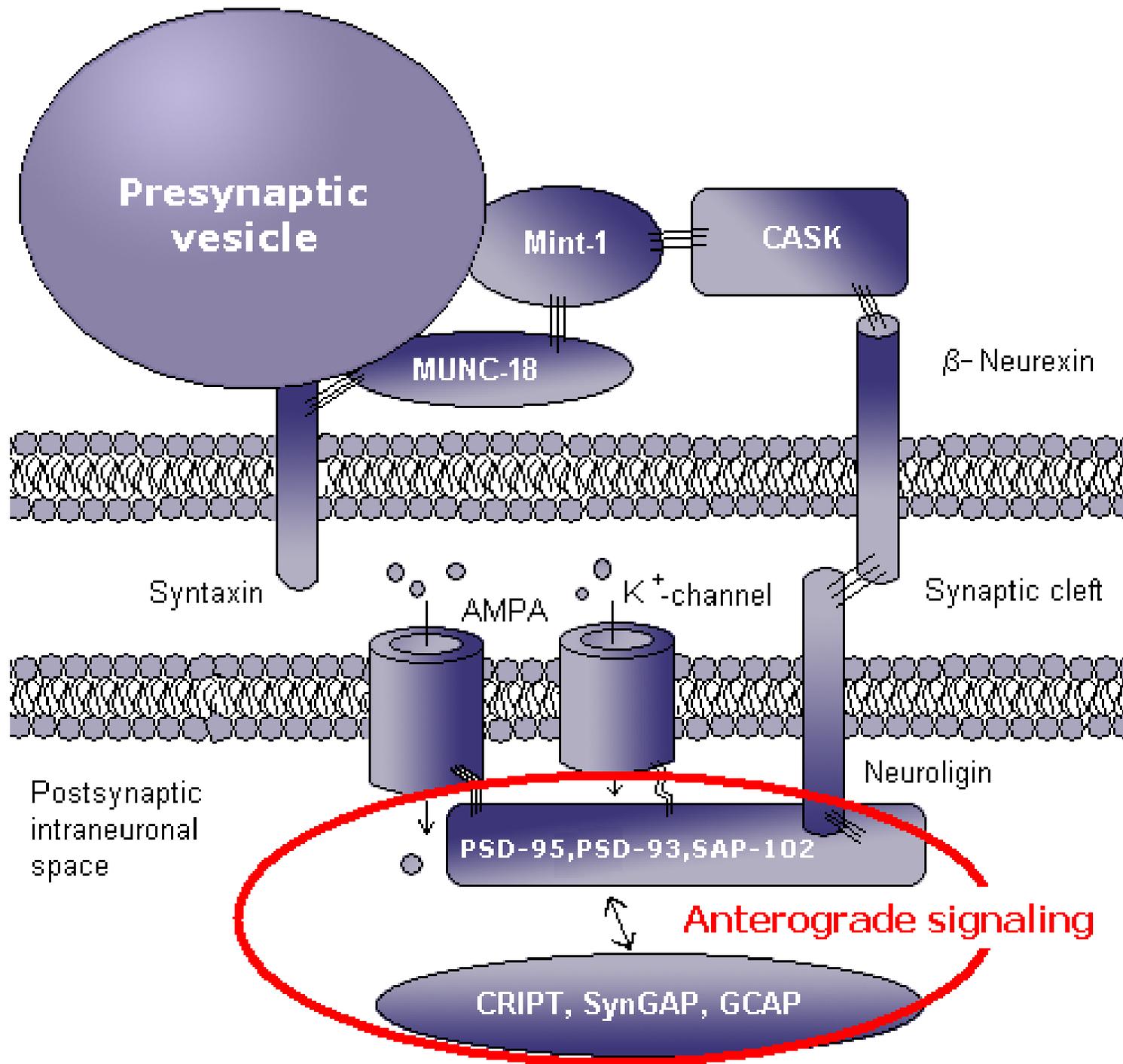
## **The $\beta$ -neurexin-neuroigin-1 link importance**

The extracellular  $\beta$ -neurexin-neuroigin-1 adhesion is the core of a newly forming excitatory synapse (Brose, 1999). About 90% of the cortico-cortical synapses are glutamatergic (excitatory).

It provides an interesting and simple mechanism for retrograde signalling during learning-dependent changes in synaptic connectivity. Indeed, the  $\beta$ -neurexin-neuroigin-1 junction allows for direct signalling between the postsynaptic nerve cell and the presynaptic transmitter secretion machinery.

Neurophysiologists and cognitive neurobiologists have postulated such retrograde signalling as a functional prerequisite for learning processes in the brain.





## **Final notes on the dynamical timescale of mind action**

In the proposed quantum model of brain cortex, mind is intimately associated with subneuronal coherent protein states, thus providing us with well-determined timescale for its action i.e. protein dynamical timescale  $\sim 10\text{-}15$  ps.

At first sight this timescale is in direct contradiction with the experimentally obtained millisecond timescale for a neural reflex. However, the neural reflex has two compounds: impulse propagation lasting milliseconds (input and output) and informational processing, which is much faster (picoseconds). So, the overall process duration is defined by the slower component.

## **Final notes on the dynamical timescale of mind action**

The input by the electromagnetic field to the microtubules supplies high quality information because the dynamical timescale of electromagnetic field is ~ 8 orders of magnitude larger than the needed time for quantum processing and transfer of information. For those 10-15 ps needed for the wave function to be collapsed by the mind, the local electromagnetic field can be considered as stable or unchangeable one. Thus mind controls the function of the intraneuronal cytoskeletal and scaffold proteins causally affecting most of the brain processes (exocytosis, cytoskeletal assembly/disassembly, synaptic plasticity etc.).

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