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THE EFFECT OF GRAPHENE ON REDOX HOMEOSTASIS AND SYNAPTIC TRANSMISSION IN NEURONS

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Introduction. Over the past decade, graphene and its derivatives (graphene oxide (GO) and reduced graphene oxide (rGO)) have attracted significant attention from researchers at the intersection of neuroscience and biochemistry due to their unique combination of physical, chemical, and electrochemical properties. In general, graphene is a two-dimensional allotrope of carbon with an atomically thin crystal lattice, which accounts for its extremely high specific surface area (~2630 m^2/g), excellent electrical conductivity, mechanical strength, and biochemical reactivity [1].

At the same time, the central nervous system is one of the most metabolically active systems: the human brain consumes about 20% of the body's oxygen - this makes neurons particularly vulnerable to any disturbances in redox balance (their mitochondria are both the primary producers of ATP and a source of reactive oxygen species (ROS)). Therefore, maintaining redox homeostasis is critical for effective synaptic transmission and neuroplasticity [2].

In general, when introduced into the neuromicroenvironment, graphene nanoparticles can directly interact with the lipid bilayer of plasma and mitochondrial membranes, as well as with soluble protein components of the synaptic apparatus. These interactions modulate the redox status of cells, the release of neurotransmitters, and the electrophysiological properties of synapses.

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Thus, the aim of this work was to systematize and critically analyze existing data on the biochemical mechanisms by which graphene affects neurons.

1. The Structure and Forms of Graphene

Structurally, graphene is a single-layer sheet of sp^2-hybridized carbon atoms arranged in a hexagonal lattice. The three most commonly mentioned forms are: GO (a covalently functionalized form with epoxy, hydroxyl, and carboxyl groups on the surface), rGO (partially stripped of oxygen-containing groups), and graphene quantum dots (nanoscale fragments with photoluminescent properties) [3].

It is the degree of oxidation, particle size, specific surface area, layer thickness, and the presence of functional groups that significantly determine the bioactivity of the material. Thus, due to the hydrophilic nature of its surface, GO disperses more easily in aqueous media and exhibits a greater propensity to interact with proteins. rGO, on the other hand, is more hydrophobic and therefore penetrates lipid membranes more effectively [4]. These structural differences directly determine the varying degrees of toxicity and modulation of cellular functions.

2. The Effect of Graphene on Redox Homeostasis in Neurons

The redox homeostasis of neurons is maintained by a delicate balance between ROS production and the capacity of antioxidant systems (a constantly self-regulating system). The main enzymatic components involved are superoxide dismutase, catalase, glutathione peroxidase, and thioredoxin reductase. Non-enzymatic protection is provided by reduced glutathione (GSH), vitamin E, ascorbic acid, and ubiquinol [2].

Thus, studies show that OG can generate superoxide anions and hydroxyl radicals via iron-catalyzed Fenton reactions (iron is present as an impurity on the surface of nanomaterials). At the same time, interaction with NADH dehydrogenase (Complex I) of the mitochondrial respiratory chain disrupts normal electron transport, increasing electron leakage and, consequently, the production of superoxide anions in the mitochondrial matrix [5].

The response of neuronal antioxidant systems to graphene is concentration-dependent and ambiguous. Thus, at low concentrations (1–10 μg/mL), GO can activate the Nrf2 transcription factor and enhance the expression of heme oxygenase-1 (HO-1), NADH quinone reductase (NQO1), and glutamate cysteine ligase (GCL), i.e., effectively exerting an adaptive hormetic effect [6]. At the same time, at concentrations exceeding 50-100 μg/mL, there is a suppression of superoxide dismutase and catalase activity, a decrease in GSH levels, and an increase in malondialdehyde concentration (a marker of lipid peroxidation), which directly indicates oxidative stress [5].

Apart from directly generating ROS, graphene can cause physical damage to mitochondrial membranes, reduce their membrane potential, inhibit cytochrome

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c oxidase activity, and disrupt mitophagy. A decrease in membrane potential leads to reduced ATP synthesis and may trigger an apoptotic cascade through the release of cytochrome C into the cytosol [5].

3. Interaction of graphene with neuronal membranes and ion channels

Among other things, graphene nanoplates have sharp edges and a large surface area, and are therefore capable of physically penetrating lipid bilayers. Consequently, upon contact with rGO particles, nanopores form in the plasma membrane of neurons, leading to an increase in the membrane's permeability for Ca^{2+} i Na^{+} [7].

Interactions with voltage-gated Na^{+}/K^{+} and Ca^{2+} channels (which are key to the generation of action potentials and synaptic transmission) are clearly of particular importance. For example, graphene oxide inhibits currents through voltage-gated K^{+} channels of the KV1.5 and KV2.1 types in primary cultures of cortical neurons, which can lead to impaired membrane repolarization and unpredictable changes in firing rates. At the same time, modulation of NMDA (N-methyl-D-aspartate) receptors is also known, where adsorption of nanostructures near the synaptic cleft limits glutamate diffusion, reducing the likelihood of receptor activation [8].

4. The Effect of Graphene on Synaptic Transmission and Neuroplasticity

Synaptic transmission is a complex cascade of events involving synthesis, vesicular storage, Ca^{2+}-dependent fusion of vesicles with the presynaptic membrane, and the direct release of the neurotransmitter into the synaptic cleft, its diffusion, binding to postsynaptic receptors, and reuptake. Graphene has mechanisms that influence each of these stages [1, 9].

At the presynaptic terminal, GO-induced oxidative stress disrupts the function of the vesicular monoamine transporter (VMAT2), thereby reducing the capacity of presynaptic vesicles. As a result, Ca^{2+} signaling is dysregulated due to an increase in intracellular Ca^{2+} concentration. This concentration leads to uncontrolled vesicle fusion and excessive release of neurotransmitters, resulting in their depletion [9]. In glutamatergic neurons, this state triggers excitotoxicity - a pathological mechanism involved in ischemia and neurodegenerative diseases [2].

On the other hand, a number of studies demonstrate the neuroprotective and neuromodulatory effects of functionalized forms of graphene. Graphene quantum dots conjugated with brain-derived neurotrophic factor (BDNF) increase synaptic density and facilitate long-term potentiation (LTP) - a cellular correlate of memory and learning—through the activation of TrkB receptors and the PI3K/Akt/mTOR signaling cascade [10]. It has also been shown that rGO scaffold substrates stimulate the differentiation of neural stem cells into functional neurons and increase the number of dendritic spines [3, 10].

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At the postsynaptic level, graphene is capable of adsorbing neurotransmitters through π-π-stacking interactions with the aromatic rings of dopamine, serotonin, and epinephrine, which reduces the effective concentration of the neurotransmitter in the synaptic cleft and attenuates activation. For dopaminergic synapses, this mechanism may be relevant in the context of the pathophysiology of Parkinson's disease and schizophrenia [2].

5. Factors Determining the Neurotoxicity of Graphene

Overall, an analysis of the literature indicates that the neurotoxicological profile of graphene is heterogeneous and depends on a combination of factors. First, there is the degree of oxidation: GO exhibits higher neurotoxicity compared to rGO due to a greater number of reactive oxygen-containing groups. Second, particle size: smaller particles (< 200 nm) demonstrate a more pronounced tendency toward internalization and disruption of the redox balance. Third, concentration: toxic effects are dose-dependent. Fourth, surface functionalization: the introduction of amine (-NH), carboxyl (-COOH), or PEG groups changes how the material interact with cellular components and can greatly lower its cytotoxicity [4].

Furthermore, the state of the neurons is also a key factor. Thus, mature post-mitotic neurons, neural stem cells, and differentiated neural precursors exhibit varying degrees of sensitivity, which is due to differences in antioxidant defense and proliferative status [6].

Conclusions. Graphene and its derivatives exert a complex, concentration-dependent effect on redox homeostasis and synaptic transmission in neurons. The main mechanisms of neurotoxicity include increased mitochondrial ROS generation, inhibition of antioxidant enzymes, destabilization of lipid membranes, disruption of ion homeostasis, and modulation of vesicular neurotransmitter release. At the same time, functionalized forms of graphene at low concentrations activate Nrf2-dependent antioxidant defense and stimulate synaptic plasticity. Thus, further study of the interaction between the CNS and graphene remains relevant.

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