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Tiny Rare-Earth Fluoride Nanoparticles Activate Tumour Cell Growth via Electrical Polar Interactions

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Abstract

Localised extracellular interactions between nanoparticles and transmembrane signal receptors may well activate cancer cell growth. Herein, tiny LaF_3 and PrF_3 nanoparticles in DMEM+FBS suspensions stimulated tumour cell growth in three different human cell lines (A549, SW837 and MCF7). Size distribution of nanoparticles, activation of AKT and ERK signalling pathways and viability tests pointed to mechanical stimulation of ligand adhesion binding sites of integrins and EGFR via a synergistic action of an ensemble of tiny size nanoparticles (< 10 nm). While tiny size nanoparticles may be well associated with the activation of EGFR, integrin interplay with nanoparticles remains a multifaceted issue. A theoretical motif shows that, within the requisite pN force scale, each ligand adhesion binding site can be activated by a tiny size dielectric nanoparticle via electrical dipole interaction. The size of the active nanoparticle stayed specified by the amount of the surface charges on the ligand adhesion binding site and the nanoparticle, and also by the separating distance between them. The polar component of the electrical dipole force remained inversely proportional to the second power of nanoparticle's size, evincing that only tiny size dielectric nanoparticles might stimulate cancer cell growth via electrical dipole interactions. The work contributes towards recognising different cytoskeletal stressing modes of cancer cells.

Keywords: Physics of cancer, Tumorigenesis, Cancer and nanoparticles, Mechanotransducers, Mechanosensors, Integrins, EGFR, Nanotechnology, Biosurfaces, Atomic force microscopy, Electrical dipole interactions

Background

Tumorigenesis is a multidimensional issue involving genomic changes. It is also activated by cell-extracellular matrix (ECM) interactions between scaffolds and cytoskeletal structures [1–4] expressed via stressing of mechanosensors, similar to integrins, from multipart cellular forces capable of altering genomic programming [5]. The interactions of tumour microenvironment with ECM scaffolds usually activate cell's membrane focal adhesion proteins and transmembrane signal receptors (TSR), epidermal growth factor receptors (EGFR), vascular endothelial growth factor (VEGFR) or nerve growth factor receptors (NGFR). The mechanosensors regulate tumour cell growth via signal transaction between the extracellular active domain of cells [6–9] and the intracellular F-actin filaments, by triggering an avalanche of phosphorylation reactions.

Protein conformational changes and excitation of TSR pathways requires the activating force to lay in the pN force range, and certainly below the nN gauge [10]. Besides random mechanical stressing and active chemical affinity strength, the binding efficiency (strength of bonding) between nanoparticles (NPs) and the proteins of the cell membrane can be modulated either via short or long-range electrical polar or other types of dispersive interactions. On the limited surface area of NPs, only a certain number of proteins can be attached for long enough to be biologically active [11], and space-confined local interactions with the biological milieu was recognised to be responsible for a set of diverging cell functionality routes [12]. Consequently, the

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