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J Nanomed Nanotechnol 2016, 7:5 (Suppl)
http://dx.doi.org/10.4172/2157-7439.C1.044

Effect of deposition potential, pH, deposition temperature and electrolytic cell concentration on formation of cobalt nanowires

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To understand the mechanism for formation of fcc-cobalt nanowires in electrodeposition, we have systematically studied the effect of deposition potential, pH, deposition temperature and electrolytic cell concentration on the formation of fcc Co nanowires by X-ray diffraction (XRD), transmission electron microscope (TEM) and scanning electron microscope (SEM). The Co nanowires deposited at the potential of -1.6 V are pure hcp phase. When increasing the value of potential to -2.0 V, there are hcp Co and fcc Co crystals in the deposited nanowires. The fraction of fcc Co crystals in the nanowires increases with increasing the potential value. At -3.0 V, the nanowires are pure fcc Co. The pH of the solution has little effect on formation of fcc Co nanowires. We have also seen that high concentration and low temperature favors fcc phase whereas low concentration and high temperature favors hcp phase. However, at 35oC the co-occurrence of hcp and fcc phases were also observed. These experimental results can be explained by the classical electrochemical nucleation theory. The formation of fcc Co crystals can be attributed to smaller critical clusters formed at a higher potential value since the smaller critical clusters favor formation of fcc nuclei.

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Liposome-based co-delivery of paclitaxel and docetaxel enhances oxidative stress in MCF-7 breast cancer cells

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Introduction: Oxidative stress has been shown to be involved in cell death based on the extent of redox unbalance. It is known that cancer cells with increased oxidative stress are likely to be more vulnerable to damage by further ROS (reactive oxygen species) when compared to normal cells. The aim of this study was to evaluate the profile of ROS production by breast cancer cells (MCF-7 cells) in the presence of co-encapsulated anticancer drugs. We developed a novel liposomal formulation encapsulating paclitaxel (PCT) and docetaxel (DCT) and analyzed the relation between ROS production and necrosis in MCF-7 cells.

Methods: Liposomes were prepared by the thin film hydration method followed by extrusion through 100 nm polycarbonate membranes. Following extrusion, the non-encapsulated drug was removed by syringe filtration of the formulation through 0.22 μm membranes. Liposomal drug concentrations were determined using reverse phase high performance liquid chromatography (HPLC). MCF-7 cells were incubated at 37°C, 5% CO2 atmosphere in the presence of the formulations for 48 h. Necrosis and ROS generation were determined by flow cytometry (FACSCalibur, BD®, Biosciences, USA) using propidium iodide (BD®, Biosciences) and the oxidation of 2,7-dichlorodihydrofluorescein diacetate (Sigma Aldrich, USA), respectively. All results were performed in triplicate of 3 independent experiments. Data were submitted to ANOVA and the post-hoc Tukey statistical test, considering $p<0.05$ as significant.

Results & Discussion: The liposomal formulations exhibited a homogeneous size distribution of approximately 150 nm, with mean zeta potential of ca. -3 mV. A significant necrosis response and increased ROS production of PCT and DCT after encapsulation into liposomes were induced when compared to the free drugs. These findings suggest that oxidative stress can be involved in the progression of cell death caused by combination of PCT and DCT co-encapsulated drugs in liposomes.

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