Evaluation of the Pulmonary Inflammatory Response in Murine Biomodels Exposed to Modified Titanium Oxide (Modified TiO2)

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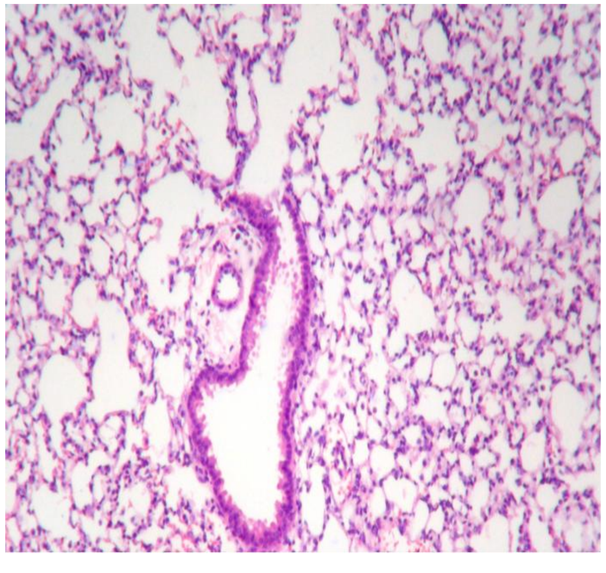
Introduction

Nanomaterials (NM) in animals can cause inflammation and tissue damage in the lung, liver and kidney, among other organs. We evaluated the toxicokinetics of TITANIUM OXIDE (modified TiO2) NM in a murine biomodel. Initially, exposure of cultivated HeLa cells (cervical cancer cells) to this NM caused 98.6% toxicity when stimulated with phototherapy, while no toxicity, genotoxicity or mutagenic toxicity were found in CHO cells (hamster ovary cells)[1]. In the murine biomodel, there was no histological change in lung tissue, apoptosis or deposit of NM, opening a further possibility for the use of these elements in diseases such as cancer.



Murine lung parenchyma (H&E) with no evidence of inflammatory reaction to exposure to doses of 5000 mg NM/kg

Methods

We used the BALB / c albino mice to assess acute and chronic toxicity. The mice were divided in the following groups:

Group A: 3 females, 21 days old, were inoculated with 300 mg / kg in the peritoneum in a single dose and then sacrificed 10 days after exposure.

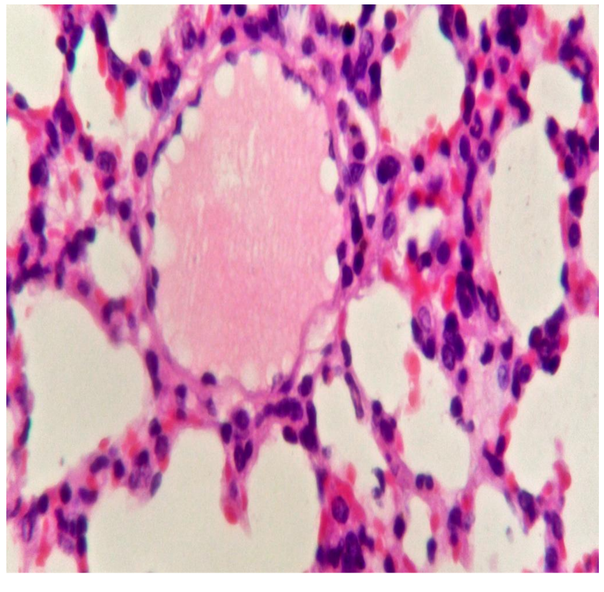
Group B: 9 females, 21 days old, were inoculated with 600 mg / kg in the peritoneum during 10 days and then sacrificed 10 days after the last exposure.

Group C: 5 females, 21 days old, were inoculated with 5000 mg / kg in the peritoneum in a single dose and sacrificed 10 days after exposure.

Three females of the same age were used as controls. The Irwin test showed no changes in behavior, neurological and physiological status in any group. In group B, the NM was applied every day during 10 days to look for forced signals of toxicity [2][3]. Histology examination was performed with H & E staining.

Results

No changes were found in mice exposed to immediate toxicity (n = 3 / n GA: 5 / GC) or chronic (n = 9 /GB) in the lungs or other organs such as liver, kidney, heart, small or large intestine, stomach or brain. In the lung specifically, no diffuse alveolar damage, alveolar hemorrhage, cilia or respiratory epithelium damage, hyperplasia of type 2 pneumocytes, acute inflammation, chronic granulomatous reaction to foreign body, ischemia, thrombosis, cellular changes cytotoxicity, apoptosis or deposit NM was present. None of the mice died during the experiment.



Murine lung parenchyma without histopathological changes suggestive of toxicity (H & E)

Conclusions

In the study of acute and chronic toxicity of NM modified TiO2 there were no histopathological changes compared to controls, with promising results, opening new therapeutic perspectives for future applicability as a therapeutic procedure in humans.

Bibliography

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